

APRAXIA OF SPEECH THEORY, ASSESSMENT, DIFFERENTIAL DIAGNOSIS, AND TREATMENT: PAST, PRESENT, AND FUTURE

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Abstract: A 50-plus year history of research on apraxia of speech (AOS) has spawned a body of literature that has been always controversial, at times contradictory, and at other times enlightening and coherent. This chapter reviews briefly the phonetic theory and neurological substrates for the disorder. We discuss neurolinguistic models and experimental evidence that have helped to set diagnostic criteria for AOS and that separate it from aphasia and the dysarthrias. The important, but underspecified, methods and procedures for the assessment of AOS are discussed, and the current status, recent trends, and advances in AOS treatment are reviewed. Although much remains to be explored about the neuropsychological mechanisms of the disorder and about its clinical identification and management, it is apparent that AOS (a) is an entity that is theoretically and clinically separable from language and other sensorimotor speech disorders, (b) is treatable, and (c) provides a model for exploring the interface between language and speech movements. Finally, we have identified what we believe are likely future trends in theory, diagnosis, assessment, and treatment of AOS that expand on current research and clinical practice rather than produce a paradigm change in any one of these areas.

The concept that there exists a distinct clinical entity that impairs speech planning and/or programming has remained stable since the 1960s when long held notions about “motor aphasia” were perturbed—most notably by Darley, Luria, and Wepman—and then mutated into what Darley (1969) called *apraxia of speech* (AOS). Since then, this underspecified

core concept has been on a nonlinear, sometimes circular, but generally forward-moving theoretical and clinical journey.¹

Theory and Neurological Underpinnings

Darley's conceptual and nosologic contributions² set the stage for studies of AOS in subsequent decades that (a) described its features and the variables that influence their emergence and (b) began to map the theoretical and clinical borders between it and its bookend disorders—aphasia and dysarthria. Although there was little controversy about distinctions between dysarthria and AOS during the 1970s and 1980s, there was considerable debate about whether AOS was indeed separable from aphasia. Confusion was fueled by definitions of AOS as a *phonological* disorder (Wertz, LaPointe, & Rosenbek, 1984) and by efforts to address its *phonemic* variability (Johns & Darley, 1970), despite the investigators' conclusions that AOS was a motor, not a linguistic, disorder. Relatedly, and more problematic, the inclusion of people with phonemic paraphasias who may or may not also have had AOS likely contaminated some experimental findings (McNeil, Doyle, & Wambaugh, 2000). Nevertheless, acoustic, physiologic, and narrow phonetic transcription studies eventually identified features not easily explained as phonological or neuromuscular in nature, at least relative to models at the time. The accumulating evidence led to further revision of the core characteristics of AOS as well as overlap features that can be phonological, dysarthric, or apraxic in nature.

Today, the term “phonological” is nowhere to be found in most definitions of AOS, beyond its explicit exclusion as the disorder's core disturbance. AOS is now broadly defined, for example, as reflecting “inefficiencies in the translation of well-formed and -filled phonological frames into previously learned kinematic information” (McNeil, Robin, & Schmidt, 2009, p. 264), or interference with “speech-specific mechanisms of the organization of vocal tract gestures” (Ziegler, 2009, p. 659). From this standpoint, the theoretical distinction between the two processes (phonologic and phonetic) and between the two clinical problems (phonologic paraphasias and AOS errors) has been clarified. Although a gray area may be reopening at the phonologic and phonetic encoding interface (discussed later), the related theoretical issues will likely be more clearly defined than they have been in the past. Clinical descriptions have also changed—migrating from AOS being considered an articulatory disorder (Darley, 1969) to an articulatory disorder with secondary prosodic compensations (Darley, Aronson, & Brown, 1975) or with primary or secondary prosodic abnormalities (Wertz et al., 1984)—to current acceptance that both articulatory and prosodic abnormalities are primary features for its identification.

Today, after a long latent period, the concept and label of AOS have spread beyond the practice and lexicon of speech-language pathology and are increasingly recognized by other neuroscience disciplines. With the increasing integration of functional neuroimaging, computational modeling, and theories of language and speech motor control, AOS is seen

¹ See Ziegler, Aichert, and Staiger (2012) for an in-depth cogent and critical review of current concepts and unresolved issues regarding the nature of AOS.

² See Rosenbek (2001) for an insightful review of Darley's contributions.

as an important exemplar of how the interaction between language and speech production can break down (Ziegler, Aichert, & Staiger, 2012). Furthermore, in the last two decades, the emergence of better specified models of phonologic and phonetic encoding has provided theoretical support for more explicit and narrower diagnostic criteria for AOS (McNeil, Robin, & Schmidt, 1997, 2009; Wambaugh, Duffy, McNeil, Robin, & Rogers, 2006a, 2006b). These more explicit criteria help focus debate and investigations of their validity (e.g., Staiger, Finger-Berg, Aichert, & Ziegler, 2012), and they increase confidence in studies that claim to be studying the disorder (McNeil, Pratt, & Fossett, 2004).

Neurobiological Underpinnings and Modeling Influences

Attempts to localize motor speech programming and AOS have probably forever drifted away from the unfulfilled classical search for a single responsible structure. There now is general consensus that speech is a product of actions within a distributed interactional network of cortical and subcortical structures and pathways that include, at the least, left premotor cortex, posterior inferior frontal lobe, supplementary motor area, sensorimotor cortex, auditory cortex, insula, basal ganglia, thalamus, cerebellum, and the parietal-frontal dorsal pathway (Bohland, Bullock, & Guenther, 2009; Ghosh, Tourville, & Guenther, 2008; Poeppel, Emmorey, Hickok, & Pylkkänen, 2012; van der Merwe, 2009). It is very unlikely that lesions in any one of these areas can reliably cause AOS, and leading candidate areas (posterior inferior frontal lobe, precentral gyrus of the insula) have not yet produced a clear winner(s) (cf. Dronkers, 1996; Hillis et al., 2004). Even if damage to a single or several structures can be reliably associated with AOS, how such damage explains specific features of AOS has yet to be determined (Miller, 2002).

There have been important changes in psycholinguistic and computational models of phonological encoding and speech programming, and increasingly sophisticated structural and functional neuroimaging are facilitating their biologic validation and refinement (Bohland et al., 2009). Many models are underspecified relative to articulation and prosody, but several models have contributed substantially to concepts of the speech production process—for example, the Nijmegen model/Word-form Encoding by Activation and VERification (WEAVER) model (Levelt, Roelofs, & Meyer, 1999; Roelofs, 1997), the Directions Into Velocities of Articulators (DIVA) and Gradient Order DIVA (GODIVA) models (Bohland et al., 2009; Peeva, Guenther, Tourville, Nieto-Castanon, & Anton, 2010), the state feedback control models (Hickok, 2012), and Ziegler's (2009) nonlinear probabilistic phonetic code model. For example, DIVA, the most thoroughly specified and investigated adaptive computational model of speech control, has generated data consistent with a number of kinematic and acoustic attributes of speech (Guenther, Ghosh, & Tourville, 2006). The GODIVA model, which interfaces with DIVA, links data from functional imaging and lesion studies to specific linguistic and motor components of the model, thus helping to validate or refine its plausibility. These efforts can help frame studies of communication disorders, including AOS. For example, in the GODIVA model, the inaccurate articulation associated with AOS could reflect either damage to motor programs or defective selection of motor programs, with each alternative localized

differently in the model's neural architecture (Bohland et al., 2009). Such hypotheses are beginning to foster efforts to examine speech planning and preparation in people with AOS using online reaction time methods (e.g., Maas & Mailend, 2012; Strand, 1986). These model-to-clinical relationships are bidirectional because clinical analysis of AOS breakdowns may help test model validity and subsequent refinements.

“The reasoning about the relationship of abstract representations of word forms to their phonetic counterparts is currently undergoing a substantial change” (Ziegler et al., 2012, p. S1486). Models associated with articulatory phonology (such as those proposed by Browman & Goldstein, 1992; Goldstein, Pouplier, Chen, Saltzman, & Byrd, 2007) propose an interactive, boundary-blurring interface between phonological and phonetic encoding processes (Goldrick & Blumstein, 2006) in which “abstract” phonemes have physical properties (gestural scores) that are associated with speech movement patterns (Ziegler, 2009). Thus, the frequent perceptual challenge of separating AOS from phonological errors may partly reflect the complexities of “an integrated hierarchical action system whose higher order components have emerged from and are rooted in the physical conditions of speaking” (Ziegler et al., 2012, p. S1498). The implications of this line of thought for explanations of errors in AOS have already received attention (e.g., Laganaro, 2012).

In the last half-century, theories about the nature of AOS and its neurological substrates have evolved. Testable hypotheses generated by neurobiologically valid models of normal speech production, and a better specified definition and model of AOS and its core behavioral features, will converge in the next decade (Ballard, Tourville, & Robin, 2014). We expect this convergence to yield a more refined theory of the disorder that is consistent with its clinical manifestations and its underlying structural and functional substrates.

Diagnosis—The Search for Unique Patterns of Behavior or Neurobiology

The behaviors, biology, and physiology by which AOS can be identified have in large measure been influenced by Darley's initial characterization of the disorder as an “apraxia” in the tradition of Liepmann (1900). Liepmann (1900, 1913) and many others since have used the criteria that the diagnosis of an apraxia could be made only when (1) the basic *sensorimotor integrity* of the system was shown to be intact, (2) the *underlying knowledge or representations* for the action were shown to be intact, (3) the *intention* to move was clearly demonstrated, and (4) the action could be performed *automatically* while being impaired when produced volitionally. These criteria have had a profound influence and have perhaps constrained research on AOS since its identification by Darley in 1969. Remnants of this influence are especially evident from the 1970s and 1980s. During these two decades, studies were conducted on oral-sensory functions (Rosenbek, Wertz, & Darley, 1973), visual and auditory perception (Halpern, Keith, & Darley, 1976; Johns & Darley, 1970; Shewan, Leeper, & Booth, 1984), the underlying linguistic knowledge, and differences between automatic and volitional speech (Deal & Darley, 1972; Johns & Darley, 1970). Other researchers attempted to describe the behaviors that united this group, and the search for the underlying mechanisms of the movement disorder began in earnest. Darley's clinical intuition was that this speech production deficit impaired individuals

who were neither aphasic nor dysarthric. However, without clear a priori criteria for participant selection, group assignment for study relied on the theoretical supposition about the nature of phonological errors in persons with aphasia (PWA) as much as it did about the underlying nature of the movement disorder. That is, it was assumed that PWA would show comparable deficits in comprehension and in production, and if they did not (especially if no phonological comprehension deficits could be demonstrated), then the participants were indeed apraxic as long as they did not have dysarthria. Models of phonological encoding such as those of Dell (1988) or Nadeau (2001) that could account for speech sound-level production errors in the absence of comparable errors on the input side were not yet discussed, and evidence for a language-level disorder required the demonstration of comparable deficits in both perception/comprehension and production. This led to selecting participants for the study of AOS who were, without a doubt, likely aphasic as apraxic—leading to a database that described phonological behaviors in addition to those that could be legitimately attributed to motor planning/programming impairments.

Circumstantial, corroborative evidence for a confounded database in the AOS literature is apparent in Halpern et al.'s (1976) study. Designed to investigate the phonological production errors in PWA, participants were excluded if they

showed groping, off target highly inconsistent articulatory errors—primarily substitutions, additions, prolongations and repetitions—in attempting to target words in the context of islands of fluent speech, these errors being especially evident on repetition tasks and increasing incidence with increase in length of word. (Halpern et al., 1976, p. 366)

They found that the great majority (93%) of their PWA did not demonstrate phonological errors, and those who did were accounted for by word-level errors (75%). As discussed by McNeil et al. (2004), abundant evidence is available that PWA, especially those labeled as having “conduction aphasia,” do make frequent phonemic errors. It is likely that this finding is the result of having included the phonological group with the AOS group that was so carefully eliminated from the study. Indeed, the elimination criteria are those that characterize individuals with phonological paraphasia (McNeil et al., 2009). Martin's (1974) objection to the term AOS (not to its existence) as applied to the groups being studied raised this possibility and motivated a number of studies designed to demonstrate that the groups being studied were indeed not aphasic (e.g., Square, Darley, & Sommers, 1981; Square-Storer, Roy, & Hogg, 1990). It also prompted a search for and study of groups that did not have co-occurring pathologies (McNeil & Adams, 1990; McNeil, Weismer, Adams, & Mulligan, 1990; Square et al., 1981) from which defining characteristics could be derived.

Studies of speech production impairments that preceded Darley's identification of AOS as a separate syndrome from aphasia and dysarthria undoubtedly included participants who were apraxic—though labeled as aphasic (e.g., Shankweiler, Harris, & Taylor, 1968). Johns and Darley (1970) conducted the first study published with participants diagnosed

with AOS. In this study, normal control and dysarthric groups were compared with a group with AOS. The selection criteria for the group with AOS were described as follows: "In each case the identification of the communication problem as apraxia of speech was made by one of the staff consultants in speech pathology after thorough examination of speech and language functions" (Johns & Darley, 1970, p. 559). Although no identification criteria were specified, the study compared perception with production (more production errors were evident in the participants with AOS and dysarthria than in the control participants), and better automatic than volitional production was reported. Additionally, the apraxic group performed with greater error inconsistency and unpredictability of error pattern than the dysarthric group. They also reported that unlike the dysarthric group, the "apraxic participants as a group made markedly fewer articulatory errors and gained in overall intelligibility when they read faster" (Johns & Darley, 1970, p. 579). Perhaps most revealing about the nature of the impairment in the AOS group was their characterization of their connected speech:

These subjects as a group did a creditable job of miming secondary stutterers, both acoustically and behaviorally. They sometimes circumlocuted and substituted words. One 21-year-old man said, "When I was a june, when I was a ju-june, when I was a june-in the eleventh grade . . ." They anticipated difficulty, made false starts, repeated, and blocked. They perseverated on phonemes, syllables, words and phrases. (Johns & Darley, 1970, p. 581)

This description implicates phonological, lexical, and sentence-level language mechanisms that are not easily accounted for by any motor planning, programming, or execution model of speech production. These and other characteristics of the 10 individuals identified as apraxic in this initial investigation became the descriptors and criteria for group membership, formalized first by the Apraxia Battery for Adults test by Dabul (1986). From the inventory of articulation characteristics of AOS from this battery, only two characteristics appear to be unique to AOS (abnormal prosody and the intrusive schwa between syllables or within consonant clusters), and some features appear to be unique to phonemic paraphasia (i.e., phonemic anticipatory, perseverative, and transposition errors). Dabul's (1986, 2000) credible list of observable behaviors that characterize AOS provide criteria for identifying phonological paraphasias and for identifying AOS (see McNeil et al., 2004, for additional discussion of this issue). Subsequent to Dabul's influential AOS battery, Wertz et al. (1984) provided a comprehensive summary and interpretation of the literature on AOS to that point in time, and they identified the four most salient features of AOS: (1) effortful, trial-and-error groping of articulatory movements and attempts at self-correction; (2) dysprosody unrelieved by extended periods of normal rhythm, stress, and intonation; (3) frequent articulatory errors (predominated by sound substitutions); and (4) articulatory variability. Between 1984 and 1997, these identified characteristics (and those derived from Dabul's, 1986, battery) became the primary criteria for the majority of studies selecting AOS participants for study and for treatment.

McNeil et al. (1997; and, subsequently, McNeil et al., 2004, 2009) proposed a different set of behaviors that better differentiate AOS from phonemic paraphasia. Importantly, it is the specific perceptually derived *cluster* of behaviors that is claimed to differentiate AOS from its clinical neighbors because it is recognized that many of the single characteristics overlap with both phonological paraphasia and with dysarthria. Nonetheless, it was proposed that the presence of (1) sound distortions (including distorted sound substitutions), (2) extended segment durations (realized as slow speech with lengthened consonants and vowels), (3) extended intersegment durations (realized as sound, syllable, and word segregation), and (4) prosodic deficits that may be at least in part the result of extended segment and intersegment durations—along with some of the characteristics shared with aphasia (e.g., trial-to-trial variability, trial-and-error articulatory searching or groping behavior, increased errors with increased word length, and increased speech demands such as that required with increased rate)—yield a pattern of speech that is unique and perceptually identifiable as AOS.

In 2006, a consensus group convened to analyze treatment studies in AOS (Wambaugh et al., 2006a, 2006b). In their evaluation of 59 treatment studies, the group first examined the evidence supporting the diagnosis of AOS for each study using a five-level descriptive scale. Level 1 specified that all primary characteristics consistent with the definition and criteria adopted from the core features of AOS identified by McNeil et al. (1997) were described. Level 2 described all primary characteristics consistent with the definition of AOS and some speech characteristics that may have been attributable to aphasia or dysarthria. Level 3 described most of the primary characteristics consistent with these criteria in addition to the identification of appropriate exclusionary behaviors. Level 4 evidence provided incomplete/inadequate description of the discriminative characteristics of AOS, without exclusionary behaviors. Level 5 evidence provided only the label, without a description of characteristics consistent with that diagnosis and/or with a description of behaviors that were contradictory to the criteria for AOS. With these criteria for participant identification, the authors concluded that only one of the studies provided Level 1 evidence in the characterization of participants. Nine studies were rated with Level 2 evidence, and the rest of the studies (83%) were judged to have provided inadequate justification for the diagnosis of AOS. This carefully considered evaluation of the literature adds to the evidence that the literature on AOS is likely constructed on an admixture of participants with AOS and with other speech production pathologies. Subsequent to these guideline documents, McNeil et al.'s (1997) description of AOS has been adopted frequently by researchers, at least in the United States, and continues to be frequently used for AOS diagnosis. It should be noted that they have not been adopted universally and that they have not been subjected to rigorous scientific study. Indeed, the kernel characteristics for the diagnosis of AOS (identified by Wertz et al., 1984) that have been argued to be inadequate for AOS diagnosis continue to be used to select AOS participants in some studies (Dronkers, 2004; Richardson, Fillmore, Rorden, LaPointe, & Fridriksson, 2012).

Josephs, Duffy, and colleagues are currently exploring additional criteria for the identification of AOS that co-occur or are the sole speech/language feature in degenerative neurologic disease. In one important study, Josephs et al. (2012) characterized primary

Table 9.1. Prevalence (highest to lowest) of speech characteristics identified in progressive apraxia of speech (Josephs et al., 2012, p. 2).

No.	Feature
1	Slow overall speech rate ^a
2	Lengthened intersegment durations (between sounds, syllables, words, or phrases; possibly filled, including intrusive schwa) ^a
3	Increased sound distortions or distorted sound substitutions with increased utterance length or increased syllable/word articulatory complexity
4	Syllable segmentation within words >1 syllable ^a
5	Sound distortions ^a
6	Syllable segmentation across words in phrases/sentences ^a
7	Audible or visible articulatory groping; speech initiation difficulty; false starts/restarts ^b
8	Lengthened vowel and/or consonant segments ^a
9	Distorted sound substitutions
10	Deliberate, slowly sequenced, segmented, and/or distorted (including distorted substitutions) speech sequential motion rates in comparison with speech alternating motion rates ^b
11	Increased sound distortions or distorted sound substitutions with increased speech rate
12	Distorted sound additions (not including intrusive schwa)
13	Sound or syllable repetitions
14	Sound prolongations (beyond lengthened segments) ^b
15	Inaccurate (off-target in place or manner) speech alternating motion rates (as in rapid repetition of “puh puh puh”) ^b
16	Reduced words per speech breath group relative to maximum vowel duration

Note. Features are ordered from most to least prevalent among the participants in this study. Features 1–5 were present in all 12 participants. All features were present in at least one participant. Both prosodic and articulatory abnormalities are captured in several of the listed features.

^aCan also be present in spastic dysarthria (only two participants had unequivocal spastic dysarthria).

^bCan also be present in aphasia, but none of the 12 participants were otherwise aphasic.

progressive AOS by listing the prevalence of 16 signs derived from 12 participants who met their extensive neurological and behavioral inclusion and exclusion criteria for isolated primary progressive AOS. Table 9.1 shows the hierarchy of perceptually identified speech production characteristics observed in these participants. They identified (1) slow overall speech rate, (2) lengthened intersegment durations, (3) increased sound distortions and distorted sound substitutions with increasing target length, (4) syllable segmentation within words greater than one syllable in length, (5) sound distortions, and (6) syllable segmentation across words in phrases/sentences as the six most frequently occurring features. Importantly, they noted that many of the 16 features can also occur in other sound-level speech production pathologies, including aphasia and spastic dysarthria, and five of the six most frequently occurring signs were also identified as perceptual signs of spastic dysarthria. This hierarchy of observed signs not only provides converging evidence

for the kernel signs composing the perceptually identifiable AOS cluster but also suggests that AOS may manifest with the same set of behaviors regardless of underlying etiology.

In summary, the diagnosis of AOS has an untidy history that was confounded by previously unrecognized and inaccurate assumptions about the nature of phonological errors. These assumptions led to inclusion of participants into studies that defined the diagnostic characteristics of the disorder. With advanced models of phonological processing and continued refinement of models of speech motor control, the behavioral characteristics of AOS have been refined and appear to form a coherent set of criteria from which AOS can be reliably diagnosed. Recent behavioral and neurological evidence from the study of progressive neurologic disease has provided converging evidence for these criteria. However, much work remains to be done in the development of refined models of speech production at all levels of organization and in the verification of the behaviors that provide sensitive and specific behaviors on which the diagnosis will be made.

Assessment of AOS

Assessment of AOS has many purposes, including diagnosis, determining severity of impairment and activity limitation, estimating prognosis for return of function and possibly return to work, and measuring unassisted and treatment-related change (World Health Organization, 2001). The World Health Organization's (2001) *International Classification of Functioning, Disability, and Health* (now the *International Classification of Functioning*) encourages clinicians to evaluate individuals within four domains: *Body Structure, Function* (i.e., the impairment), *Activity*, and *Participation*. In acquired motor speech disorders, assessing the Body Structure and Function domains allows identification of craniofacial or other neuromuscular anomalies across the subsystems of respiration, phonation, resonance, and articulation. In the Activity domain, one considers speech intelligibility and perceptions of communication success in activities of daily communication. Finally, in the Participation domain, degree and quality of involvement across relevant social communication contexts reflect the impact of the communication impairment on the person's quality of life and emotional well-being.

Speech-language pathologists have extensive training in the assessment of bodily structure and function as well as activity limitation, and well-established resources are available to guide this assessment (e.g., Duffy, 2013; Freed, 2012; McNeil et al., 2009; Yorkston & Beukelman, 1981). Although there has been debate over the need to assess both nonspeech and speech motor behaviors, we maintain that a more comprehensive assessment allows for a richer understanding of the concomitant disorders (e.g., dysarthria or orofacial apraxia) that may be present and possibly influence treatment programs designed to address AOS.

As discussed earlier, some consensus has been reached on the most common signs and symptoms of AOS that result from spatial and temporal movement errors during speaking. These errors fall into two main categories: (1) segmental errors that affect the quality of the speech sounds produced, perceived as distortions or distorted substitutions of consonants and vowels as well as extended segment durations, and (2) suprasegmental

errors that affect the prosodic contour of speech, including pauses or hesitations within and between segments and the perception of more equal stress across adjacent syllables within and across words, likely associated with extended durations of segments.

Currently, the predominant method for assessing presence, type, and degree of errors is perceptual judgment of speech in single words and connected speech tasks. Perceptual analysis is critical for identifying deviations from normal-sounding speech and the impact of the disorder on speech intelligibility and comprehensibility (Duffy, 2013; Yorkston, Beukelman, Strand, & Hakel, 2010). A range of assessments addressing each of these areas allows for the evaluation of the Body Function, Activity, and Participation domains to obtain information necessary for designing client-centered treatments that target the improvement of speech motor skills in meaningful and motivating contexts (Kleim & Jones, 2008). Such perceptual assessments include articulation tests, broad and narrow transcription of connected speech samples, comprehensive motor speech examinations that test nonspeech and speech-like behaviors as well as speech production over a range of task difficulties (e.g., Duffy, 2013; Freed, 2012), and intelligibility tests (e.g., Assessment of Intelligibility of Dysarthric Speech; Yorkston & Beukelman, 1981) that reveal specific patterns or clusters of behaviors (e.g., McNeil et al., 1997, 2009).

It is well known, however, that perceptual analyses of speech are susceptible to influences that can undermine their reliability (Kent, 1996; Kent, Weismer, Kent, & Rosenbek, 1989), such as clinician expertise and perceptual drift. This, in turn, affects the speech-language pathologist's confidence and objectivity in quantifying the influence of physiological changes or behavioral intervention on recovery of speech production skills. Although these threats require attention, they do not invalidate perceptual analyses. Indeed, recent studies have reported high interrater agreement on some perceptual measures that differentiate individuals with AOS plus aphasia from those with aphasia alone and from healthy controls. For example, Haley, Jacks, de Riesthal, Abou-Khalil, and Roth (2012) found individuals with AOS to have (a) a higher number of words with segmental distortions (i.e., sounds for which "a diacritic mark other than prolongation would have been used in narrow phonetic transcription," p. S1507); (b) a higher number of words with segmental prolongation (i.e., sounds "perceived to have been produced with longer than normal duration," p. S1508); and (c) a higher rate of sequential inconsistency when repeating multisyllabic words five times in a row (i.e., three or more different productions for a given word). Interrater reliability for these measures was high at 0.87 and 0.89 for judging prolongations and distortions, respectively, and 0.98 for inconsistency. This high reliability is likely achieved by having raters simply score the number of sounds perceived to be distorted rather than attempting to narrowly transcribe each distortion, which was more commonly the practice in earlier studies that struggled with reliability (McNeil et al., 1997). Additional support for modern approaches to transcription of speech sound distortions for describing and diagnosing AOS is the finding that perceptual judgment of speech intelligibility for single syllable word production correlates very highly with broad transcription of phoneme distortions in AOS, providing that multiple raters are used to obtain consensus intelligibility ratings (Haley & Martin, 2011). There are indeed important clinical reasons to adhere to perceptual measures as the gold standard.

Motivated in part by concerns about interrater reliability of transcription methods, as well as the considerable time required to collect perceptual-based scores and judgments from multiple raters, there is a long history in the AOS research literature supplementing perceptual measures with both acoustic and physiological measures of speech production. Although these instrumental measures require solid evidence of their direct mapping onto perceptual measures to replace them, such measures are appealing because of their typically high interrater reliability (>0.90) and the potential to automate all or part of the measurement process, thus saving time and negating the need for multiple raters. They can at times also provide additional insight into the underlying kinematic or physiologic mechanisms for the perceived abnormalities.

Numerous acoustic measures have been used to capture both segmental (phonetic) and suprasegmental (prosodic) features of speech (see Baken & Orlikoff, 2000), and many of these measures have been used with the AOS population (Kent & Rosenbek, 1983). Examples of acoustic measures that map onto the common perceptual errors of AOS are provided in Table 9.2. Although some acoustic measures of segmental distortions have been tested (e.g., Forrest, Weismer, Milenkovic, & Dougall, 1988), they may be perceived as more complex and arduous to collect than the current method of perceptually based transcription. At this time, it is likely that perceptual measures will remain the clinical gold standard for examining segmental distortions. However, measures of prosody in the assessment of AOS both for diagnosis and for monitoring change over time are well-suited to acoustic measurement.

Acoustic measurement of prosodic features—such as extended segment, syllable, and word durations and pause durations as well as variability of loudness and pitch—is relatively straightforward and is easily implemented in freeware such as Praat (Boersma, 2001; <http://www.praat.org>). This is fortunate given the observation that temporal prosody in AOS “has emerged as the most distinctive [feature] for differential diagnosis” (Haley et al., 2012, p. S1503; also see Ballard, Savage, et al., 2014; McNeil et al., 1997; Murray, McCabe, Heard, & Ballard, 2015; Vergis et al., 2014). The pairwise variability index of vowel duration—which quantifies degree of lexical stress contrastiveness in multisyllabic words (Ballard, Robin, McCabe, & McDonald, 2010; Ballard, Savage, et al., 2014; Courson et al., 2012; Vergis et al., 2014) and syllable durations within sentences (Haley et al., 2012), along with the median pause duration and variability of pause duration in connected speech (Ballard, Savage, et al., 2014; McKechnie et al., 2008)—has been shown to differentiate individuals with AOS plus aphasia from those with aphasia alone and from healthy controls. Automated software routines have been developed for all of these measures (e.g., de Jong & Wempe, 2009; Shahin, Ahmed, & Ballard, 2012; Vogel, Fletcher, & Maruff, 2010; Vogel, Shirbin, Churchyard, & Stout, 2012), and significant correlations between perceptual judgments and acoustic measures support their use. The measure of syllable duration in sentences correlated highly with judgments of overall prosody (0.78), slow speech rate (0.75), and restricted pitch range (0.73; Haley et al., 2012). The pairwise variability index of vowel duration in multisyllabic words has been shown to correlate highly with judgments of “goodness” of the stress pattern produced (0.80–0.91; Ballard et al., 2010).

Table 9.2. Some acoustic measures that capture features associated with the perception of segmental and suprasegmental errors in apraxic speech, along with example references reporting use of the measure(s) for individuals with apraxia of speech.

Perceptual feature(s)	Acoustic measure	Movement error	Reference
Segmental errors			
Plosive and fricative distortions due to voicing errors	Voice onset time	Mistiming of phonatory and supralaryngeal gestures	Kent & Rosenbek (1983); Ballard et al. (2007)
Fricative distortions ^a	Spectral average, tilt, and peakedness	Misplacement/mis-shaping of tongue relative to palate	Forrest et al. (1988); Tjaden & Turner (1997)
Vowel distortions	Formant frequencies for F1 and F2	Mis-shaping of the tongue movement	Haley et al. (2001)
Vowel distortions due to <ul style="list-style-type: none"> • Increased durations • Failure to reduce vowel length as word length increases • Disproportionately increased duration in unstressed syllables^b 	Vowel duration	Prolongation of movement gesture	Courson et al. (2012); Haley et al. (2012); Haley & Overton (2001); Marquardt et al. (1995); Strand & McNeil (1996)
Reduced coarticulation; slowed syllable transitions; syllable segmentation ^b	Formant frequencies and linear prediction reflection coefficients	Reduced overlap of movement gestures	Whiteside & Varley (1998); Ziegler & von Cramon (1986)
Variability of phoneme production	Voice onset time	Unstable timing of laryngeal–supralaryngeal movements	Ballard et al. (2007); Wambaugh et al. (2004)
Suprasegmental errors			
Slowed rate with syllable segmentation and reduced coarticulation ^b	Duration of between-word transitions	Slowed transitions between words	Kent & Rosenbek (1983); Strand & McNeil (1996)
Syllable segmentation	Percentage of pause time and duration of pauses in reading	Hesitations or pauses between speech units	Ballard et al. (2015); McKechnie et al. (2008)
Equal and excess stress in multisyllabic words with disproportionately increased vowel duration/intensity on unstressed syllables	Relative duration/intensity of vowels within word	Prolongation of vowels; reduction in stress contrastiveness	Ballard et al. (2015); Courson et al. (2012)

^aWhere an example reference was not found for apraxia of speech, example studies of individuals with dysarthria are provided.

^bFeature/measure that may capture aspects of both segmental and suprasegmental error types; Baken and Orlikoff (2000) provided an extensive list and description of acoustic measurements of speech and voice that can be used for apraxia of speech and dysarthria.

Normative data for some of these acoustic measures are readily available (e.g., voice onset time of consonants, vowel formant frequencies), whereas for others, data are available only for small samples of healthy control comparison groups. As the ecological validity of these various acoustic measures is established through correlation analyses with related perceptual judgments, and their discriminative power among different patient populations (e.g., aphasia, dysarthria, AOS) is investigated, more comprehensive normative studies will likely emerge (e.g., Ballard et al., 2010).

Despite limited normative data on some acoustic measures, individuals can serve as their own controls for measuring change as long as those measures have been validated against their perceptual standard. With recordings and measurements taken over multiple responses and times before treatment is initiated or degeneration is observed, a stable baseline of performance can be obtained (i.e., average and standard deviation or range). Graphical presentations of these changes can be highly motivating for adults as they see their performance improving. Also, visual displays of specific features (e.g., voicing, sharpness of plosive onsets, consonant and vowel durations, vowel formants, smooth syllable transitions vs. segmentation, pitch and intensity contours) can be used during treatment to explain how a specific feature needs to change to improve speech intelligibility or naturalness, and to provide real-time or delayed feedback on production attempts. Treatment-related or degenerative-induced changes can then be checked and validated with intermittent probes of segment or prosodic quality, intelligibility, and comprehensibility by unfamiliar listeners.

With the proliferation of small lightweight portable computers, tablets, and smartphones, it is remarkably easy to obtain recordings of speech at bedside or in the clinic that are of suitable quality for many acoustic analyses (e.g., Pocket WavePad, NCH Software; for software and freeware resources, see <http://www.phon.ucl.ac.uk/resource/software.php>). Combined speech recording and analysis freeware (e.g., Boersma, 2001; <http://www.praat.org>) along with automated analysis programs (e.g., Hosom, Shriberg, & Green, 2004; Vogel et al., 2012; to download the free speech theory application *icSpeech*, see <http://www.rose-medical.com>) make reliable measurement of acoustic features of speech production accessible and, perhaps for the first time, feasible with respect to skill level needed and time required for analysis.

Physiological measures of speech production have also yielded valuable information about spatial inaccuracies or temporal decoupling of articulatory movements in AOS that appear to underlie perceptual features such as distorted sounds and syllable segmentation (e.g., Forrest, Adams, McNeil, & Southwood, 1991; McNeil et al., 2010). Such approaches have potential to reveal the nature of the movement disruptions that give rise to various perceptually realized features of speech in AOS, which, in turn, can inform development of new interventions targeting the disruption or compensatory maneuvers. However, physiological measures require specialized tools such as electromyography, electropalatography, ultrasound, and electromagnetic articulography, which are not readily available in most clinics; furthermore, specialized clinical laboratories have not been developed to gather or analyze these types of data. Perhaps this is due in part to the fact that these measures, like their acoustic counterparts, require cross-validation

with perceptual measures if they are to be used as indices of impairment, severity, or improvement, and these studies have not been done.

In summary, with the strong consensus that has been realized relative to the nature of the problem and the key perceptual features of apraxic speech, its assessment has also advanced over the past two decades. This has allowed systematic experimentation that has yielded operationally defined and reliable perceptual and acoustic measures. Further work is needed to refine these measures, to identify other relevant acoustic/physiologic features of AOS, and to evaluate their differential diagnostic power with larger and more diverse patient samples.

Current Status of AOS Treatment: The Foundation

Development and testing of treatments for AOS began to appear in the scientific literature in the early 1970s, led by Rosenbek, Lemme, Ahern, Harris, and Wertz's (1973) influential investigation examining the effects of their eight-step task continuum. AOS treatment research progressed slowly but steadily over subsequent decades, and in 2006, the first AOS treatment guidelines were published as part of the Academy of Neurologic Communication Disorders and Sciences practice guidelines project (Wambaugh et al., 2006a, 2006b).

Fifty-nine AOS treatment reports, published in English, were included in the systematic review that served as the basis for these guidelines (Wambaugh et al., 2006a). Although more than half of the AOS treatment reports were case studies that lacked internal validity, an encouraging trend toward the use of single-subject experimental methods was noted by the guideline developers (Wambaugh et al., 2006a). An important component of the AOS guidelines project was objective evaluation of the quality of the evidence. The guideline developers used the American Academy of Neurology classification system to rate studies and to provide effectiveness ratings (Rutschmann, McCrory, Matchar, & the Immunization Panel of the Multiple Sclerosis Council for Clinical Practice Guidelines, 2002). On the basis of the objective evidence, it was concluded that gains in speech production could be expected to result from AOS treatment, particularly when therapy was focused on articulation.

At the time of the guidelines report, there was no single treatment that had amassed a sufficient database to warrant evaluation of that treatment alone. Therefore, similarities in rationale, therapy techniques, and treatment targets were used to group treatments into the following categories for evaluation: (1) articulatory kinematic, (2) rate and/or rhythm control, (3) intersystemic facilitation/reorganization, and (4) alternative/augmentative communication. More than half of the reports in the review involved articulatory kinematic treatments; consequently, stronger evidence was available to support their use compared with the other approaches.

Current Status of AOS Treatment: Recent Trends and Advances

An updating of the 2006 review has been completed in the form of a systematic review that includes 26 new AOS treatment investigations (Ballard et al., 2015). As with the studies included in the existing guidelines, the majority of the more recent investigations are considered to be articulatory-kinematic in nature. In addition, a few investigations have

examined the effects of rate and/or rhythm treatments, and a few studies have included alternative/augmentative communication techniques.

Earlier AOS treatment studies were devised primarily to detect the presence of a treatment effect. In contrast, movement toward designing studies to elucidate the effects of specific aspects of treatment is evident. In addition, technological innovations are beginning to be incorporated into AOS therapies. Importantly, experimental rigor is evident to a greater degree in the more current investigations. The following sections briefly address some of the emerging trends, new directions, and important developments in AOS treatment research.

A clear trend toward incorporating principles of motor learning (PML) in treatment has occurred over the past decade; this trend likely stemmed from a few preliminary, promising investigations of PML (e.g., Knock, Ballard, Robin, & Schmidt, 2000) that were included in the 2006 AOS guidelines report. Recent investigations have noted inclusion of various PML, but only a few have experimentally examined the effects of specific principles (e.g., Austermann Hula, Robin, Maas, Ballard, & Schmidt, 2008). Bislick, Weir, Spencer, Kendall, and Yorkston (2012) conducted a systematic review of evidence supporting claims that application of PLM enhances acquisition and generalization of speech skills. Five AOS treatment investigations were included, and only one investigation (with two participants) “yielded consistent results that were supportive of the implementation of PML in the rehabilitation of AOS” (Bislick et al., 2012, p. 726). The reviewers noted that further research is warranted on the basis of encouraging, albeit limited, findings.

The incorporation of technological advances in the therapy process or in treatment delivery is becoming evident. The use of instrumentation and/or computer-based technologies was reported rarely in studies in the 2006 AOS guidelines review. In comparison, approximately one third of more recent investigations have used such technologies, with applications including the following: (1) kinematic biofeedback (e.g., McNeil et al., 2010); (2) telerehabilitation (e.g., Lasker, Stierwalt, Spence, & Cavin-Root, 2010); (3) computerized therapy (e.g., Whiteside et al., 2012); and (4) transcranial direct current stimulation (Marangolo et al., 2011). With the exception of kinematic biofeedback using electromagnetic articulography, the effects of most technology-related treatment reports have not yet been replicated.

As noted previously, the recent AOS treatment literature has shown progress toward evaluating components of treatment. For example, Wambaugh, Nessler, Cameron, and Mauszycki (2012) recently evaluated the effects of repeated practice on articulatory accuracy with 10 AOS speakers. More than 40 years ago, Rosenbek, Lemme, et al. (1973) advocated “intensive and extensive” drill in the treatment of AOS, and repeated practice has become a ubiquitous component of AOS treatments (Wambaugh et al., 2006b). Wambaugh et al. (2006b) found that repeated practice alone, with the provision of limited feedback concerning production accuracy, resulted in substantial improvements in sound production accuracy for the majority of the participants. In addition to repeated practice and the aforementioned PML, other aspects of treatment that have been investigated include properties of the treatment stimuli (e.g., Aichert & Ziegler, 2008), modality of

treatment (Rose & Douglas, 2006), and treatment intensity (Wambaugh, Nessler, Cameron, & Mauszycki, 2013).

Although the preponderance of evidence supporting AOS treatment efficacy continues to be derived from articulatory kinematic treatments, rate and/or rhythm treatments continue to receive attention. Rate and/or rhythm treatments had previously been found to have only three small *n* studies with internal validity supporting their use (Wambaugh et al., 2006a). Several more recent reports have now provided additional empirical support for the use of rate and/or rhythm treatments to improve speech production for persons with AOS (e.g., Brendel & Ziegler, 2008). Brendel and Ziegler (2008) also evaluated the effects of metrical pacing therapy in comparison with a control therapy; both treatments were associated with improved segmental accuracy, but only metrical pacing therapy was associated with gains in rate and fluency. Overall, the new systematic review revealed improved experimental rigor and reporting of AOS diagnostic criteria in recent studies. A substantial body of new research has strengthened the evidence supporting the use of articulatory-kinematic and rate/rhythm control treatments (Ballard et al., 2015).

The Future

What might the coming decade yield for understanding AOS and the persons who have it? A simple prediction is that theory, diagnostic and assessment approaches, and management will be modified rather than mutate, and that currently recognized gaps in researchers' knowledge and unresolved competing approaches will drive the research. Here, we offer several nonprioritized examples of broad needs and directions, flowing from theory to diagnosis and assessment to management, many of which incorporate efforts that are already under way or that are predicted as next logical steps.

1. The focus on what is involved in speech programming will include efforts to understand the following: the motor attributes that are programmed; the units (e.g., syllables or subphonemic representations) that undergird the process; the role of factors such as syllable, word, or utterance frequency, and the subsegmental-to-rhythmic complexity of their structural properties; and the network of structures and pathways that support stored and computed operations. Better understanding of these variables will also influence methods for assessment and diagnosis.
2. The need "to overcome the hermetic division of labor between investigations of phonological and of motor speech impairment" (Ziegler et al., 2012, p. S1498) likely will be addressed. This may involve efforts to establish the degree to which phonologic operations are independent, intertwined, or inseparable from phonetic encoding. Similarly, the largely neglected issue of the perceptual and anatomic distinctiveness of AOS from unilateral upper motor neuron dysarthria may receive attention (Duffy, 2013; Ziegler et al., 2012). Results might require rethinking the origin of some of the surface features of AOS and could alter how researchers think about the disorder.
3. It is unlikely that refining models of normal speech production will by itself improve the understanding of AOS because an impaired system may not obey normal

model rules. However, such refinements plus increasingly valid and reliable clinical descriptions of AOS may lead to a more refined model of AOS that better explains its core (and related) features (e.g., the phonetic gestures most and least likely to go awry; their perceptual, acoustic, and kinematic correlates; the nature of their relationships with each other; their neural network loci). The combination of converging evidence from neurally verified models of normal and abnormal speech production, refined specification of the underlying sources of surface features, and improved behavioral description and measurement of the disorder are most likely to yield progress (McNeil et al., 2009).

4. Competing explanations for specific aspects of AOS likely will continue to receive attention. Examples include whether AOS programs are damaged, cannot be accessed, or cannot be created (Bohland et al., 2009). Additional examples include whether (a) there is reduced processing buffer capacity for programming multiple syllables or syllable constituents (Rogers & Storkel, 1999); (b) there is failure of feed-forward processes (impaired generalized motor programs) versus feedback control problems (Robin, Jacks, Hageman, Clark, & Woodworth, 2008); (c) there is differential involvement of direct versus indirect programming routes (Varley & Whiteside, 2001); and (d) there is a central, modality-independent programming problem versus a speech-specific modular problem (e.g., Ballard, Robin, & Folkins, 2003; Ziegler, 2003).
5. The possibility of AOS subtypes has been raised (e.g., Croot, 2002; Duffy & Josephs, 2012; Varley & Whiteside, 2001; Wertz et al., 1984). Assuming speech planning and programming are separable and multistage processes, and assuming that researchers are better able to identify unique components of a model of AOS, researchers may begin to generate testable hypotheses about the clinical features and localization correlates of what might become recognizable subtypes of AOS.
6. It is now well established that AOS can be neurodegenerative and sometimes unaccompanied by aphasia or dysarthria (Croot, Ballard, Leyton, & Hodges, 2012; Duffy & Josephs, 2012). It is likely that the next decade will see increased attention to neurodegenerative AOS, at least partly because it represents another source of data that may enrich the understanding of the clinical and anatomic substrates of the disorder. For example, the opportunity to observe systematic increments in impairment in individuals with progressive AOS, independent from impairments of other levels of speech production (i.e., phonological encoding and basic neuromuscular control found with flaccid dysarthria), may yield patterns of speech errors that help define unique attributes of AOS and that offer insight into a category system for determining levels of severity.
7. The next decade is likely to bring increased precision in the behavioral identification of patterns or clusters of speech behavior that will allow the differential diagnosis of AOS from its nearest clinical neighbor—phonological paraphasia. We make the following predictions that will influence or determine the diagnostic criteria for AOS: (a) Research will clarify whether errors generated at various levels of control (e.g., phonological encoding and motor planning) are unidirectional, bidirectional, or noninteractive. (b) A sufficiently large sample of individuals identified as having

AOS by a consensus of expert judges will yield statistically derived patterns or clusters of perceptually derived behaviors (in the tradition of Darley et al., 1975) for AOS. (c) With advances in brain imaging from magnetoencephalography and high-definition tractography, there is the likelihood that unique patterns of neural activity, distributed across large neural networks, will be found that instantiate unique patterns of AOS and that will put to rest the search for “the” lesion that causes it.

8. A theoretically grounded research assessment protocol with high sensitivity and specificity and that has demonstrated reliability and validity will be available in the next decade. This will likely include a set of perceptual and acoustic measurements of speech taken from a small set of well-designed speech tasks.
9. It is likely that there will be a better understanding of how performance on the select measures correlates with neurological damage and the integrity of the motor speech network. Identification of a defined set of measures will trigger large-scale normative studies that will serve to establish clear objective criteria for arriving at a diagnosis of AOS. Such consensus will lay the foundation for consistent participant selection in research studies, reducing the confusion that has plagued the AOS research literature.
10. Clinically, speech-language pathologists will be able to collect samples at bedside using freeware on their smartphone. Customized software routines will automatically extract selected and relevant acoustic measures and generate reports of performance against stored normative databases.
11. For community-based services, individuals will be able to use similar technologies to regularly record their performance on treatment tasks and to upload their speech samples to a remote server for analysis by their speech-language pathologist. This will allow interactive monitoring and adjustment of home-based practice and efficient generation of reports, including visual representations of performance on key speech measures and changes in response to treatment. Such systems are already in existence and undergoing experimental testing and refinement.
12. Robey and Schultz’s (1998) five-phase model of clinical outcome research continues to be appropriate for advancing AOS treatment. The AOS treatment research base in total appears to be generally progressing according to the model. That is, favorable outcomes have been demonstrated across a variety of AOS treatments (Phase 1), and investigators are beginning to evaluate variations in treatment protocols, participant characteristics, and treatment intensities (late Phase 1). Phase 2 research will continue to emerge, and the optimization of treatment will continue to be the focus of efficacy studies. Unfortunately, very few discrete treatments are being advanced systematically following the model, and it is imperative that this occurs. Findings with one treatment will not necessarily translate to another treatment or to AOS treatments in general. For example, if positive effects of PML are found for “Treatment A,” one cannot assume that the same effects will occur for “Treatment B.” Crucial issues such as treatment population, optimal schedule and duration of treatment, and outcome measurement are likely to be treatment-specific and thus demand methodical study with a particular treatment. This is likely to occur in the next decade.

13. Experimental control has been improving in the study of AOS treatments, and the increased rigor will contribute to a stronger evidence base. Single-case designs (SCDs) remain appropriate options for the initial phases of treatment development and testing, and will continue to compose the majority of AOS treatment evidence. Renowned experts in SCDs have proposed rigorous SCD standards, with corresponding evidence standards (Kratochwill et al., 2010). These standards represent goals toward which AOS treatment researchers should and will strive, and they will serve as a guide in the planning and implementation of SCDs.
14. Rating scales have been developed to assess the methodological quality of SCDs as a means of critically appraising the literature (e.g., Romeiser Logan, Hickman, Harris, & Heriza, 2008; Tate et al., 2008). The Single-Case Experimental Design Scale (Tate et al., 2008) is being used in the ongoing AOS guidelines review. Rating scales, such as the Single-Case Experimental Design Scale, provide a useful, reliable method for determining whether basic components of SCDs have been utilized. They do not necessarily reflect the highest level of experimental rigor that can be achieved with SCDs, and they do not generally take into account all critical aspects of evaluating treatment effects. As such, AOS treatment researchers should and will be wary of viewing rating scales as benchmarks for SCD quality. AOS treatment studies strive to meet qualitatively demanding standards, such as those described by Kratochwill et al. (2010).
15. Methods for evaluating combined SCDs have been proposed (Kratochwill et al., 2010), and meta-analyses of SCDs may have utility if sufficient numbers of scientifically sound investigations using similar therapy approaches become available. Larger *n* group investigations will certainly be needed, especially when treatments become sufficiently refined through Phase 1 and 2 investigations. In particular, comparative investigations of well-developed treatments are expected in the future, and group designs will be required. That is, there are limited SCD options for comparing treatments, and these options require clear control of generalization effects, which may or may not be achievable. Multisite studies are expected to be necessary to accomplish larger scale investigations, and we hope that they are forthcoming in the next decade.
16. A critical aspect of group research and comparative investigations is the selection of outcome measures. Outcome measures for AOS treatments are particularly problematic from many perspectives, including sensitivity, reliability, and ecological validity. It is likely that future research will focus on the development of appropriate outcome measures for documenting the effects of AOS treatments.
17. Comparisons of different AOS treatments continue to be limited in number, likely because of the early stage of development of AOS treatments as well as difficulties associated with the conduct of comparative effectiveness investigations. Given the current trajectory of treatment studies for AOS, we predict limited development in this area, although we do predict that the preparatory studies necessary for comparative effectiveness investigations will set the stage for their arrival.
18. The selection of AOS treatments to be studied in the future will likely be multifactorial. The current trends that were discussed earlier—such as PML, adjuvant therapies,

and computerized treatments—will certainly continue, and new technologies will be incorporated. It is important that advances in models of speech production and neurorehabilitation be translated into clinically applied treatment research. Finally, it is of utmost importance that concerted efforts be made to promote dissemination of treatment evidence to facilitate evidence-based practice.

References

- Aichert, I., & Ziegler, W.** (2008). Learning a syllable from its parts: Cross-syllabic generalisation effects in patients with apraxia of speech. *Aphasiology*, *22*, 1216–1229.
- Austermann Hula, S. N., Robin, D. A., Maas, E., Ballard, K. J., & Schmidt, R. A.** (2008). Effects of feedback frequency and timing on acquisition, retention, and transfer of speech skills in acquired apraxia of speech. *Journal of Speech, Language, and Hearing Research*, *51*, 1088–1113.
- Baken, R. J., & Orlikoff, R. F.** (2000). *Clinical measurement of speech and voice* (2nd ed.). Clifton Park, NY: Thomson Delmar Learning.
- Ballard, K. J., Maas, E., & Robin, D. A.** (2007). Treating voicing control in apraxia of speech with variable practice. *Aphasiology*, *21*, 1195–1217.
- Ballard, K. J., Robin, D. A., & Folkins, J. W.** (2003). An integrative model of speech motor control: A response to Ziegler. *Aphasiology*, *17*, 37–48.
- Ballard, K. J., Robin, D. A., McCabe, P., & McDonald, J.** (2010). Treating dysprosody in childhood apraxia of speech. *Journal of Speech, Language, and Hearing Research*, *53*, 1227–1245.
- Ballard, K. J., Savage, S., Leyton, C. E., Vogel, A. P., Hornberger, M., & Hodges, J. R.** (2014). Logopenic and nonfluent variants of primary progressive aphasia are differentiated by acoustic measures of speech production. *PLoS ONE*, *9*, e89864. doi:10.1371/journal.pone.0089864
- Ballard, K. J., Tourville, J., & Robin, D. A.** (2014). Behavioral, computational, and neuroimaging studies of acquired apraxia of speech. *Frontiers in Human Neuroscience*, *8*, 892. doi:10.3389/fnhum.2014.00892
- Ballard, K. J., Wambaugh, J. L., Duffy, J. R., Layfield, C., Maas, E., Mauszycki, S., & McNeil, M. R.** (2015). Treatment for acquired apraxia of speech: A systematic review of intervention research between 2004 and 2012. *American Journal of Speech-Language Pathology*, *24*, 316–337. doi:10.1044/2015_AJSLP-14-0118
- Bislick, L. P., Weir, P. C., Spencer, K., Kendall, D., & Yorkston, K. M.** (2012). Do principles of motor learning enhance retention and transfer of speech skills? A systematic review. *Aphasiology*, *26*, 709–728.
- Boersma, P.** (2001). Praat: A system for doing phonetics by computer. *Glott International*, *5*, 341–345.
- Bohland, J. W., Bullock, D., & Guenther, F. H.** (2009). Neural representations and mechanisms for the performance of simple speech sequences. *Journal of Cognitive Neuroscience*, *22*, 1504–1529.

- Brendel, B., & Ziegler, W.** (2008). Effectiveness of metrical pacing in the treatment of apraxia of speech. *Aphasiology*, *22*, 77–102.
- Browman, C. P., & Goldstein, L.** (1992). Articulatory phonology: An overview. *Phonetica*, *49*, 155–180.
- Courson, M., Ballard, K. J., Canault, M., Layfield, C., Scholl, D., & Gentil, C.** (2012). Lexical stress production in healthy and apraxic speakers of Australian English or French. *Journal of Medical Speech-Language Pathology*, *20*, 47–52.
- Croot, K.** (2002). Diagnosis of AOS: Definition and criteria. *Seminars in Speech and Language*, *23*, 267–279.
- Croot, K., Ballard, K., Leyton, C. E., & Hodges, J. R.** (2012). Apraxia of speech and phonological errors in the diagnosis of nonfluent/agrammatic and logopenic variants of primary progressive aphasia. *Journal of Speech, Language, and Hearing Research*, *55*, S1562–S1572.
- Dabul, B.** (1986). *Apraxia Battery for Adults*. Tigard, OR: CC Publications.
- Dabul, B.** (2000). *Apraxia Battery for Adults—Second Edition*. Tigard, OR: CC Publications.
- Darley, F. L.** (1969, November). *Aphasia: Input and output disturbances in speech and language processing*. Paper presented at the annual convention of the American Speech and Hearing Association, Chicago, IL.
- Darley, F. L., Aronson, A. E., & Brown, J. R.** (1975). *Motor speech disorders*. Philadelphia, PA: Saunders.
- Deal, J. L., & Darley, F. L.** (1972). The influence of linguistic and situational variables on phonemic accuracy in apraxia of speech. *Journal of Speech and Hearing Research*, *15*, 639–653.
- de Jong, N. H., & Wempe, T.** (2009). Praat script to detect syllable nuclei and measure speech rate automatically. *Behavioral Research Methods*, *41*, 385–390.
- Dell, G. S.** (1988). The retrieval of phonological forms in production: Tests of predictions from a connectionist model. *Journal of Memory and Language*, *27*, 124–142.
- Dronkers, N. F.** (1996, November 14). A new brain region for coordinating speech articulation. *Nature*, *384*, 159–161.
- Dronkers, N. F.** (2004). Brain areas involved in speech production. *Brain*, *127*, 1461–1462.
- Duffy, J. R.** (2013). *Motor speech disorder: Substrates, differential diagnosis, and management* (3rd ed.). St. Louis, MO: Elsevier.
- Duffy, J. R., & Josefs, K. A.** (2012). The diagnosis and understanding of apraxia of speech: Why including neurodegenerative etiologies may be important. *Journal of Speech, Language, and Hearing Research*, *55*, S1518–S1522.
- Forrest, K., Adams, S., McNeil, M. R., & Southwood, H.** (1991). Kinematic, electromyographic, and perceptual evaluation of speech apraxia, conduction aphasia, ataxic dysarthria, and normal speech production. In C. A. Moore, K. M. Yorkston, & D. R. Beukelman (Eds.), *Dysarthria and apraxia of speech: Perspectives on management* (pp. 147–171). Baltimore, MD: Brookes.
- Forrest, K., Weismer, G., Milenkovic, P., & Dougall, R.** (1988). Statistical analysis of word-initial voiceless obstruents: Preliminary data. *The Journal of the Acoustical Society of America*, *84*, 115–123.

- Freed, D. B.** (2012). *Motor speech disorders: Diagnosis and treatment* (2nd ed.). Clifton Park, NY: Delmar Cengage Learning.
- Ghosh, S. S., Tourville, J. A., & Guenther, F. H.** (2008). A neuroimaging study of premotor lateralization and cerebellar involvement in the production of phonemes and syllables. *Journal of Speech, Language, and Hearing Research, 51*, 1183–1202.
- Goldrick, M., & Blumstein, S. E.** (2006). Cascading activation from phonological planning to articulatory processes: Evidence from tongue twisters. *Language and Cognitive Processes, 21*, 649–683.
- Goldstein, L., Poupier, M., Chen, L., Saltzman, E., & Byrd, D.** (2007). Dynamic action units slip in speech production errors. *Cognition, 103*, 386–412.
- Guenther, F. H., Ghosh, S. S., & Tourville, J. A.** (2006). Neural modeling and imaging of the cortical interactions underlying syllable production. *Brain and Language, 96*, 280–301.
- Haley, K. L., Jacks, A., de Riesthal, M., Abou-Khalil, R., & Roth, H. L.** (2012). Toward a quantitative bases for assessment and diagnosis of apraxia of speech. *Journal of Speech, Language, and Hearing Research, 55*, S1502–S1517.
- Haley, K. L., & Martin, G.** (2011). Production variability and single word intelligibility in aphasia and apraxia of speech. *Journal of Communication Disorders, 44*, 103–115.
- Haley, K., Ohde, R. N., & Wertz, R. T.** (2001). Vowel quality in aphasia and apraxia of speech: Phonetic transcription and formant analyses. *Aphasiology, 15*, 1107–1123.
- Haley, K. L., & Overton, H. B.** (2001). Word length and vowel duration in apraxia of speech: The use of relative measures. *Brain and Language, 79*, 397–406.
- Halpern, H., Keith, R., & Darley, F. L.** (1976). Phonemic behavior of aphasic subjects without dysarthria or apraxia of speech. *Cortex, 12*, 365–372.
- Hickok, G.** (2012). Computational neuroanatomy of speech production. *Nature Reviews Neuroscience, 13*, 135–145.
- Hillis, A. E., Work, M., Barker, P. B., Jacobs, M. A., Breese, E. L., & Maurer, K.** (2004). Re-examining the brain regions crucial for orchestrating speech articulation. *Brain, 127*, 1479–1487.
- Hosom, J.-P., Shriberg, L. D., & Green, J. R.** (2004). Diagnostic assessment of childhood apraxia of speech using automatic speech recognition (ASR) methods. *Journal of Medical Speech-Language Pathology, 12*, 167–171.
- Johns, D. F., & Darley, F. L.** (1970). Phonemic variability in apraxia of speech. *Journal of Speech and Hearing Research, 13*, 556–583.
- Josephs, K. A., Duffy, J. R., Strand, E. A., Machulda, M. M., Senjem, M. L., Master, A. V., . . . Whitwell, J. L.** (2012). Characterizing a neurodegenerative syndrome: Primary progressive apraxia of speech. *Brain, 1*, 1–15. doi:10.1093/brain/aws032
- Kent, R. D.** (1996). Hearing and believing: Some limits to the auditory-perceptual assessment of speech and voice disorders. *American Journal of Speech-Language Pathology, 5*, 7–23.
- Kent, K. D., & Rosenbek, J. C.** (1983). Acoustic patterns of apraxia of speech. *Journal of Speech and Hearing Research, 26*, 231–249.

- Kent, R. D., Weismer, G., Kent, J. F., & Rosenbek, J. C.** (1989). Toward phonetic intelligibility testing in dysarthria. *Journal of Speech and Hearing Disorders, 54*, 482–499.
- Kleim, J. A., & Jones, T. A.** (2008). Principles of experience-dependent neural plasticity: Implications for rehabilitation after brain damage. *Journal of Speech, Language, and Hearing Research, 51*, S225–S239.
- Knock, T. R., Ballard, K. J., Robin, D. A., & Schmidt, R. A.** (2000). Influence of order of stimulus presentation on speech motor learning: A principled approach to treatment for apraxia of speech. *Aphasiology, 14*, 653–668.
- Kratochwill, T. R., Hitchcock, J., Horner, R. H., Levin, J. R., Odom, S. L., Rindskopf, D. M., & Shadish, W. R.** (2010). *Single-case design technical documentation*. Retrieved from What Works Clearinghouse website: http://ies.ed.gov/ncee/wwc/pdf/wwc_scd.pdf
- Laganaro, M.** (2012). Patterns of impairments in AOS and mechanisms of interaction between phonological and phonetic encoding. *Journal of Speech, Language, and Hearing Research, 55*, S1535–S1543.
- Lasker, J. P., Stierwalt, J. A. G., Spence, M., & Cavin-Root, C.** (2010). Using webcam interactive technology to implement treatment for severe apraxia: A case example. *Journal of Medical Speech-Language Pathology, 18*, 71–76.
- Levelt, W. J. M., Roelofs, A., & Meyer, A. S.** (1999). A theory of lexical access in speech production. *Behavioral and Brain Sciences, 22*, 1–75.
- Liepmann, H.** (1900). Das krankheitsbild der apraxie (motorische asymbolie) auf grund eines falles von einseitiger apraxie [The clinical picture of apraxia (motor asymbolia) on the basis of a case of one-sided apraxia]. *Monatschrift fur Psychiatrie and Neurologie, 9*, 15–40.
- Liepmann, H.** (1913). Motor aphasia, anarthria and apraxia. *Transactions of the 17th International Congress of Medicine, Section XI, Part II*, 97–106.
- Maas, E., & Mailend, M. L.** (2012). Speech planning happens before speech execution: Online reaction time methods in the study of apraxia of speech. *Journal of Speech, Language, and Hearing Research, 55*, S1523–S1534.
- Marangolo, P., Marinelli, C. V., Bonifazia, S., Fiori, V., Ceravolo, M. G., Provinciali, L., & Tomaiuolo, F.** (2011). Electrical stimulation over the left inferior frontal gyrus (IFG) determines long-term effects in the recovery of speech apraxia in three chronic aphasics. *Behavioural Brain Research, 225*, 498–504.
- Marquardt, T. P., & Cannito, M. P.** (1995). Acoustic analysis of accurate word stress patterning in patients with apraxia of speech and Broca's aphasia. *American Journal of Speech-Language Pathology, 4*, 180–185.
- Martin, A. D.** (1974). Some objections to the term apraxia of speech. *Journal of Speech and Hearing Disorders, 39*, 53–64.
- McKechnie, J., Ballard, K. J., Robin, D. A., Jacks, A., Palethorpe, S., & Rosen K.** (2008, September). *An acoustic typology of apraxic speech: Toward reliable diagnosis*. Paper presented at the 9th Annual Conference of the International Speech Communication Association, Brisbane, Queensland, Australia.

- McNeil, M. R., & Adams, S.** (1990). A comparison of speech kinematics among apraxic, conduction aphasic, ataxic dysarthric, and normal geriatric speakers. *Clinical Aphasiology, 18*, 279–294.
- McNeil, M. R., Doyle, P. J., & Wambaugh, J.** (2000). Apraxia of speech: A treatable disorder of motor planning and programming. In S. E. Nadeau, L. J. Gonzalez Rothi, & B. Crosson (Eds.), *Aphasia and language: Theory to practice* (pp. 221–266). New York, NY: Guilford Press.
- McNeil, M. R., Katz, W. F., Fossett, T. R. D., Garst, D. M., Szuminsky, N. J., Carter, G., & Lim, K. Y.** (2010). Effects of online augmented kinematic and perceptual feedback on treatment of speech movements in apraxia of speech. *Folia Phoniatrica et Logopedica, 62*, 127–133.
- McNeil, M. R., Pratt, S. R., & Fossett, T. R. D.** (2004). The differential diagnosis of apraxia of speech. In B. R. Maassen, R. Kent, H. Peters, P. van Lieshout, & W. Hulstijn (Eds.), *Speech motor control in normal and disordered speech* (pp. 389–413). New York, NY: Oxford University Press.
- McNeil, M. R., Robin, D. A., & Schmidt, R. A.** (1997). Apraxia of speech: Definition, differentiation, and treatment. In M. R. McNeil (Ed.), *Clinical management of sensorimotor speech disorders* (pp. 311–344). New York, NY: Thieme.
- McNeil, M. R., Robin, D. A., & Schmidt, R. A.** (2009). Apraxia of speech: Definition, differentiation, and treatment. In M. R. McNeil (Ed.), *Clinical management of sensorimotor speech disorders* (2nd ed., pp. 249–268). New York, NY: Thieme.
- McNeil, M. R., Weismer, G., Adams, S., & Mulligan, M.** (1990). Oral structure nonspeech motor control in normal, dysarthric, aphasic, and apraxic speakers: Isometric force and static position control. *Journal of Speech and Hearing Research, 33*, 255–268.
- Miller, N.** (2002). The neurological basis of apraxia of speech. *Seminars in Speech and Language, 23*, 223–230.
- Murray, E., McCabe, P., Heard, R., & Ballard, K. J.** (2015). Differential diagnosis of children with suspected childhood apraxia of speech. *Journal of Speech, Language, and Hearing Research, 58*, 43–60. doi:10.1044/2014_JSLHR-S-12-0358
- Nadeau, S. E.** (2001). Phonology: A review and proposals from a connectionist perspective. *Brain and Language, 79*, 511–579.
- Peeva, M. G., Guenther, F. H., Tourville, J. A., Nieto-Castanon, A., & Anton, J. L.** (2010). Distinct representations of phonemes, syllables, and supra-syllabic sequences in the speech production network. *NeuroImage, 50*, 626–638.
- Poepfel, D., Emmorey, K., Hickok, G., & Pylkkänen, L.** (2012). Towards a new neurobiology of language. *Journal of Neuroscience, 32*, 14125–14131.
- Richardson, J. D., Fillmore, P., Rorden, C., LaPointe, L. L., & Fridriksson, J.** (2012). Re-establishing Broca's initial findings. *Brain and Language, 123*, 125–130.
- Robey, R. R., & Schultz, M. C.** (1998). A model for conducting clinical-outcome research: An adaptation of the standard protocol for use in aphasiology. *Aphasiology, 12*, 787–810.
- Robin, D. A., Jacks, A., Hageman, C., Clark, H. M., & Woodworth, G.** (2008). Visuomotor tracking abilities of speakers with apraxia of speech. *Brain and Language, 106*, 98–106.

- Roelofs, A.** (1997). The WEAVER model of word-form encoding in speech production. *Cognition*, *64*, 249–284.
- Rogers, M. A., & Storkel, H. L.** (1999). Planning speech one syllable at a time: The reduced buffer capacity hypothesis in apraxia of speech. *Aphasiology*, *13*, 793–805.
- Romeiser Logan, L., Hickman, R. R., Harris, S. R., & Heriza, C. B.** (2008). Single-subject research design: Recommendations for levels of evidence and quality rating. *Developmental Medicine & Child Neurology*, *50*, 99–103.
- Rose, M., & Douglas, J.** (2006). A comparison of verbal and gesture treatments for a word production deficit resulting from acquired apraxia of speech. *Aphasiology*, *20*, 1186–1209.
- Rosenbek, J. C.** (2001). Darley and apraxia of speech in adults. *Aphasiology*, *15*, 261–273.
- Rosenbek, J. C., Lemme, M. L., Ahern, M. B., Harris, E. H., & Wertz, R. T.** (1973). A treatment for apraxia of speech in adults. *Journal of Speech and Hearing Disorders*, *4*, 462–472.
- Rosenbek, J. C., Wertz, R. T., & Darley, F. L.** (1973). Oral sensation and perception in apraxia of speech and aphasia. *Journal of Speech and Hearing Research*, *16*, 22–36.
- Rutschmann, O. T., McCrory, D. C., Matchar, D. B., & the Immunization Panel of the Multiple Sclerosis Council for Clinical Practice Guidelines.** (2002). Immunization and MS: A summary of published evidence and recommendations. *Neurology*, *59*, 1837–1843.
- Shahin, M. A., Ahmed, B., & Ballard, K. J.** (2012). Automatic classification of unequal lexical stress patterns using machine learning algorithms. In *2012 IEEE Spoken Language Technology Workshop* (pp. 388–391). doi:10.1109/SLT.2012.6424255
- Shankweiler, D., Harris, K. S., & Taylor, M. L.** (1968). Electromyographic studies of articulation in aphasia. *Archives of Physical Medicine and Rehabilitation*, *49*, 1–8.
- Shewan, C. M., Leeper, H. A., & Booth, J. C.** (1984). An analysis of voice onset time (VOT) in apraxic and normal subjects. In J. C. Rosenbek, M. R. McNeil, & A. E. Aronson (Eds.), *Apraxia of speech* (pp. 197–220). San Diego, CA: College-Hill Press.
- Square, P. A., Darley, F. L., & Sommers, R. I.** (1981). Speech perception among patients demonstrating apraxia of speech, aphasia and both disorders. *Clinical Aphasiology*, *9*, 83–88.
- Square-Storer, P. A., Roy, E. A., & Hogg, S. C.** (1990). The dissociation of aphasia from apraxia of speech, ideomotor limb, and buccofacial apraxia. In G. E. Hammond (Ed.), *Cerebral control of speech and limb movements* (pp. 451–476). Amsterdam, the Netherlands: North-Holland.
- Staiger, A., Finger-Berg, W., Aichert, I., & Ziegler, W.** (2012). Error variability in apraxia of speech: A matter of controversy. *Journal of Speech, Language, and Hearing Research*, *55*, S1544–S1561.
- Strand, E. A.** (1986). *Acoustic and response time measures in utterance production: A comparison of apraxic and normal speakers* (Unpublished doctoral dissertation). University of Wisconsin–Madison.

- Strand, E. A., & McNeil, M. R.** (1996). Effects of length and linguistic complexity on temporal acoustic measures in apraxia of speech. *Journal of Speech and Hearing Research, 39*, 1018–1033.
- Tate, R., McDonald, S., Perdices, M., Togher, L., Schultz, R., & Savage, S.** (2008). Rating the methodological quality of single-subject designs and *n*-of-1 trials: Introducing the Single-Case Experimental Design (SCED) Scale. *Neuropsychological Rehabilitation, 18*, 385–401.
- Tjaden, K., & Turner, G. S.** (1997). Spectral properties of fricatives in amyotrophic lateral sclerosis. *Journal of Speech, Language, and Hearing Research, 40*, 1358–1372.
- Van der Merwe, A.** (2009). A theoretical framework for the characterization of pathological speech sensorimotor control. In M. R. McNeil (Ed.), *Clinical management of sensorimotor speech disorders* (2nd ed., pp. 3–18). New York, NY: Thieme.
- Varley, R., & Whiteside, S. P.** (2001). What is the underlying impairment in acquired apraxia of speech? *Aphasiology, 15*, 34–49.
- Vergis, M., Ballard, K. J., Duffy, J. R., McNeil, M. R., Scholl, D., & Layfield, C.** (2014). An acoustic measure of lexical stress differentiates aphasia and aphasia plus apraxia of speech after stroke. *Aphasiology, 28*, 554–575.
- Vogel, A. P., Fletcher, J., & Maruff, P.** (2010). Acoustic analysis of the effects of sustained wakefulness on speech. *The Journal of the Acoustical Society of America, 128*, 3747–3756.
- Vogel, A. P., Shirbin, C., Churchyard, A. J., & Stout, J. C.** (2012). Speech acoustic markers of early stage and prodromal Huntington's disease: A marker of disease onset? *Neuropsychologia, 50*, 3273–3278.
- Wambaugh, J. L., Duffy, J. R., McNeil, M. R., Robin, D. A., & Rogers, M.** (2006a). Treatment guidelines for acquired apraxia of speech: A synthesis and evaluation of the evidence. *Journal of Medical Speech-Language Pathology, 14*, xv–xxxiii.
- Wambaugh, J. L., Duffy, J. R., McNeil, M. R., Robin, D. A., & Rogers, M.** (2006b). Treatment guidelines for acquired apraxia of speech: Treatment descriptions and recommendations. *Journal of Medical Speech-Language Pathology, 14*, xxxv–lxvii.
- Wambaugh, J. L., Nessler, C., Bennett, J., & Mauszycki, S.** (2004). Variability in apraxia of speech: A perceptual and VOT analysis of stop consonants. *Journal of Medical Speech-Language Pathology, 12*, 221–227.
- Wambaugh, J. L., Nessler, C., Cameron, R., & Mauszycki, S.** (2012). Acquired apraxia of speech: The effects of repeated practice and rate/rhythm control treatments on sound production accuracy. *American Journal of Speech-Language Pathology, 21*, S5–S27.
- Wambaugh, J. L., Nessler, C., Cameron, R., & Mauszycki, S.** (2013). Treatment for acquired apraxia of speech: Examination of treatment intensity and practice schedule. *American Journal of Speech-Language Pathology, 22*, 1–19.
- Wertz, R. T., LaPointe, L. L., & Rosenbek, J. C.** (1984). *Apraxia of speech in adults: The disorder and its management*. San Diego, CA: Singular.
- Whiteside, S. P., Inglis, A. L., Dyson, L., Roper, A., Harbottle, A., Ryder, J., . . . Varley, R. A.** (2012). Error reduction therapy in reducing struggle and grope behaviours in apraxia of speech. *Neuropsychological Rehabilitation, 22*, 267–294.

- Whiteside, S. P., & Varley, R. A.** (1998). Coarticulation in apraxia of speech: An acoustic study of nonwords. *Logopedics, Phoniatrics, and Vocology*, 23, 155–163.
- World Health Organization.** (2001). *International classification of functioning, disability, and health*. Geneva, Switzerland: Author.
- Yorkston, K. M., & Beukelman, D. R.** (1981). *Assessment of Intelligibility of Dysarthric Speech*. Tigard, OR: CC Publications.
- Yorkston, K. M., Beukelman, D. R., Strand, E., & Hakel, M.** (2010). *Management of motor speech disorders in children and adults*. Austin, TX: Pro-Ed.
- Ziegler, W.** (2003). Speech motor control is task specific: Evidence from dysarthria and apraxia of speech. *Aphasiology*, 17, 3–36.
- Ziegler, W.** (2009). Modeling the architecture of phonetic plans: Evidence from apraxia of speech. *Language and Cognitive Processes*, 24, 631–661.
- Ziegler, W., Aichert, I., & Staiger, A.** (2012). Apraxia of speech: Concepts and controversies. *Journal of Speech, Language, and Hearing Research*, 55, S1485–S1501.
- Ziegler, W., & von Cramon, D.** (1986). Disturbed coarticulation in apraxia of speech: Acoustic evidence. *Brain and Language*, 29, 34–47.

