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Prosody-voice characteristics of children and adults with apraxia of speech

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Abstract

We address explanatory issues raised by prior findings on the prosody-voice characteristics of suspected apraxia of speech in children (AOSc). Prosody-voice patterns for 14 adults with apraxia of speech (AOS) were compared to the prosody-voice patterns of 14 children with suspected apraxia of speech and inappropriate stress (AOSci) using the same assessment instruments and analysis methods. Compared to the speakers with AOSci, speakers with AOS had significantly fewer utterances meeting criteria for inappropriate stress, and significantly more utterances meeting criteria for inappropriate phrasing and inappropriate rate of speech. Discussion focuses on the implications of these three dissociations for the psycholinguistic locus of the stress deficit in AOSci including candidate loci within linguistic, motor speech, and self-monitoring processes.

Keywords: articulation, phonology, rate, speech disorder, stress.

Introduction

Apraxia (or dyspraxia) is an inability to volitionally plan, organize, and perform movements to produce actions; this inability cannot be attributed to motor weakness or to lack of co-ordination, reduced sensation, inadequate task comprehension, attention, or memory (Liepman, 1913; Darley, Aronson and Brown, 1975; Wertz, LaPointe and Rosenbek, 1991). In the discipline of communicative disorders, these widely cited inclusionary and exclusionary criteria are used to classify apraxia of speech in adults (AOS) as a motor speech impairment. The definition's emphasis on motor movements has also provided the core explanatory rationale for suspected

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apraxia of speech in children (*AOSc*),[1] which reportedly has the same symptomatology and hence may have similar underlying deficits in motor speech processing.

In a study of AOSc that included clinical samples from six investigators, Shriberg, Aram and Kwiatkowski (1997c) proposed an alternative explanatory perspective for at least one subtype of AOSc. Analyses of the speech characteristics of 48 children with suspected AOSc indicated speech error patterns that were no different than those of 71 similarly aged children with speech delays of unknown origin. However, analysis of the prosody-voice characteristics of speakers in these two groups indicated that 52% of the children with suspected AOSc had inappropriate sentential stress (AOSci) on over 20% of their conversational speech utterances, whereas only 10% of the children with speech delays of unknown origin were found to have this type of prosodic impairment. Based on several conceptual rationales—some of which are also addressed later in the present paper—these investigators proposed that the locus of the stress deficit was most likely within linguistic-representational stages of speech processing, rather than within selection-retrieval or prearticulatory stages presumed to reflect motor speech processing immediately prior to articulatory execution (cf. Shriberg, Aram and Kwiatkowski, 1997a, figure 1). In a subsequent study using a sub-sample of children with AOSci from the Shriberg *et al.* (1997c) study series, Velleman and Shriberg (1999) found that the distributional profiles of inappropriate stress in these children were consistent with predictions from metrical theory, with errors occurring more frequently in later developing prosodic contexts. The findings were viewed as additional support for locating the stress deficit at linguistic-representational stages of speech processing, rather than at selection-retrieval or prearticulatory stages of motor speech processing.

Prosodic variables such as those identified in the AOSc studies described above have also figured prominently in characterizations of AOS (e.g., Kent and Rosenbek, 1983; Kent and McNeil, 1987; Square-Storer, Darley and Sommers, 1988; McNeil, Robin and Schmidt, 1997). On review of descriptions in this literature, however, Shriberg *et al.* (1997c) suggested that the sentential stress symptomatology that purportedly characterizes AOS was not consistent with the stress patterns observed in the study of children with AOSc. Three recent studies have reported comparative data on apraxia in adults and children.

As part of a larger study, Morgan Barry (1995a; b) examined the speech of one adult speaker with AOS, one adult speaker with dysarthria, and two 9-year-old children who were described as having 'persisting articulation disorders beyond the age of 7 years' but were unclassified relative to AOSc (Morgan Barry, 1995b: 280). Based on comparative analysis of the speech of the four speakers, both children were ultimately diagnosed as having a motor speech disorder that was more similar to apraxia of speech than to dysarthria. Prosodic production errors in the children included variable rate of speech with bursts of rapid speed in longer utterances, uneven word stress, monotonous speech, and dysfluency including inconsistent pausing, sound/syllable repetition, and articulatory searching. Similarities between the adult with AOS and the children included variability in speech rate, word-stress errors, and fluency disruptions, the last of these more apparent in the adult. A notable difference was the monotonous speech in the children versus a restricted, but not completely unchanging, pitch range in the adult speaker.

Van der Merwe and Grimbeek (1990) compared the voice onset times (VOT), vowel durations, and utterance duration patterns of four adults with AOS to those of one 10-year-old child with suspected AOSc. Findings indicated that all speakers

produced errors in VOT, increased vowel durations, and lengthened utterance durations; however, in comparison to the four AOS speakers, the speaker with suspected AOSc had less severely abnormal vowel durations and utterance durations. In a subsequent conference paper report on the same speaker with suspected AOSc and a different speaker with AOS, Van der Merwe, Mlog, and Grimbeek (1996) reported that although both speakers produced many articulatory and suprasegmental errors, the speaker with suspected AOSc self-corrected only once, whereas the speaker with AOS self-corrected 10 times. In contrast to the speaker with suspected AOSc, the adult with AOS produced frequent dysfluency behaviours (sound blocks, groping, repetitions).

The present report is an attempt to address the implications of the prosody findings summarized above toward an eventual explanatory framework for AOSc. The design compares the prosody-voice patterns of 14 adults with AOS to the prosody-voice patterns of 14 children with suspected AOSc, using the same assessment instruments and data reduction methods to quantify the types and levels of involvement of participants in each disorder group. Because the response of a developing organism to a deficit might well differ from the response of a mature organism with late onset injury to the same deficit, prosodic findings cannot be directly compared relative to models of speech processing. Nevertheless, findings indicating that the two groups have similar prosody-voice profiles—especially for sentential stress—would be interpreted as providing at least preliminary support for the hypothesis of common speech processing deficits, with consequent implications for research on explanatory models of AOSc.

Speech-prosody processing

Figure 1 is adapted from a schema used previously to organize a comparative review of theoretical approaches to AOSc (Shriberg *et al.*, 1997a, figure 1) and to speculate on the potential locus of the stress deficit reported in that study series.

Modifications in the present figure accommodate the increasingly detailed views of motor speech processing in the AOS literature. The schema is purposefully underspecified in comparison with both classical speech and speech-language processing proposals (Garrett, 1980; Bock, 1982; Shattuck-Huffnagle, 1983; Dell, 1986; Levelt, 1989) and theories of motor speech control (cf. Kent, Adams and Turner, 1996). It does not presuppose a common speech processing model that subserves both developmental and acquired speech sound disorders (cf. Locke, 1994; Bishop, 1997). Rather, by restricting the elaboration of cognitive, linguistic, speech, and motor control elements involved in speech production, this minimal schema allows focus on the topic under investigation, potentially common deficits in prosody-voice in two groups of speakers. Hence, the framework in figure 1 is solely an heuristic for literature reviews and to organize later discussion of findings.

Within decoding and encoding phases of speech production, as shown in figure 1, the three processing stages that have been implicated as the locus of articulatory-prosodic impairment in the child and adult literatures on apraxia of speech are:

(a) input processing, which includes auditory-temporal and perceptual-memorial processes;
(b) organizational processing, which includes representational forms (lexical, syntactic, and phonologic) and transformational rules (allophonic, morphologic, and morphosyntactic) that act on representational forms; and

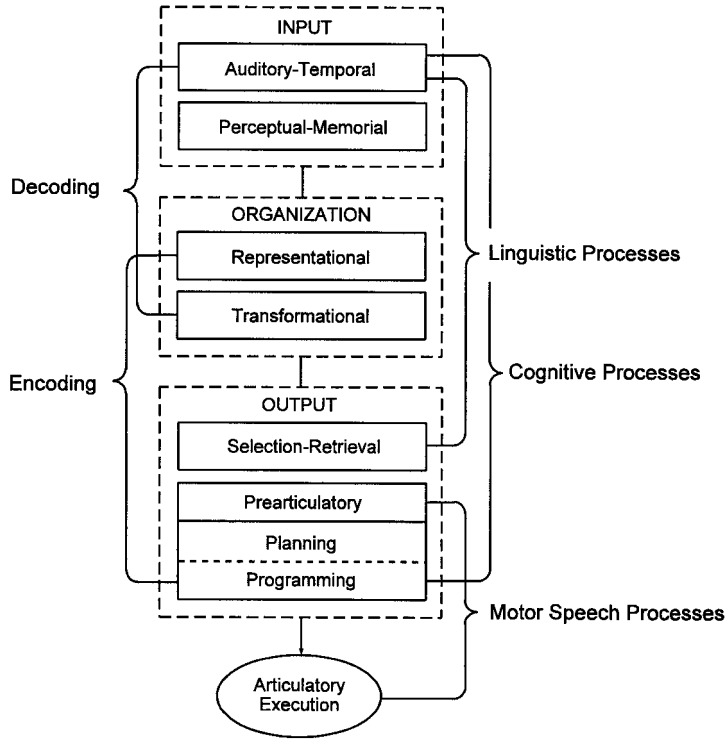


Figure 1. *Alternative loci of speech and prosody deficits in adults with apraxia of speech (AOS) and children with suspected developmental apraxia of speech and inappropriate stress (AOSci).*

(c) output processing, which includes selection-retrieval of linguistic forms and two stages of prearticulatory processing of the word, phrase, and utterance termed planning and programming.

As described by investigators of AOS and indicated by the brace on the right side of figure 1, cognitive processes are operative at all seven phases prior to articulatory execution. The brace labelled linguistic processes includes the first five phases, and the other two phases subordinated by motor speech include processes within which cognitive activities with motor goals are assembled. The dashed line separating planning from programming within prearticulatory processes reflects continued discussion of the properties ascribed to the development of the articulatory-phonetic plan versus those involved in specification of the kinematic program (e.g., Darley *et al*., 1975; Rosenbek, Kent and LaPointe, 1984; Wertz *et al*., 1991; Hageman, Robin, Moon and Folkins, 1994; McNeil *et al*., 1997; Van der Merwe, 1997; Caruso and Strand, 1999). For the present purposes, it is useful to provide a brief description of prosodic processes as they might occur at the encoding and decoding phases depicted in figure 1.

At the representational level of speech production, lexical stress (stress assignment within a word) is presumably represented in the lexical entry, as is at least partial information about speech timing (e.g., vowel lengthening before voiced obstruents (Bybee, 1994)), which also involves allophonic transformational rules. The representational and transformational levels also are presumed to decode and encode

information on phrasal and sentential prominence (stress assignment within an utterance), reflecting the stress rules of a language based on the morphological and syntactic function of each word within the utterance.

At the selection-retrieval phase, individual phonemes available at the representational level are selected and placed in the appropriate order (cf. slots-and-fillers conceptualization; Shattuck-Huffnagle, 1983) to yield a target word. Marking of word-level prosodic features, specified at the representational level and based on the linguistic function of the word, accompanies the segmental retrieval and sequencing. Impairment at the representational level would presumably produce consistent differences from the ambient segmental or prosodic form if the representation were incorrect, or inconsistent differences if correct, but not well established. Impairment in selection or retrieval processes could result in inconsistent segmental or prosodic errors, whichever the correctness or stability of the target representation.

Hypotheses about operations at the planning phase in figure 1 include processes at two subphases (Levelt, 1989; Van der Merwe, 1997; Caruso and Strand, 1999). At the first subphase, a plan may be generated to phonetically specify the targets identified at the prior phase of phonological selection and retrieval. Templates or spatial-temporal schema for familiar phonetic units (syllables, according to Levelt and Wheeldon, 1994) are retrieved from long-term memory. These templates have been hypothesized to be grossly detailed and invariant in form, corresponding to the core phonetic qualities of the unit (Kent and Rosenbek, 1983; Van der Merwe, 1997). In addition to general phonetic features, such as place of articulation and specific articulator involvement, the template includes prosodic information such as intrinsic timing elements (Van der Merwe, 1997; Caruso and Strand, 1999). As these presumably syllable-sized templates are concatenated to create a preliminary phonetic plan, the sequence of articulator movement (e.g., first tongue tip to alveolar ridge, then velar lift) is thus defined.

At the second planning subphase, the preliminary phonetic plan for each syllable is adapted to its articulatory-phonetic context within the word or phrase (Van der Merwe, 1997). Using an example by Grela and Gandour (1998), at the representational level, the word 'thirteen' is marked for stress on the second syllable. When spoken in the phrase 'thirteen women', stress changes from the second to the first syllable to be consistent with English metrical rhythm of alternating stress. For Levelt (1989), this adaptation function is carried out by a prosody generator (PG), not shown in figure 1. The PG also receives and integrates into the phonetic plan prosody information arising from the affective state of the individual. Because spoken output often differs from citation forms of words, Levelt proposes that the PG can look beyond the current single unit (e.g., 'thirteen') to upcoming units ('women') in the phonological and grammatical information it receives to make the necessary adjustments. The purview of this look-ahead mechanism is controversial: it may be a few words (Levelt) or an entire utterance (Kent and McNeil, 1987). According to Levelt, the PG spells out aspects of duration, loudness, and pitch that will affect word- and phrase-level stress patterns.

The programming phase of speech production, as shown in figure 1, is commonly considered the site for speech motor control. Its function is to select and retrieve from long-term memory the appropriate motor control mechanisms, such as a generalized motor program (GMP) for the phonetic units activated at the planning stage (Schmidt, 1988). Upon retrieval, the GMP is further specified, including parameters for range, velocity, duration, force, and the timing schedule for individual

articulator movement (Schmidt, 1988; Clark and Robin, 1998). These parameters constitute the end stage of abstract instructions, which, when effected, produce variation in stress and rate of speech.

Not depicted in figure 1 are the large number and varying routes of prearticulatory editing and internal or external self-monitoring processes that have been described in the many models of speech production. During internal editing prior to production, a speaker who is aware that a lexical, syntactic, phonologic, articulatory, or prosodic element about to be produced is not the intended target may initiate a self-correction. External self-monitoring routes deploy auditory and proprioceptive feedback systems to assess the accuracy of speech targets after they have been produced. Levelt (1989) and Motley, Baars and Camden (1983) note that the potential for editing and self-monitoring occurs at multiple levels within and between linguistic and motor speech representations, including routes that monitor speech in various social contexts. However, both Garnsey and Dell (1984) and Motley *et al.* (1983) posit that speakers monitor their own speech prior to production only at the prearticulatory level, when all elements (conceptual linguistic, and motoric) have been specified but not yet produced.

To summarize, the schema in figure 1 depicts eight potential origins or loci of segmental or suprasegmental deficits in speech production, and assumes multiple internal editing and external self-monitoring routes within and among processing loci. We will revisit these concepts throughout the following sections and later in the discussion of findings.

Acquired apraxia of speech

The clinical profile of AOS includes diagnostic markers in speech, language, prosody, and motor domains. Relative to inclusionary criteria, AOS speakers have either normal language comprehension and normal language production, or they have language comprehension that is markedly superior to language production (Kent and McNeil, 1987; Wertz *et al.*, 1991), both of which differ from language profiles for speakers with aphasia. An exclusionary criterion used in the diagnosis of AOS is the absence of clinically evident muscle weakness, slowness, or lack of co-ordination that characterizes dysarthria. Wertz *et al.* (1991: 81) proposed a cluster of four speech and prosody characteristics for AOS:

(a) obvious difficulty initiating utterances;
(b) articulatory inconsistency on repeated productions of the same utterance;
(c) effortful, trial-and-error, groping articulatory movements and attempts at self-correction; and
(d) dysprosody unrelieved by extended periods of normal rhythm, stress, and intonation.

McNeil *et al.* (1997) proposed that, because the first three features are also observed in other disorders, particularly conduction aphasia (Pierce, 1991), it is the presence of dysprosody (characterized as slow speech) in the context of the other three features that is the defining feature of AOS.

Three types of dysprosody have been documented in AOS. As described above, one salient prosodic feature is slow rate of speech. Abnormal productions that contribute to a perception of slow speech rate include lack of co-ordination or slowness of articulatory movement from segment to segment (i.e., transitionalizing

(Kent and Rosenbek, 1983; Rosenbek *et al.*, 1984; McNeil, Caligiuri and Rosenbek, 1989)), prolonged consonant and vowel articulation time (Collins, Rosenbek and Wertz, 1983; Kent and Rosenbek, 1983; Kent and McNeil, 1987; Strand and McNeil, 1996), lengthened intersegment durations (Kent and McNeil, 1987), and lengthened intraword and interword intervals (Kent and Rosenbek, 1983; McNeil, Liss, Tseng and Kent, 1990). A second dysprosodic feature presumed to characterize speakers with AOS is abnormal stress, frequently described as 'equal and even' (Darley *et al.*, 1975). A construct proposed to contribute to the percept of abnormal stress is syllable segregation—syllables perceived to be produced as separate units of similar duration and uniform intensity and frequency contours in words and phrases (Kent and Rosenbek, 1983; Kent and McNeil, 1987; Gandour *et al.*, 1989). Finally, Wertz *et al.* (1991) and other investigators have characterized AOS as lacking in normal smooth flow and rhythm, associated with groping, sound and syllable repetitions, and attempts to self-correct articulatory errors.

Several research groups have proposed that the three prosodic characteristics described above reflect processing constraints at the planning (Kent and Rosenbek, 1983; Strand and McNeil, 1996) or programming (Kent and McNeil, 1987; Seddoh, Robin, Sim, Hageman, Moon and Folkins, 1996; McNeil *et al.*, 1997) stages of speech processing depicted in figure 1. When impairments at linguistic-representationa l, transformational, or selection-retrieval levels (i.e., aphasia), or the articulatory-execution level (i.e., dysarthria), can be eliminated as sources of dysprosody in a speaker, one or both of the prearticulatory (planning, programming) stages and editing and self-monitoring routes noted previously remain as potential loci of the impairment in AOS. Kent and McNeil (1987) and Levelt (1989) suggest that, although segmental information and prosodic information are processed interdependently at the two prearticulatory stages, they are initially represented separately. Thus, there is the possibility of independent or modular impairment of either domain.

Empirical support for the prearticulatory stages as the loci of deviant prosody in AOS includes findings comparing prosodic patterns in AOS to those observed in adults with aphasia accompanied by prosodic impairment, particularly conduction aphasia. For example, McNeil, Odell, Miller and Hunter (1995) examined situations in which speakers with each disorder had notable difficulty initiating utterances. Speakers with conduction aphasia tended to produce whole-word repetitions, and AOS speakers (without aphasia) tended to produce sound/syllable repetitions. Whole-word repetitions in the context of aphasia imply deficits at lexical or even earlier stages of representation and access to those representations. In contrast, sound/syllable repetitions without aphasia imply that the deficit is not in the representation of linguistic forms or within self-monitoring processes, but rather within the sequencing and assembly processes in end-stage motor speech planning and/or programming.

Developmental apraxia of speech

Since the two classic studies that identified AOSc as a putative child speech disorder (Rosenbek and Wertz, 1972; Yoss and Darley, 1974), researchers have pointed to impairments at each of the processing phases shown in figure 1 as causes or correlates of apraxia of speech in children. Shriberg *et al.* (1997a; c) provide overviews of this literature, and book- and chapter-length reviews in the past decade are available in several sources (e.g., Marquardt and Sussman, 1991; Love, 1992; Stackhouse, 1992; Crary, 1993; Hall, Jordan and Robin, 1993; Hodge, 1994; Velleman and Strand,

1994; Ozanne, 1995; Hodge, 1998). More recent empirical studies, theoretical proposals, and internet dialogue (e.g., Apraxia Kids Listserve; Dewey, 1995; Thoonen, Maassen, Gabreels, Schreuder and de Swart, 1997; Code, 1998; Davis, Jakielski and Marquardt, 1998; McCabe, Rosenthal and McLeod, 1998; Strand, 1998; Thoonen, 1998; Velleman, 1998; Skinder, Strand and Mignerey, 1999; Strand and Skinder, 1999) continue to speculate about both the diagnostic markers that define AOSc as a clinical entity and the psycholinguistic locus and possible neurological substrates (cf. Vargha-Khadem, Watkins, Price, Ashburner, Alcock, Connelly, Frackowiak, Friston, Pembrey, Mishkin, Gadian and Passingham, 1998; Bennett and Netsell, 1998) of the speech processing deficit that is pathognomonic of the disorder.

To date, perhaps the most promising research in AOSc involves neural imaging, molecular genetic analysis, and detailed speech-language studies of a large British kindred, approximately half of whom have a speech-prosody pattern that meets most check-list criteria for AOSc (Vargha-Khadem, Watkins, Alcock, Fletcher and Passingham, 1995; Fisher, Vargha-Khadem, Watkins, Monaco and Pembrey, 1998; Lai *et al.*, 2000). One advantage of large-scale genetics studies, such as the British study and others currently underway at several research centres, is that investigators can relate phenotypes that vary considerably in expressivity and severity to alternative genotypic loci. The present study attempts to contribute to the available descriptions of this challenging child speech disorder toward an eventual explanatory account.

Method

Participants

Acquired apraxia of speech

Adult participants were obtained from two sources. Five conversational speech samples were available from the audiotape archives of the Speech Pathology Service, Veteran's Administration Hospital in Madison, WI (courtesy of J. C. Rosenbek). Each of the speakers had participated in one or more AOS studies over an approximately 16-year period (Kent and Rosenbek, 1982; 1983; Kent and McNeil, 1987; McNeil, Liss, *et al.*, 1990; McNeil and Adams, 1991; Odell, McNeil, Rosenbek and Hunter, 1991; McNeil *et al.*, 1995; Liss, 1998). A total of 12 individuals were referred to the first author from five speech-language pathology service programmes in three Wisconsin cities. The request to clinicians in these programmes was to identify speakers whose primary communication disability was AOS with minimal or no aphasia. Each of the eight referring clinicians for these nine speakers was clinically certified by the American Speech-Language-Hearing Association and had 10–35 years of experience in the diagnosis and treatment of neurogenic speech-language disorders.

The inclusionary criterion for the adult participants was a clinical diagnosis of AOS, and exclusionary criteria were the absence of dementia, dysarthria, or substantial aphasia. Confirmation of the diagnosis of AOS was made by the first author, a certified SLP with 15 years of clinical and research experience discriminating AOS from other neurogenic speech-language disorders. Using the Wertz *et al.* (1991) inclusionary criteria, referred speakers were classified as potentially apraxic if they were perceived to demonstrate any one or more of the following four impairments:

(a) difficulty initiating speech;
(b) articulation errors, including sound-segment distortions and substitutions;

(c) sound/syllable repetitions; or
(d) prosodic impairment, including slow rate and misplacement of stress.

Judgements of the occurrence and frequency of these speech-prosody characteristics were made from several sources: speakers' live- and audio-recorded responses during administration of the Apraxia Battery for Adults (ABA, Dabul, 1979), the verbal subtests from the Western Aphasia Battery (WAB, Kertesz, 1982) or the Boston Diagnostic Aphasia Examination (BDAE, Goodglass and Kaplan, 1983); and a conversational speech sample and/or spontaneous responses during the motor speech exam (Wertz and Rosenbek, 1976). Three of the 12 speakers referred to the first author were eliminated from further consideration because they failed to produce a minimum of 24 intelligible utterances in the conversational speech sample, a requirement for the prosody-voice analysis (Shriberg, Kwiatkowski and Rasmussen, 1990). A total of 13 of the 14 participants were classified as mild-moderate AOS on the ABA and the remaining speaker was classified as moderately impaired.

Supplemental tests were administered to identify conditions resembling AOS, specifically, substantial aphasia, dementia, hearing loss or dysarthria. The same basic assessment protocol was administered to all potential participants, excluding the five VA speakers who had been tested with different protocols. Criteria for the presence of aphasia followed the Darley (1982) definition. For nine of the 14 speakers, aphasia was ruled out by performance near or within criteria for normal individuals on the WAB (Kertesz, 1982), the BDAE (Goodglass and Kaplan, 1983), the Porch Index of Communicative Ability (PICA, Porch, 1967), or the Revised Token Test (RTT, McNeil and Prescott, 1978). Although five of the eventual 14 speakers scored in the range typical of aphasic individuals based on either WAB or RTT, their aphasia was not considered to be the major barrier to communication. One of these five speakers (AOS1), who scored poorly on the WAB and RTT, often spoke in full and grammatical sentences without substantial word-finding difficulty. A non-standardized language task administered to another of these five speakers (AOS14) confirmed this speaker's ability to comprehend complex three-step auditory and written commands and to name pictures. On oral expressive language tasks, the eventual 14 speakers with AOS did not produce language that was indicative of aphasia; sentences produced were normal in lexical selection, grammatical structure, and length. Performance on the Coloured Progressive Matrices (Raven, Court and Raven, 1990) indicated that the 14 participants were within the normal range on this non-language cognitive functioning task. On the Story Retelling subtests (immediate and delayed) of the Arizona Battery for Communication Disorders in Dementia (ABCD, Bayles and Tomoeda, 1993), all AOS participants tested on this measure performed above the level associated with mild dementia. Finally, all speakers, except the VA speakers who were not tested on this protocol, passed the non-instrumental hearing screening test on the ABCD administered by the first author.

Absence of dysarthria in potential AOS participants was supported by information obtained from three sources: there were no entries indicating dysarthria in the medical records, there was no mention of dysarthria by the referring clinician, and there was no perceptual evidence of dysarthria as assessed by the first author during the motor speech exam (Wertz and Rosenbek, 1976). For all adults who met the study criteria and participated, clinical examination by the first author did not indicate notable abnormal tone or weakness. However, previous instrumental studies of speakers with relatively pure AOS have indicated deficits in force and position

control (McNeil, Weismer, Adams and Mulligan, 1990; Hageman *et al*., 1994) as well as in articulatory kinematics (cf. Hough and Klich, 1987; McNeil *et al*., 1989; Forest, Adams, McNeil and Southwood, 1991; McNeil and Adams, 1991). Therefore, in the absence of instrumental analyses, the present speakers cannot with certainty be said to have normal motor tone, reflexes, and co-ordination.

Table 1 is a summary of the demographic and assessment information for the 14 AOS speakers.

The study sample includes 12 males and two females, ranging in age from 50–81 years. All were native speakers of American English. None of the participants had premorbid histories of speech-language disorders. As shown in the column titled 'MPO' (months post onset), length of time between the onset of brain damage and the speech-language assessment data used in the present study ranged from less than 1 month to 15 years. Each of the 14 speakers had a unilateral left hemisphere lesion in the distribution of the middle cerebral artery, as documented either by radiological reports or physician comments in the medical records. The most prevalent etiology was left cerebral vascular accident.

Developmental apraxia of speech

Table 2 is a summary of the demographic and assessment information for 14 children with suspected AOSci. These speakers are a subsample of 25 children with suspected AOSci who were described in Shriberg *et al.* (1997c). The identification of AOSc for each child in the AOSc sample was based on a definition of AOSc adopted by the clinician-researcher making the diagnosis. The inclusionary criterion for the AOSci group was the presence of inappropriate stress codes in at least 20% of the 24 conversational speech utterances coded for prosody-voice (the other speakers in the AOSc group produced few utterances with inappropriate stress coding). Exclusionary criteria were the absence of hearing loss and dysarthria. Prosody-voice data on 11 of the 25 children in the Shriberg *et al*. series were obtained using a preliminary version of the Prosody-Voice Screening Profile (PVSP, Shriberg *et al.*, 1990; Shriberg, Kwiatkowski, Rasmussen, Lof and Miller, 1992); they could not be included in this study because their prosody-voice data could not be directly compared to the prosody-voice data for the adult speakers with AOS included in the present study. Prosody-voice data for the 14 children with suspected AOSci included in table 2 were coded by a transcriber with extensive research experience using the most recent, standardized version of the PVSP.

The assessment information, based on performance on standardized and informal tasks, was collected by six clinical researchers. These results were forwarded to the second author using a common data sheet. The conversational speech samples met technical and linguistic criteria used in prior studies of typical and atypical speech acquisition (Shriberg and Kwiatkowski, 1980; Shriberg, Austin, Lewis, McSweeny and Wilson, 1997b). As shown in table 2, two children had cognitive involvement and most were delayed in language comprehension and/or language production. Several children had oral nonverbal apraxia. Medical records indicated that none of the 14 children had histories of developmental or acquired brain damage or neurological disease.

Analysis of the conversational speech samples

Conversational speech sampling

The speech samples for both speaker groups met criteria for conversational speech analyses developed and validated in prior work (Shriberg, 1986; 1993; Shriberg

Table 1. *Demographic and assessment data for the adult speakers with apraxia of speech (AOS)*

Participant	Age (years)	Sex	MPO[a]	Cognition	Motor	Language
AOS1	50	F	11	27[b] 14, 13[e]	44, 32[c]	57[d] 9.46[f]
AOS2	50	M	180	21[b] 13, 13[e]	–	93.60[d] 13.74[f]
AOS3	54	M	39	30[b]	48, 49[c]	14.53[g] 118[h]
AOS4	55	M	6	–	–	14.10[g]
AOS5	55	M	20	32[b] 11, 11[e]	–	96.8[d] 14.59[f]
AOS6	58	M	53	35[b] 14, 14[e]	43, 48[c]	117.50[h] 12.18[f]
AOS7	59	M	98	27[b]	47, 43[c]	14.66[g] 116[h]
AOS8	62	M	2	24[b] 14, 14[e]	44, 42[c]	96.20[d] 13.41[f]
AOS9	62	M	64	28[b]	50, 37[c]	14.33[g] 113[h]
AOS10	67	M	38	32[b] 14, 14[e]	–	96[d] 14.59[f]
AOS11	68	M	47	–	–	87.5[h] 11.72[f]
AOS12	68	F	143	34[b] 14, 13[e]	44, 47[c]	96.20[d] 12.95[f]
AOS13	71	M	3	18[b] 12, 13[e]	50, 50[c]	92.2[d] 12.14[f]
AOS14[i]	81	M	1			

Note. Dashes indicate data were not obtained.
[a] Months post onset (of brain damage).
[b] Coloured Progressive Matrices (Raven, Court and Raven, 1990).
[c] Oral and limb apraxia scores, respectively, on the Apraxia Battery for Adults (Dabul, 1979).
[d] Aphasia quotient on the Western Aphasia Battery (Kertesz, 1982).
[e] Raw scores on the Story Retelling task (immediate and delayed, respectively) on the Arizona Battery for Communication Disorders of Dementia (Bayles and Tomoeda, 1993).
[f] Raw overall score on the Revised Token Test (McNeil and Prescott, 1978).
[g] Overall score on the Porch Index of Communicative Ability (Porch, 1967).
[h] Score on the subtests of auditory comprehension, Boston Diagnostic Aphasia Examination (Goodglass and Kaplan, 1983).
[i] Formal tests were not administered.

and Kwiatkowski, 1980; 1982; 1994). Specifically, speech signals were technically adequate for narrow phonetic transcription and prosody-voice coding, and linguistic corpora were adequate for phonetic and phonologic analyses. Each participant in the AOS and AOSci groups had been engaged in a conversation with an experienced speech-language pathologist. The five adult speakers whose samples were obtained from archival records had been assessed by two clinical investigators; the first author

Table 2. *Demographic and assessment data for the speakers with suspected developmental apraxia of speech and inappropriate stress (AOSci)*

Participant	Age (years;months)	Sex	Cognition	Language Comprehension	Language Production
AOSci1	4;7	M	LN[a]	LN	LN
AOSci2	4;8	F	WNL[b]	WNL	BAL[c]
AOSci3	4;9	F	WNL	LN/WNL	BAL
AOSci4	5;4	M	WNL	WNL	BAL
AOSci5	5;4	F	WNL	BAL	BAL
AOSci6	5;7	M	WNL	WNL	BAL
AOSci7	5;10	M	WNL	LN/WNL	BAL
AOSci8	7;10	F	WNL	LN/WNL	BAL
AOSci9	8;6	M	WNL	WNL	BAL
AOSci10	9;11	M	BAL	BAL	BAL
AOSci11	12;11	M	BAL	BAL	BAL
AOSci12	13;6	M	LN	BAL	BAL
AOSci13	14;11	M	WNL	WNL	BAL
AOSci14	14;4	F	WNL	WNL	BAL

[a] Low Normal.
[b] Within Normal Limits.
[c] Below Age Level: below one standard deviation from age level on a standardized test.

was the interlocutor for each of the remaining nine subjects. Six of the conversational samples from the children were obtained by the same experienced investigator at a large Midwestern hospital, and the remaining eight samples were obtained by five clinical investigators at five research sites in North America.

Transcription and prosody-voice coding

All speech samples were transcribed and prosody-voice coded by three research transcribers with extensive experience using systems for narrow transcription (Shriberg, Kwiatkowski and Hoffmann, 1984; Shriberg and Lof, 1991; McSweeny and Shriberg, 1995; Shriberg and Kent, 1995) and prosody-voice coding (Shriberg *et al.*, 1990; Shriberg *et al.*, 1992). The transcribers were provided with only age and sex information for each audio-taped speech sample. For the 14 children with suspected AOSci, the most experienced transcriber had transcribed and coded each of the samples approximately 3 years earlier in the context of a larger study of child speech disorders.

Additional conversational speech samples from adults with neurogenic disorders were obtained by the first author to obviate possible transcription bias associated with the age of the adults with AOS in comparison to the children with AOSci, and possible bias associated with the percept of neurogenic disorder. Conversational speech samples from 11 adults with dysarthria and four adults with aphasia were obtained by the first author following the same protocol used with the 14 speakers with AOS. The age range of these speakers was similar to the age range of the speakers with AOS. The data set consisting of the randomized audiocassette tapes of all 29 adult speakers was assigned to two research transcribers for transcription and prosody-voice coding. Twenty-two of the samples were transcribed and prosody-voice coded by consensus by the two transcribers (Shriberg *et al.*, 1984). The remaining seven randomly assigned samples were transcribed by the more experienced transcriber.

Reliability

Phonetic transcription and prosody-voice coding were error checked by a research assistant and processed by a suite of programs for speech and prosody-voice analysis (Programs to Examine Phonetic and Phonologic Evaluation Records (PEPPER), Shriberg, 1986; 1993; Shriberg, Austin, Lewis, McSweeny and Wilson, 1997a; b). The suite of analyses includes a reliability program (PEPAGREE, Shriberg and Olson, 1988) that provides detailed information on transcription agreement; reliability of prosody-voice coding was hand computed. Segmental and suprasegmental reliability data for the child data were reported in Shriberg, Austin, *et al.* (1997b). Segmental and suprasegmental reliability data for each of the research transcribers in the present study have been reported in detail in McSweeny and Shriberg (1995) and updated in a recent reliability study in preparation. For the child data, interjudge prosody-voice coding agreement with the second author and another research transcriber on 28 randomly selected conversational samples from a tape archive ranged from 74% to 96% on the seven summative prosody-voice parameters to be described. Intrajudge agreement ranged from 85% to 99%. Interjudge and intrajudge samples for the transcribers who prosody-voice coded the adult samples are also in the 75–95% point-to-point agreement range on the seven summative prosody-voice variables.

Prosody-voice analyses

Detailed findings on the speech characteristics of study participants are not addressed in the present report. Speech data for the 14 children with AOSci are provided in Shriberg, Aram and Kwiatkowski (1997b; c) and speech data for 5 of the 14 adults with AOS have been reported in the previously cited studies conducted over the past two decades. The following review summarizes the major procedural elements of the PVSP method used for the prosody-voice analyses and referenced in the discussion of findings.

The PVSP is a perceptually based coding system used to profile a speaker's prosody and voice characteristics as obtained from conversational speech utterances. The first step after glossing utterances is to exclude utterances that meet criteria for the 31 exclusion codes shown at the top of figure 2. These codes concern both the adequacy of the signal for auditory-perceptual decisions and the content of each utterance relative to prosody-voice coding needs.

A total of 24 utterances that meet requirements are then coded to yield summative percentages on seven non-overlapping suprasegmental parameters: phrasing, rate, stress, loudness, pitch, laryngeal quality and resonance. Summative percentages for the seven codes are obtained from tallies on 31 subtype codes for inappropriate prosody-voice (see figure 2). Coders learn to discriminate each prosody-voice (PV) code by training practice that includes learning the perceptual criteria for each code and listening to several hundred audio-taped exemplars obtained from samples of child and adult speakers representing a wide spectrum of speech disorders, including speakers with motor speech disorders (Shriberg *et al.*, 1990).

Some of the 31 inappropriate PV subcodes shown in figure 2, themselves, have subcodes. As discussed later, for example, the subcode for inappropriate sentential stress, PV15: Excessive/Equal/Misplaced Stress, includes four types of behaviour that coders annotate as the basis for their assignment of PV15 (excessive-equal stress, misplaced stress, blocks, and prolongations). Thus, the prosody-voice analyses

Exclusion Codes

Content/Context	Environment	Register	States
C1 Automatic Sequential ___	E1 Interfering Noise ___	R1 Character Register ___	S1 Belch ___
C2 Back Channel / Aside ___	E2 Recorder Wow / Flutter ___	R2 Narrative Register ___	S2 Cough / Throat Clear ___
C3 I Don't Know ___	E3 Too Close to Microphone ___	R3 Negative Register ___	S3 Food in Mouth ___
C4 Imitation ___	E4 Too Far from Microphone ___	R4 Sound Effects ___	S4 Hiccup ___
C5 Interruption / Overtalk ___		R5 Whisper ___	S5 Laugh ___
C6 Not 4 (+) Words ___			S6 Lip Smack ___
C7 Only One Word ___			S7 Body Movement ___
C8 Only Person's Name ___			S8 Sneeze ___
C9 Reading ___			S9 Telegraphic ___
C10 Singing ___			S10 Yawn ___
C11 Second Repetition ___			
C12 Too Many Unintelligibles ___			

Prosody-Voice Codes

Prosody

Phrasing	Rate	Stress
1 Appropriate ___	1 Appropriate ___	1 Appropriate ___
2 Sound / Syllable Repetition ___	9 Slow Articulation / Pause Time ___	13 Multisyllabic Word Stress ___
3 Word Repetition ___	10 Slow / Pause Time ___	14 Reduced / Equal Stress ___
4 Sound / Syllable and Word Repetition ___	11 Fast ___	15 Excessive / Equal / Misplaced Stress ___
5 More than One Word Repetition ___	12 Fast / Acceleration ___	16 Multiple Stress Features ___
6 One Word Revision ___		
7 More than One Word Revision ___		
8 Repetition and Revision ___		

Voice

Loudness	Pitch	Quality	
		Laryngeal Features	Resonance Features
1 Appropriate ___	1 Appropriate ___	1 Appropriate ___	1 Appropriate ___
17 Soft ___	19 Low Pitch / Glottal Fry ___	23 Breathy ___	30 Nasal ___
18 Loud ___	20 Low Pitch ___	24 Rough ___	31 Denasal ___
	21 High Pitch / Falsetto ___	25 Strained ___	32 Nasopharyngeal ___
	22 High Pitch ___	26 Break / Shift / Tremulous ___	
		27 Register Break ___	
		28 Diplophonia ___	
		29 Multiple Laryngeal Features ___	

Figure 2. *Prosody-Voice Profile key for the categories and codes shown in figure 3.*

to be reported involve between-group prosody-voice comparisons at the level of summative percentages, subordinate code percentages, and, for one variable, third level subordinate codes.

Parametric descriptive statistics (means, standard deviations) were used for numeric and graphic summaries. For inferential statistical analyses, nonparametric tests (Wilcoxon-Mann-Whitney test of rank differences) were deemed necessary due to the small cell sizes, the distributional characteristics of some of the variables, the

large number of 0% and 100% scores, and the often unequal standard deviations for each group-wise comparison (Siegel and Castellan, 1988). The null hypothesis for all comparisons was that the averaged prosody-voice profiles of the two speaker groups were not significantly different, but no precedent data were available from which effect sizes could be derived to estimate the power of rejecting the null hypothesis. For the goals of the study, each of the seven prosody-voice domains was considered a separate, family-wise dependent variable. To avoid potential Type II errors due to the limited cell sizes, Bonferroni alpha level corrections were not applied to the inferential statistics used in the additional analyses in each domain.

Results

Figure 3, panel A, termed a *Prosody-Voice Profile*, is a panel from a PEPPER printout (Shriberg, 1993) that compares the group-averaged performance of the AOS and AOSci speakers.

The top section of the panel provides numeric data (means, standard deviations) for the seven prosody-voice characteristics and the lower section is a graphic display of the mean data for each speaker group. The data points in the graph are the mean percentage of utterances for each group coded as appropriate for each prosody-voice domain. The two bold horizontal dashed lines in Panel A indicate the 90% cut off for 'pass' and the 80% cut off for 'questionable pass' on the PVSP.

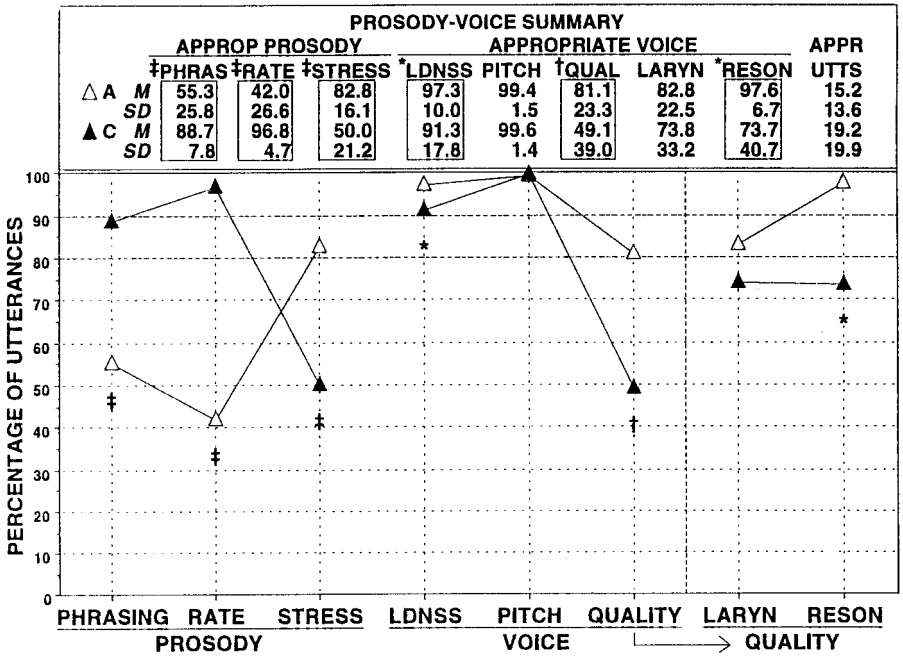
Statistically significant Wilcoxon-Mann-Whitney rank-order differences (indicated by boxes in the numeric section of figure 3, panel A, and conventional alpha-level symbols in the graphic section) were obtained for the domains of phrasing, rate, stress, loudness, quality, and resonance. The summative quality data points reflect utterances with inappropriate laryngeal quality and/or inappropriate resonance quality; the present analysis focuses individually on the two quality variables. Figure 3, panel B, provides graphic and inferential statistical findings for the 15 inappropriate phrasing, rate, and stress subcodes, including Wilcoxon-Mann-Whitney between-group statistical comparisons.

Table 3 is a per-participant summary of the summative codes for the five prosody-voice variables on which groups differed statistically, and table 4 provides grouped data on subcodes for phrasing, rate, and stress. The following reviews of group and per-participant findings for each of the five prosody-voice variables reference the information in figure 3 and tables 3 and 4.

Phrasing

As shown in the numeric and graphic sections of figure 3, panel A, AOS speakers produced a significantly lower percentage of utterances with appropriate phrasing (55.3%) compared to AOSci speakers (88.7%, $p = 0.0004$). As indicated by the Wilcoxon-Mann-Whitney test results in figure 3, panel B, AOS speakers averaged significantly more utterances with inappropriate phrasing on five of the seven inappropriate phrasing codes. All AOS speakers had one or more inappropriate phrasing codes (table 3), ranging from one instance of one code (one speaker) to more than 10 instances across several codes (six speakers). Of the 14 speakers with AOS, 12 had 20% or more of their utterances coded for inappropriate phrasing.

Additional examination of the patient records for the two AOS speakers with questionably appropriate or appropriate (i.e., $> 80\%$) phrasing (AOS1, AOS3) was



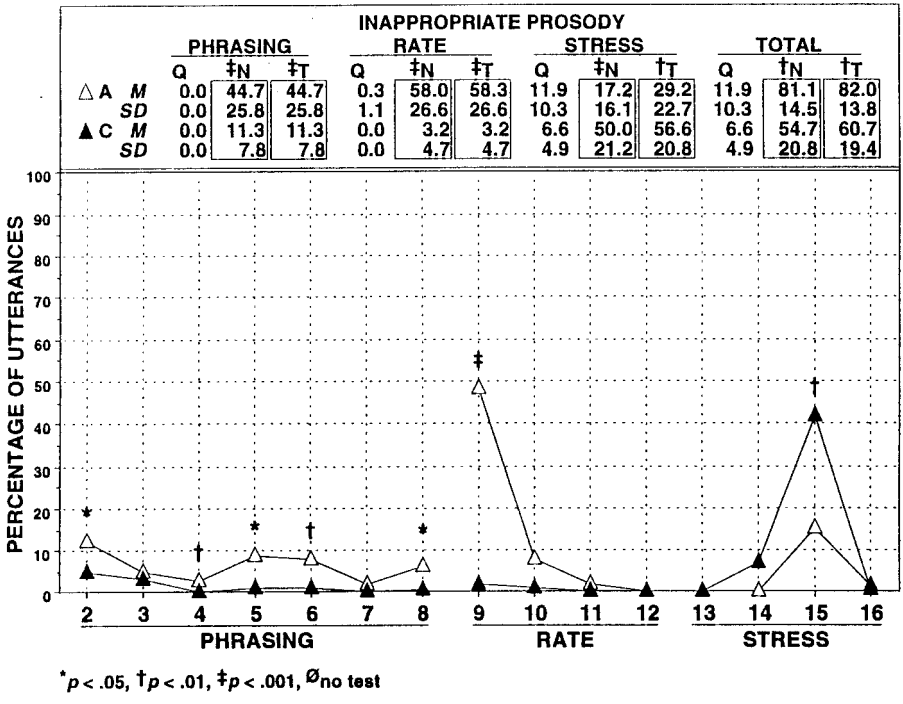


Figure 3. *Prosody-Voice Profile comparison of adult speakers with apraxia of speech (AOS [unfilled triangles]) and speakers with developmental apraxia of speech and inappropriate stress (AOSci [filled triangles]). See text for a description of the information in Panel A (top) and Panel B (bottom).*

Table 3. *Individual data for adult speakers with apraxia of speech (AOS) and child speakers with suspected developmental apraxia of speech and inappropriate stress (AOSci). The prosody-voice values are percentages of appropriate utterances in each category*

Participant	Phrasing	Rate	Stress	Laryngeal quality	Resonance quality	PCC[a]
AOS speakers						
AOS1	95.8	8	87.5	91.3	100	88
AOS2	62.5	62	100	0	100	97.1
AOS3	91.7	4	91.7	95.8	95.8	90.2
AOS4	62.5	17	50	95.8	100	94.8
AOS5	20.8	67	91.7	91.7	95.8	99.4
AOS6	66.7	46	83.3	95.8	100	96.9
AOS7	62.5	0	75	87.5	75	89.1
AOS8	41.7	79	58.3	91.7	100	90.8
AOS9	75	29	91.7	95.8	100	96.2
AOS10	45.8	46	66.7	100	100	96.6
AOS11	37.5	71	91.7	45.8	100	92.1
AOS12	75	83	100	87.5	100	99.4
AOS13	12.5	50	100	33.3	100	93.1
AOS14	23.8	33	71.4	47.6	100	95.1
AOSci speakers						
AOSci1	100	100	70	60	90	69.5
AOSci2	86.7	100	73.3	86.7	100	71.9
AOSci3	100	87.5	66.7	100	100	68.8
AOSci4	86.4	86.4	27.3	81.8	100	79.3
AOSci5	83.3	100	33.3	4.2	100	52.5
AOSci6	100	100	52.6	94.7	89.5	42.8
AOSci7	87.5	100	4.2	16.7	91.7	44.6
AOSci8	80	95	45	90	100	87.2
AOSci9	87.5	95.8	29.2	100	70.8	82.6
AOSci10	85	95	40	78.9	90	68.8
AOSci11	75	95.8	50	91.7	0	80.5
AOSci12	83.3	100	66.7	29.2	100	88.2
AOSci13	95.8	100	70.8	100	0	82.3
AOSci14	91.7	100	70.8	100	0	68.1

[a] Percentage of Consonants Correct (Shriberg, Austin, Lewis, McSweeny and Wilson, 1997a).

unrevealing. One of the two speakers (AOS3) was among the five speakers who had participated in many of the AOS studies in the past 15 years. The speech severity scores (i.e., PCC) of AOS1 and AOS3 were among the lowest of the speakers with AOS (see table 3). Scores on the cognitive and language measures for these speakers were not associated with their Phrasing scores.

AOSci speakers, both as a group (figure 3, panel B) and individually (table 3), had relatively few utterances coded as inappropriate phrasing. Three of the 14 AOSci speakers had 100% of their utterances coded as appropriate for phrasing, and only 1 of the remaining 11 children had more than five utterances (20% of total utterances) coded for inappropriate phrasing on any of the seven subcodes. On inspection, the profile of the one AOSci speaker who did produce more than 20% of utterances with inappropriate phrasing did not differ from the other AOSci speakers across other prosody-voice or speech measures, but he was one of the speakers with below average levels on the cognitive and language measures.

Table 4. *Summative data (percentage appropriate utterances) and subcode data (percentage inappropriate utterances) for speakers with AOS and AOSci on Phrasing, Rate and Stress. See figure 2 for key to subcodes*

	AOS speakers		AOSci speakers		
PVSP category	Mean %	*SD*	Mean %	*SD*	*p* value
Appropriate Phrasing	55.3	25.8	88.7	7.8	0.0004
PV2	12.3	8.7	5.1	5.8	0.0193
PV3	5.1	5.3	3.1	4.4	0.2998
PV4	2.8	3.7	0	0	0.0078
PV5	8.6	10.8	1	2	0.0214
PV6	7.9	6.8	1.2	2	0.0017
PV7	1.8	3.5	0.3	1.1	0.1473
PV8	6.3	7.6	0.6	1.5	0.0107
Appropriate Rate	42	26.6	96.8	4.7	0.0000
PV9	48.2	32	1.9	3.7	0.0000
PV10	7.7	18.6	0.9	2.6	0.1059
PV11	2.1	4.5	0.3	1.1	0.1473
PV12	0	0	0	0	1.0000
Appropriate Stress	82.8	16.1	50	21.2	0.0004
PV	0.3	1.1	0.4	1.4	1.0000
PV14	0.3	1.1	7.1	18.7	0.0631
PV15	15.1	13.8	41.5	24.2	0.004
PV16	1.5	3.5	0.7	1.8	0.705

Rate

Rate of speech in conversational contexts was considered appropriate by PVSP criteria when within normal limits for a speaker's age, dialect, and emotional state. Based on literature findings and local normative studies (cf. Shriberg *et al.*, 1990), the PVSP defines appropriate rate for children below 12 years of age as 2–4 syllables per second; for child and young adult speakers above age 12, the criterion is 4–6 syllables per second. For the more advanced ages of the adults with AOS in the present study, Duchin and Mysak's (1987) findings for rate (calculated as syllables per second including pause time) were considered appropriate reference data. Adult utterances in the current study were considered slow if their rates were below one standard deviation from the mean for their peer age group, as reported in the Duchin and Mysak data. For speakers aged 45–54 years, the reference criterion for appropriate rate was 3.39 ($SD = 0.53$) syllables per second or higher; for speakers aged 55–64 years, 3.55 ($SD = 0.53$) syllables per second or higher; for speakers aged 65–74 years, 3.20 ($SD = 0.50$) syllables per second or higher; and, for speakers aged 75–91 years, 3.13 ($SD = 0.51$) syllables per second or higher. Rates higher than 6 syllables per second were coded as inappropriately fast.

Using the above criteria for speakers in each age group, AOS speakers averaged 42% utterances with appropriate rate, whereas AOSci speakers averaged nearly 97% of utterances with appropriate rate (figure 3, panel A). As indicated in figure 3, panel B, the one statistically significant finding among the four rate subcodes was PV9: Slow Rate due to reduced articulation times and increased pause times. All AOS speakers (table 3) produced slow rate (PV9) on more than 15% of their utterances, ranging from 17% (AOS12) to 100% (AOS7) of utterances. Only one

speaker (AOS12) produced slow rate on fewer than 20% of her utterances, which met the PVSP criterion for 'pass' on this variable. This speaker was rated as having mild AOS (table 1) and was among the speakers with the highest speech scores (PCC = 99.4%). As shown in table 3, slow rate was rarely coded for any of the 14 AOSci speakers, ranging from 4% to 13% of utterances coded as PV9.

Stress

By design, the AOSci speakers were selected because fewer than 80% of their utterances met PVSP criteria for appropriate stress. The goal was to emphasize the subtype coded as PV15: Excessive/Equal/Misplaced Stress; for one of the 14 AOSci speakers (AOSci9), the significant stress deficit was limited to PV14: Reduced-Equal Stress. As shown in figure 3, panel A, AOSci speakers averaged significantly fewer utterances with appropriate stress (50.0%) compared to the average for the AOS speakers (82.8%). Among the four subcodes for stress in figure 3, panel B, the only subcode on which groups were significantly different was PV15: Excessive/Equal/Misplaced Stress. As listed in table 4, an average of 41.5% of AOSci speakers' utterances were coded as PV15, whereas the average for AOS speakers was 15.1%. Three adults (AOS2, AOS12, AOS13) had 100% appropriate stress. Additional analysis of the profiles of the three adults with no inappropriate stress codes was unrevealing. Although each of these speakers with AOS had a relatively mild speech impairment and few utterances rated as slow, similar profiles were observed for the AOS speakers with at least one utterance coded as inappropriate stress. Among the AOS speakers with inappropriate stress, the percentage of utterances coded PV15 ranged from 8.3% (AOS3, AOS5, AOS9, AOS11) to 50% (AOS4). In comparison, the percentage of utterances with PV15 in the AOSci speakers ranged from 26.7% to 95.8%. Note that appropriate stress was variable in each AOSci speaker; none of the 14 speakers had inappropriate stress on all utterances.

An account of the subcodes for each occurrence of PV15: Excessive/Equal/Misplaced Stress in AOS and AOSci speakers is central to the goals of this study. Recall that PV15 includes four types of third-level subcodes for inappropriate stress (cf. Shriberg *et al.*, 1990). One subtype is excessive-equal stress, characterized by forceful, robotic-sounding monostress, including stressing of words that are normally unstressed. A second subtype is misplaced stress on a word relative to expected phrasal or emphatic stress assignment. A third subcode is a block on a sound, similar to those occurring in dysfluency. The fourth subcode is a prolongation of a consonant or vowel/diphthong anywhere in the word. The last two categories were included in PV15 because, when present, they were observed to yield the percept of misplaced stress.

Figure 4 provides percentages for each of these subcodes of PV15 for the two speaker groups. Data at this level were available for each of the 14 speakers with AOS transcribed for the present study. Data at this level were not available for all speakers with AOSci. AOSci9 did not have any utterances coded PV15 (each utterance with inappropriate stress was coded PV14), and for three additional AOSci speakers, PV15 codes had not been subcoded into the four subtypes on the original PVSP data sheets completed by transcribers in the prior study. The 14 AOS speakers had 60 utterances subcoded for PV15, and the 10 AOSci speakers eligible for this analysis had a total of 85 utterances in which one of the four PV15 subcodes was assigned. These totals were used as the denominators for the percentage values

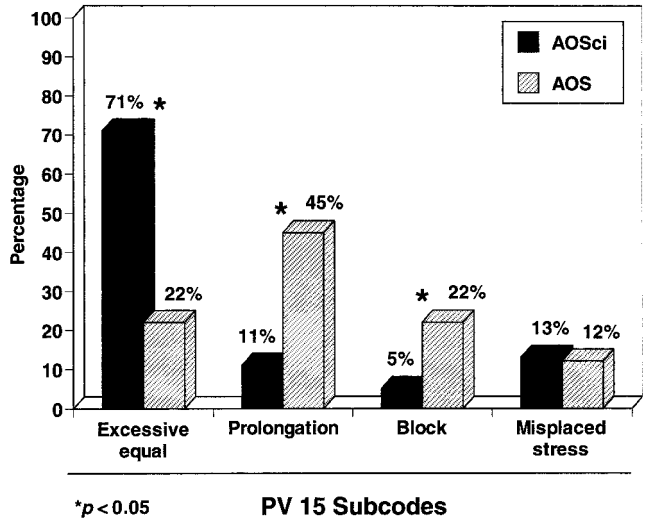


Figure 4. *Percentage of occurrence of each of the four subcodes for PV15: Excessive/Equal/Misplaced Stress for adult speakers with apraxia of speech (AOS) and child speakers with developmental apraxia of speech and inappropriate stress (AOSci).*

shown in figure 4. The overall chi-square analysis was significant (χ^2 [3, 28] = 34.38, $p = 0.000$), as were the between-groups comparisons for excessive-equal (χ^2 [1, 28] = 33.67, $p = 0.000$), prolongations (χ^2 [1, 28] = 22.32, $p = 0.000$), and blocks (χ^2 [1, 28] = 9.78, $p = 0.002$). For the AOSci speakers, 71% of their PV15 codes was due to excessive-equal stress, compared to only 22% for the AOS speakers' PV15 codes. In contrast, a total of 67% of the AOS speakers' PV15 codes was due to sound-level difficulties, coded as blocks or prolongations, whereas 16% of the AOSci speakers' PV15 codes was based on sound-level stress difficulties. Thus, although an average of 15% of AOS speakers' utterances was coded as having inappropriate (PV15) stress (figure 3), 67%, or nearly two-thirds, of these utterances were typologically different from the majority of the utterances coded as inappropriate stress (PV15) for the AOSci speakers.

Subgroup analysis

A supplementary analysis was conducted to compare subsets of speakers in each group with comparable stress deficits and normal cognitive-linguistic status. The AOS subgroup included the five adult speakers who produced fewer than 80% of their utterances with appropriate stress (AOS4, AOS7, AOS8, AOS10, and AOS14). The AOSci subgroup included those ten child speakers with cognitive and receptive language scores in the normal range (AOSci1 - AOSci4; AOSci6 - AOSci9; AOSci13 - AOSci14). Prosody findings were similar to those obtained for the analyses of the total groups reported above. As planned, the average stress scores for the two subgroups were essentially similar: AOS = 64.3% and AOSci = 50%. The distributions of subcodes in the two groups were markedly different, however, and consistent with the proportions reported above for the total group analysis. For the ten AOSci speakers, most (69%) of their PV15 codes were excessive/equal stress, with the remaining stress errors coded as prolongations (17%) or misplaced stress (15%). For

the five AOS speakers, most (61%) of their PV15 codes were prolongations, with the remaining stress errors coded as blocks (24%), excessive/equal stress (6%), or misplaced stress (9%). Findings for the phrasing and rate variables were also consistent with those reported previously for the total group analysis. The subgroup analysis indicated that, in comparison to the speakers with AOSci, speakers with AOS had lower scores on both phrasing (AOS: 47.3%, AOSci: 91.5%) and rate (AOS: 35%, AOSci = 90.5%).

Loudness and resonance

The remaining two statistically significant findings shown in figure 3 (excluding the summative finding for quality; see next paragraph) occurred for the voice variables of loudness and resonance. As indicated by the data in figure 3, panel A, the perceived loudness of speakers in both AOS and AOSci groups was in the normal range, but the average 6% difference between 97% appropriate loudness for the AOS speakers and 91% for the AOSci speakers was statistically significant. The inappropriate loudness code most often used for the AOSci speakers was PV18: Too Loud.

The statistically significant summative finding for quality, as shown in figure 3, panel A, was associated with significant differences in resonance quality. Whereas the averaged utterances for the AOS speakers were within the normal range (i.e., above 80% appropriate), an average of only 73.7% of utterances of AOSci speakers were judged to have appropriate resonance. As discussed in Shriberg *et al.* (1997b; c), most of the utterances judged as inappropriate resonance for the AOSci speakers were coded PV32: Nasopharyngeal Resonance. Additional analysis of this finding in the Shriberg *et al.* study indicated that these group-level findings were associated with only a few male adolescent speakers in the AOSci group, and the trend did not replicate when subgroups were blocked on age and sex.

Summary

Findings from the prosody-voice analyses indicate a triple dissociation between the prosody profiles for the two disorder groups. For the AOSci speakers' utterances, phrasing (88.7%) and rate (96.8%) were within the normal range, whereas approximately half of the utterances of the AOS speakers (phrasing: 53.3%; rate: 42.0%) were judged to be inappropriate on these variables. Conversely, whereas stress for the AOS speakers' utterances (82.8%) averaged in the normal range, half (50.0%) of the AOSci speakers' utterances met PVSP criteria for inappropriate stress. Examination of the subcodes for the stress variable for 10 of the 14 AOSci speakers indicated that 71% of their PV15 codes met criteria for excessive-equal stress, compared to 22% meeting these criteria for the 14 AOS speakers. Thus, whereas the majority of the AOSci speakers' stress errors included syllables, the majority (67%) of the AOS speakers' inappropriate stress was coded as sound-level blocks and prolongations. The per-participant data indicated that all or most of the speakers in each group contributed to those between-group averaged differences for the three prosody variables.

Discussion

Design issues

Two constraints associated with the research design warrant initial consideration. First, the participants in the AOS and AOSci groups do not represent the full range of severity of involvement for these disorders. Because the PVSP procedure required that prosody-voice coding be obtained from conversational speech samples, eligible speakers had to be able to produce intelligible speech in sentence-length utterances. Generalizations from the present data cannot be made to persons with AOS or AOSci who do not meet this inclusionary criterion.

A second limitation concerns the methods used for data reduction, which included behavioural measures using auditory-perceptual criteria to transcribe speech and code inappropriate prosody-voice. Especially for the prosody-voice variables that are the focus of this study, additional and alternative measurement approaches, including instrumental analysis, could have yielded more detailed information on segmental and suprasegmental variables (cf. Kent, 1996). For example, if the speakers engaged in any nonverbal groping or struggle for articulatory positioning during the speech sample, it was lost to coding by the transcribers using auditory-perceptual coding from audiocassette recordings. As noted previously, some reports of both AOS and AOSc propose that such inaudible behaviours contribute significantly to the percept of disrupted fluency. Thus, the overall severity of fluency interruptions, coded as phrasing errors or stress (prolongations, blocks) in the PVSP, may have been underestimated for either or both groups due to sensitivity constraints inherent in the data reduction.

These design limitations should be viewed in relation to several potential design strengths, including:

(a) the number and clinical representativeness of participants recruited for both disorder groups;
(b) use of standardized methods for speech sampling, transcription, and prosody-voice analysis; and
(c) transcription and prosody-voice coding by experienced transcribers who were unaware of each participant's clinical diagnosis.

Design issues are important to weigh in the following discussion, which considers the implications of findings separately for each disorder group, beginning with implications of findings for the loci of deficits in speakers with AOS.

Implications of findings for prosodic processing in AOS

Stress

The AOS speakers produced approximately 83% of their utterances with appropriate stress. Three of the 14 AOS speakers had no utterances coded for inappropriate stress (although two of these three speakers had some utterances for which the coder was unsure whether stress was appropriate). These findings suggest that, for most utterances produced by the AOS speakers, information about sentential stress was accurately coded at the linguistic-representational level, retrieved appropriately at the selection-retrieval level, incorporated without error into a prearticulatory phonetic plan and kinematic program, accessed appropriately for production at the

execution level, and, presumably, appropriately self-monitored before and after manifest speech. Fluctuations in the cognitive resources required to access representations at linguistic and motor speech levels and to self-monitor the products of each stage may constitute a sufficient cause for the relatively few stress errors that did appear (cf. Kent and McNeil, 1987). Other possibilities for the utterances with inappropriate stress include deficits within planning and programming processes themselves, including errors generated when the segmental and suprasegmental phonetic plans are merged or in specifying the parameters of the GMP (cf. Clark and Robin, 1998).

The finding that inappropriate stress in AOS was relatively infrequent differs from the view emphasizing the centrality of abnormal stress as a diagnostic marker for AOS (Darley *et al.*, 1975; Kent and Rosenbek, 1983; Square-Storer *et al.*, 1988; Odell *et al.*, 1991; Wertz *et al.*, 1991; McNeil *et al.*, 1997). However, reconsideration of several prior studies indicates that stress abnormalities are not consistently evident in all utterances or in all speakers described in the archival literature. Only one of the four relatively pure AOS speakers in Square-Storer *et al.* (1988) had perceptually even stress. In their study of seven speakers with AOS, Kent and Rosenbek (1983) noted vowel prolongation and flattening of the relative peak intensity across syllables, both features contributing to the perception of inappropriate stress; however, significant deviations from normal speakers in the intensity envelope across a sequence of syllables occurred on only two of the four stimulus sentences in this study. Odell *et al.* (1991) noted that, although the four apraxic speakers in their study had higher rates of stress errors than speakers in the dysarthric and aphasic groups, the actual AOS stress error rate averaged 44.5%, indicating that over half of the words evidenced appropriate stress.

Rate

The finding that over half of the AOS speakers' averaged utterances were rated as inappropriately slow is consistent with previous reports in the literature (e.g., Kent and Rosenbek, 1983; Square-Storer *et al.*, 1988; Pierce, 1991; McNeil *et al.*, 1997). Slow rate in previous acoustic studies (Kent and Rosenbek, 1983; Kent and McNeil, 1987; Strand and McNeil, 1996) has been characterized by both reduced articulatory rate and increased intraword or interword intervals; it is routinely attributed to disruption at some point in the prearticulatory phases of speech production. For the AOS speakers in the present study, reduced rate cannot be explained either by muscle slowness or weakness, or by cognitive-linguistic deficits affecting previously acquired segmental and suprasegmental representations at the linguistic level. Rather, the candidate explanations for reduced rate are:

(a) difficulties in the development or accessing of a phonetic plan;
(b) difficulties in the development of a kinematic program with an accurate spatial-temporal template and/or timing information, or difficulties accessing a kinematic organizational structure (e.g., GMP); or
(c) difficulties in self-monitoring for the successful realization of the kinematic program prior to or after manifest speech.

Phrasing

The finding that the AOS speakers had considerable difficulties with the behaviours subsumed within the prosodic parameter of phrasing is consistent with reports in

the adult neurogenics literature. As discussed previously, phrasing and stress codes in the PVSP include verbal behaviours associated with articulatory groping or struggle, frequently occurring at the beginning of words or phrases. Although such behaviours are not unique to AOS (Pierce, 1991; McNeil *et al*., 1997), most researchers and clinicians have observed them as routine features of speakers classified as having AOS on the basis of other diagnostic markers (e.g., Johns and LaPointe, 1976; Dabul, 1979; Kent and Rosenbek, 1983; Square-Storer *et al*., 1988; Odell *et al*., 1991; Pierce, 1991; Wertz *et al*., 1991; McNeil *et al*., 1995). AOS speakers seem to know when deviations in their speech or preparation for speech have occurred, and they attempt corrections, though not always successfully (Wertz *et al*., 1991; McNeil *et al*., 1995). The need to repair indicates that, at some point in the output planning, an error or underspecification of the target has occurred. In AOS speakers with good language and without substantial dysarthria, such deficits could be due to processing constraints in either the representational or access phases of planning or programming templates. Access difficulties might include slow or intermittent selection or retrieval of an intact spatial-temporal template (Kent and Rosenbek, 1983) at the planning stage, or slow or intermittent retrieval of a GMP or specification of the parameters of the GMP at the programming stage (Clark and Robin, 1998).

Implications of findings for prosodic processing in AOSci

Stress

As described previously, all of the AOSci speakers met criteria for having inappropriate stress, with 71% of the PV15 codes for 10 of the 14 AOSci speakers subtyped as excessive-equal stress. Comparative analyses of the profiles of the six children whose inappropriate stress rates exceeded 50% (AOSci4, AOSci5, AOSci7, AOSci8, AOSci 9, AOSci10) with the profiles for the remaining eight children did not yield strong associations with other aspects of their communication ability. Cognition was within normal limits for five of the six children and language comprehension was within normal limits for four of the six children. Speech competence was not low in all of these six children (table 3); PCCs ranged from 44.6% to 87.2%, the latter being the second highest speech score in the AOSci group. Thus, it is important to note that the inappropriate stress deficit identified in these children with AOSci was not linked to status in other cognitive-linguistic domains. Unlike what is found in some adult and child speech disorders, all of the speakers with AOS and most of the speakers with AOSc had adequate and comparable cognitive resources, yet only the speakers in the AOSci group had significant stress deficits. Although this dissociation is counterevidence for the perspective that AOS and AOSci have common speech processing deficits, it does not directly address the validity of the representational account of AOSci proposed in Shriberg *et al*. (1997c). In the following two sections, discussions of the dissociations obtained in rate and phrasing directly address the level of support for alternative accounts of the processing deficit underlying the stress deficit in AOSci.

Rate

In the children with AOSci, rate of speech was, without exception, within normal limits (greater than 80% appropriate utterances), with the group-averaged percentage of utterances with appropriate rate reaching nearly 97% (table 4). Such findings

would appear to provide counterevidence for the perspective that the deficit in AOSci is at the level of prearticulatory planning or programming—for the very reasons that the slow speech of adult AOS speakers is routinely viewed as primary evidence supporting the locus of deficits at this level. Appropriate rates of speech coded in conversational contexts suggest that the speakers with AOSci did not have segmental timing errors encoded at the representational or prearticulatory stages, nor were they slow in either preparing the phonetic plan or unpacking it to specify the kinematic program.

Despite AOSci speakers' appropriate speech rates, deficits in motor speech processes could still be implicated in AOSci. As reviewed previously, segmental and suprasegmental forms have been modelled as having independent or modular sources of development, access, and monitoring at each of the levels of speech processing prior to and possibly within prearticulatory stages. From this perspective, evidence of intact speech processing in the temporal domain (i.e., speech rate) is not sufficient to rule out the possibility of deficits elsewhere in spatial specification and other dimensions of segmental and suprasegmental assembly and sequencing. Rather, constraints in the phonetic plan, kinematic program, and/or monitoring processes could presumably account for circumscribed prosodic deficits such as those described in the prior section on sentential stress in speakers with AOSc.

Phrasing

In contrast to the findings for the AOS speakers, the finding of essentially normal phrasing for the AOSci speakers in the present study is not consistent with some reports in the AOSc literature (Rosenbek and Wertz, 1972; Yoss and Darley, 1974; Aram and Glasson, 1979; Hall *et al*., 1993) but is consistent with the findings of Morgan Barry (1995b) and Van der Merwe *et al*. (1996) reviewed previously. These later two reports indicated fewer repetition errors in children with suspected AOSc than observed in adults with AOS. Two possible design explanations for the differences among studies are potential differences in subject selection and/or the assessment methods. Participants for the present study were selected to be maximally representative of children with suspected AOSc who had deficits in the realization of stress (i.e., AOSci). As suggested in Shriberg *et al*. (1997c), such children could comprise a subgroup of children with AOSc, perhaps different from children with suspected AOSc who have the repetitions and revisions associated with inappropriate phrasing. As well, the methods used to identify and quantify phrasing differences in the present study could be less sensitive to the types of fluency problems described in other studies of children with suspected AOSc. This latter possible explanation is made less plausible by the evident sensitivity of the PVSP procedures to the phrasing deficits obtained for the adults with AOS.

Potential methodological constraints notwithstanding, the absence of phrasing errors in the children with suspected AOSci was viewed in the Shriberg *et al*. (1997c) study as one of several sources of support for a linguistic-representational, rather than selection-retrieval or prearticulatory, deficit in these children. It is useful to revisit this conclusion in light of findings from the current study indicating that adults with AOS have significant phrasing deficits (i.e., repetitions and revisions primarily of sounds and syllables), which are assumed to reflect self-monitoring processes. There are two alternative hypotheses to account for the significantly reduced occurrence of self-monitoring in the children with suspected AOSci: (a) they are aware of their errors but elect not to repair, or (b) they are unaware of many

of their errors due to a processing deficit somewhere in the decoding or encoding phases depicted in figure 1.

There is little evidence or rationale to support the first explanatory hypothesis for the phrasing dissociation—that children with AOSci and adults with AOS are both aware of their errors, but children with AOSci elect not to try to repair errors. The issue centres on the salience of the errors for children with AOSci and the learned probability that repairs might be successful. Most of the speech samples used in the present study were obtained by the clinical investigators, who were likely perceived by the children as speech clinicians; indeed, many of the interlocutors had been the clinicians for these children at some point in time. As observed in other children with histories of long-term treatment for speech-sound distortions (Shriberg, 1975; 1980), attempts to demonstrate the ability to correct error targets would be expected, especially when talking with adults who were associated with speech treatment. Unlike the speech characteristics of dysarthria, in which many or most sounds are consistently affected in all verbal exchanges, the variability of AOS and reported variability in AOSci results in some or most sounds and words being produced accurately on at least some occasions. Speakers with AOS, who typically have good cognitive and language skills, appear to be aware of their errors and to believe there is a reasonable probability of improvement with attempted revisions (Darley, 1982; Wertz *et al*., 1991; McNeil *et al*., 1995). This was not observed in the children with AOSci; considering the sampling conditions, it is difficult to envision that they simply elected not to try to self-correct.

The validity of the second hypothesis—that the failure to self-correct in children with AOSci may be due to lack of awareness of at least some of their errors—seems the more plausible explanation. Among the seven candidate explanatory loci for the observed inappropriate sentential stress depicted in figure 1, processing deficits at two loci warrant discussion. Possibly, self-correction (i.e., inappropriate phrasing codes) does not occur because there is no mismatch between the underlying linguistic representation of the target prosody form and the errored output, indicating a deficit at the highest levels of the representational aspects of stress. This was the proposal to account for the stress deficits in the larger sample of children with AOSci reported in Shriberg *et al*. (1997c). Alternatively, lack of self-correction could be due to a deficit at any of the self-monitoring phases of speech production described previously. Such self-monitoring routes purportedly occur throughout speech processing. Thus, deficits in self-monitoring of the products of some processing stage could, itself, be the origin of these AOSci children's lack of repairs, as documented by their high phrasing scores.

Summary and conclusions

For the descriptive-explanatory goals of this paper, we compared the prosody-voice profiles of 14 children with suspected apraxia of speech and inappropriate stress (i.e., AOSci) with profiles for 14 adults with apraxia of speech (AOS) resulting from neurologic lesions. Explanatory perspectives on the findings for the prosodic variables of phrasing, rate, and stress are compatible with hypotheses of processing deficits in AOSci at linguistic, motor speech, and/or self-monitoring phases of speech production.

Linguistic perspectives

The linguistic-explanatory perspective for AOSci is centered on the need to account for the variability of utterance-to-utterance realization of appropriate versus inappropriate stress in children with AOSci. As reported in Velleman and Shriberg (1999), this seeming variability is, in fact, well described by predictions from metrical theory, thus pointing to immature and unstable representational forms as the possible psycholinguistic loci of the stress deficit. The fact that the adults with AOS did not strongly evidence this form of a prosodic deficit weakens support for the view that the two disorders have similar explanatory origins, but it does not invalidate this perspective. Unlike adults, whose apraxia was acquired after the developmental period for speech-language acquisition, the stress deficits in the children with AOSci in this study presumably reflect difficulty or delays in the acquisition of English stress rules. Some investigators have reported impaired auditory processing skills in children with suspected AOSc (e.g., Robin, Hall, and Jordan, 1986; Groenen, Maassen, Crul, and Thoonen, 1996). Deficient auditory or auditory-temporal processing could affect both the initial decoding and eventual representation of metrical forms, as well as the on-line encoding and self-monitoring of appropriate sentential stress. The attractiveness of such hypotheses about input processes is that they address the need for a causal explanation for linguistic claims about entities such as unstable or fuzzy underlying representations. That is, the later explanations are devoid of explanatory force; some causal mechanism must be proposed to account for the failure of children to reliably instantiate stable segmental and suprasegmental representational forms.

Motor speech perspectives

Children with the type of stress deficit observed in this study did not have the slow speech observed in the adults with AOS associated with planning and/or programming impairments. The motor speech explanatory perspective is weakened by this finding, but theoretical views on prearticulatory processing can accommodate normal speech rates in children with AOSci. As described previously, processing tasks at planning and programming phases could be selectively impaired. Thus, the sentential stress deficits in AOSci could reflect motor speech constraints (e.g., within the prosody generator, Levelt, 1989) in the presence of age-appropriate rate. Motor speech explanations also gain indirect support by the data indicating that these AOSci children's stress deficits appear to be independent of their cognitive, language, and speech status.

Especially important from a motor speech perspective is further specification of the articulatory versus metrical elements of the stress deficit that defines children with AOSci. As proposed and illustrated in the PVSP procedural materials (Shriberg *et al.*, 1990; 1992), excessive articulatory force on either consonant or vowel segments meets criteria for PV15: Excessive/Equal/Misplaced Stress. Although stressing of typically unstressed vowels is the primary prosodic (i.e., stress) behaviour that defines AOSci, there is an articulatory-phonetic component of stress that can also be ascribed to speech motor control.

Self-monitoring perspectives

The AOSci children in the present study did not have the deficits in phrasing associated with the AOS speakers' high frequency of attempts at self-repairs. As

suggested previously, this finding implies that children with AOSci may have some form of an internal and/or external self-monitoring deficit that could be an added source of the stress deficit, especially causal to the persistence of the stress deficit over time. Lacking clear guidance from speech processing models to date, failure to attend to mismatches between intention and performance could be posited to occur between any two phases of speech production, including those associated with linguistic forms or prearticulatory motor speech forms. AOSci children's lack of revisions of inappropriately articulated or inappropriately stressed forms is an especially strong finding, suggesting some deficit or delay in typical self-monitoring processes that occurs after manifest speech. Whichever its locus or multiple loci at linguistic or motor speech processing phases, an eventual explanatory model of AOSci needs to account as well for these speakers' lack of revisions of incorrect speech and prosody.

Nosological perspectives

The conclusions above are necessarily preliminary, relative to the central nosological issue: do the present findings support continued use of terms such as AOSc? Certainly there are no data in the present report that can be used to demonstrate that the deficits in children with AOSci are unequivocally entered in linguistic, motor speech, or self-monitoring processes. Contemporary AOSc studies, including the present report, have yet to assemble a large, representative sample of children with the types of speech deficits described over 25 years ago by Yoss and Darley (1974) and Rosenbek and Wertz (1972). Moreover, unaddressed to date is the possibility of ontogenetic differences in the relative contributions of processes in each of these three areas. Because AOSci is presumed to date back to the earliest stages of speech production (i.e., prebabbling), motor speech deficits from the onset of speech could prevent the formulation of well-developed representations or self-monitoring processes; as well, the lack of veridical underlying representations could play a significant role in the development of speech motor control and self-monitoring systems. On nosological issues, therefore, we suggest that, despite the three dissociations between AOSci and AOS found in this study, *suspected* AOSc (Shriberg *et al.* 1997a) remains a useful classificatory term for continuing research and for service delivery needs in child speech disorders.

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Note

1. The terms used in this paper for apraxia of speech in adults and children meet three nosological needs. First, recent discussions of theoretical and service delivery issues suggest that the adjective 'developmental' (i.e., developmental apraxia of speech, developmental verbal dyspraxia) can be interpreted inappropriately to mean that this putative childhood disorder is time-limited and should be treated in an educational context (cf. Apraxia Kids Listserve; Shriberg, Aram, and Kwiatkowski, 1997a; c; Shriberg, 1998). Second, although Apraxia of Speech (AOS) is a well-established term in the context of adult neurogenic disorders, it is ambiguous in studies such as the present that include both adults and children with apraxia of speech. Finally, a term is needed for children with apraxia of speech and inappropriate stress. To meet these three needs, we use the conventional, 'unmarked' term AOS for adults with apraxia of speech, AOSc for children with suspected apraxia of speech, and AOSci for children with suspected apraxia of speech and inappropriate stress.

References

Apraxia Kids Listserve, Apraxia-Kids@listserv.syr.edu

Aram, D. M. and Glasson, C., 1979, *Developmental apraxia of speech*. Paper presented at the Annual Convention of the American Speech-Language-Hearing Association, Los Angeles, CA.

Bayles, K. and Tomoeda, C., 1993, *Arizona Battery for Communication Disorders of Dementia* (Tucson: Canyonlands Publishing, Inc.).

Bennett, S. and Netsell, R. W., 1998, Possible roles of the insula in speech and language processing. *Journal of Medical Speech-Language Pathology*, **7**, 255–272 .

Bishop, D. V. M., 1997, Cognitive neuropsychology and developmental disorders: uncomfortable bedfellows. *The Quarterly Journal of Experimental Psychology: Human Experimental Psychology*, **50**, 899–923.

Bock, J. K., 1982, Toward a cognitive psychology of syntax: Information processing contributions to sentence formulation. *Psychological Review*, **89**, 1–47.

Bybee, J. L., 1994, A view of phonology from a cognitive and functional perspective. *Cognitive Linguistics*, **5**, 285–305.

Caruso, A. J. and Strand, E. A., 1999, Motor speech disorders in children: Definitions, background, and a theoretical framework. In A. J. Caruso and E. A. Strand (Eds) *Clinical management of motor speech disorders in children* (New York: Thieme Medical Publishers, Inc.), pp. 1–27.

Clark, H. and Robin, D., 1998, Generalized motor programme and parameterization accuracy in apraxia of speech and conduction aphasia. *Aphasiology*, **12**, 699–713.

Code, C., 1998, Major review: Models, theories and heuristics in apraxia of speech. *Clinical Linguistics and Phonetics*, **12**, 47–66.

Collins, M. J., Rosenbek, J. C. and Wertz, R. T., 1983, Spectrographic analysis of vowel and word durations in apraxia of speech. *Journal of Speech and Hearing Research*, **26**, 224–230.

Crary, M. A., 1993, *Developmental motor speech disorders* (San Diego, CA: Singular Publishing Group).

Dabul, B., 1979, *Apraxia Battery for Adults* (Tigard, OR: C. C. Publications, Inc.).

Darley, F. L., 1982, *Aphasia* (Philadelphia: W. B. Saunders).

Darley, F. L., Aronson, A. E. and Brown, J. R., 1975, *Motor speech disorders* (Philadelphia: W. B. Saunders).

Davis, B. L., Jakielski, K. J. and Marquardt, T. P., 1998, Developmental apraxia of speech: Determiners of differential diagnosis. *Clinical Linguistics and Phonetics*, **12**, 25–46.

Dell, G., 1986, A spreading-activation theory of retrieval in sentence production. *Psychological Review*, **93**, 283–321.

Dewey, D., 1995, What is developmental dyspraxia? *Brain and Cognition*, **29**, 254–274.

Duchin, S. W. and Mysak, E. D., 1987, Dysfluency and rate characteristics of young adult, middle-aged, and older males. *Journal of Communication Disorders*, **20**, 245–257.

Fisher, S. E., Vargha-Khadem, F., Watkins, K. E., Monaco, A. P. and Pembrey, M. E.,

1998, Localisation of a gene implicated in a severe speech and language disorder. *Nature*, **18**, 168–170.

Forest, K., Adams, S., McNeil, M. R. and 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 and D. R. Beukelman (Eds) *Dysarthria and apraxia of speech: Perspectives on management* (Baltimore: Paul H. Brookes), pp. 147–171.

Gandour, J., Petty, S. H. and Dardarananda, R., 1989, Dysprosody in Broca's aphasia: A case study. *Brain and Language*, **37**, 232–257.

Garnsey, S. M. and Dell, G. S., 1984, Some neurolinguistic implications of pre-articulatory editing in production. *Brain and Language*, **23**, 64–73.

Garrett, M., 1980, Levels of processing in sentence production. In B. Butterworth (Ed.) *Language production*, Volume 1(London: Academic), pp. 177–220.

Goodglass, H. and Kaplan, E., 1983, *The assessment of aphasia and related disorders, second edition* (Philadelphia: Lea and Febiger).

Grela, B. and Gandour, J., 1998, Locus of functional impairment in the production of speech rhythm after brain damage: A preliminary study. *Brain and Language*, **64**, 361–376.

Groenen, P., Maassen, B., Crul, T. and Thoonen, G., 1996, The specific relation between perception and production errors for place of articulation in developmental apraxia of speech. *Journal of Speech and Hearing Research*, **39**, 468–482.

Hageman, C. F., Robin, D. A., Moon, J. B. and Folkins, J. W., 1994, Oral motor tracking in normal and apraxic speakers. *Clinical Aphasiology*, **22**, 219–229.

Hall, P. K., Jordan, L. S. and Robin, D. A., 1993, *Developmental apraxia of speech: Theory and clinical practice* (Austin, TX: Pro-Ed).

Hodge, M. M., 1994, Assessment of children with developmental apraxia of speech: A rationale. *Clinics in Communication Disorders*, **4**, 91–101.

Hodge, M. M., 1998, Developmental coordination disorder: A diagnosis with theoretical and clinical implications for developmental apraxia of speech. *Language Learning and Education*, 8–12.

Hough, M. S. and Klich, R. J., 1987, Effects of word length on lip EMG activity in apraxia of speech. *Clinical Aphasiology*, **15**, 271–276.

Johns, D. F. and LaPointe, L. L., 1976, Neurogenic disorders of output processing: Apraxia of speech. In W. Whitaker and H. A. Whitaker (Eds) *Studies in neurolinguistics 1* (New York, NY: Academic Press), pp. 161–200.

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, R. D., Adams, S. G. and Turner, G. S., 1996, Models of speech production. In N. J. Lass (Ed.) *Principles of experimental phonetics* (St. Louis, MO: Mosby-Year Book), pp. 3–45.

Kent, R. D. and McNeil, M. R., 1987, Relative timing of sentence repetition in apraxia of speech and conduction aphasia. In J. H. Ryall (Ed.) *Phonetic approaches to speech production in aphasia and related disorders* (Boston: College-Hill Publishing), pp. 181–220.

Kent, R. D. and Rosenbek, J. C., 1982, Prosodic disturbance and neurologic lesion. *Brain and Language*, **15**, 259–291.

Kent, R. D. and Rosenbek, J. C., 1983, Acoustic patterns of apraxia of speech. *Journal of Speech and Hearing Research*, **26**, 231–249.

Kertesz, A., 1982, *Western Aphasia Battery* (New York, NY: Grune and Stratton).

Lai, C. S. L., Fisher, S. E., Hurst, J. A., Levy, E. R., Hodgson, S., Fox, M., Jeremiah, S., Povey, S., Curtis Jamison, D., Green, E. D., Vargha-Khadem, F. and Monaco, A. P., 2000, The SPCH1 region on human 7q31: Genomic characterization of the critical interval and localization of translocations associated with speech and language disorder. *American Journal of Human Genetics*, **67**, 357–368.

Levelt, W. J. M., 1989, *Speaking* (Cambridge, MA: MIT Press).

Levelt, W. J. M. and Wheeldon, L., 1994, Do speakers have access to a mental syllabary? *Cognition*, **50**, 239–269.

Liepman, H., 1913, Motor aphasia, anarthria, and apraxia. *Transactions of the 17th International Congress of Medicine, Section XI, Part II*, 97–106.

Liss, J. M., 1998, Error-revision in the spontaneous speech of apraxic speakers. *Brain and Language*, **62**, 342–360.

Locke, J. L., 1994, Gradual emergence of developmental language disorders. *Journal of Speech and Hearing Research*, **37**, 608–616.

Love, R. J., 1992, Developmental verbal dyspraxia. In R. J. Love (Ed.) *Childhood motor speech disability* (New York, NY: Macmillan), pp. 94–111.

Marquardt, T. P. and Sussman, H. M., 1991, Developmental apraxia of speech: Theory and Practice. In D. Vogel and M. Cannito (Eds) *Treating disordered speech motor control* (Austin, TX: Pro-Ed), pp. 341–390.

McCabe, P., Rosenthal, J. B. and McLeod, S., 1998, Features of developmental dyspraxia in the general speech-impaired population? *Clinical Linguistics and Phonetics*, **12**, 105–126.

McNeil, M. R. and Adams, S., 1991, A comparison of speech kinematics among apraxic, conduction aphasic, ataxic dysarthric, and normal geriatric speakers. *Clinical Aphasiology*, **19**, 279–294.

McNeil, M. R., Caligiuri, M. and Rosenbek, J. C., 1989, A comparison of labio-mandibular speech kinematic durations, displacement, velocities and dysmetrias in ataxic and normal adults. *Clinical Aphasiology*, **17**, 173–193.

McNeil, M. R., Liss, J., Tseng, C.-H. and Kent, R. D., 1990, Effects of speech rate on the absolute and relative timing of apraxic and conduction aphasic sentence production. *Brain and Language*, **38**, 135–158.

McNeil, M. R., Odell, K. H., Miller, S. B. and Hunter, L., 1995, Consistency, variability, and target approximation for successive speech repetitions among apraxic, conduction aphasic, and ataxic speakers. *Clinical Aphasiology*, **23**, 39–55.

McNeil, M. R. and Prescott, T., 1978, *Revised Token Test* (Austin, TX: College Hill Press).

McNeil, M. R., Robin, D. A. and Schmidt, R. A., 1997, Apraxia of speech: Definition, differentiation, and treatment. In M. R. McNeil (Ed.) *Clinical management of motor speech disorders* (New York, NY: Thieme Medical Publishers, Inc), pp. 331–344.

McNeil, M. R., Weismer, G., Adams, S. and 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.

McSweeny, J. L. and Shriberg, L. D., 1995, *Segmental and suprasegmental transcription reliability* (Tech. Rep. No. 2). Phonology Project, Waisman Center on Mental Retardation and Human Development, University of Wisconsin-Madison.

Morgan Barry, R., 1995a, The relationship between dysarthria and verbal dyspraxia in children: A comparative study using profiling and instrumental analyses. *Clinical Linguistics and Phonetics*, **9**, 277–309.

Morgan Barry, R., 1995b, A comparative study of the relationship between dysarthria and verbal dyspraxia in adults and children. *Clinical Linguistics and Phonetics*, **9**, 311–332.

Motley, M. T., Baars, B. J. and Camden, C. T., 1983, Experimental verbal slip studies: A review and an editing model of language encoding. *Communication Monographs*, **50**, 79–101.

Odell, K., McNeil, M. R., Rosenbek, J. C. and Hunter, L., 1991, Perceptual characteristics of vowel and prosody production in apraxic, aphasic, and dysarthric speakers. *Journal of Speech and Hearing Research*, **34**, 67–80.

Ozanne, A., 1995, The search for developmental verbal dyspraxia. In B. Dodd (Ed.) *Differential diagnosis and treatment of children with speech disorder* (San Diego, CA: Singular Publishing Group, Inc.), pp. 91–109.

Pierce, R. S., 1991, Apraxia of speech versus phonemic paraphasia: Theoretical, diagnostic, and treatment considerations. In D. Vogel and M. P. Cannito (Eds) *Treating disorders of speech motor control: For clinicians by clinicians* (Austin, TX: Pro-Ed.), pp. 185–216.

Porch, B., 1967, *Porch Index of Communicative Ability* (Palo Alto, CA: Consulting Psychologists Press).

Raven, J. C., Court, J. H. and Raven, J., 1990, *Coloured Progressive Matrices* (Oxford: Oxford Psychologists Press).

Robin, D. A., Hall, P. K. and Jordan, L. S., 1986, Auditory processing deficits in developmental verbal apraxia. Paper presented at the Annual Convention of the American Speech-Language-Hearing Association, November, Detroit, MI.

Rosenbek, J. C., Kent, R. D. and LaPointe, L. L., 1984, Apraxia of speech: An overview and

some perspectives. In J. C. Rosenbek, M. R. McNeil and A. E. Aronson (Eds) *Apraxia of speech: Physiology, acoustics, linguistics, management* (San Diego, CA: College-Hill Press), pp. 1–72.

Rosenbek, J. C. and Wertz, R.T., 1972, A review of fifty cases of developmental apraxia of speech. *Language, Speech and Hearing in the Schools*, **3**, 23–33.

Schmidt, R. A., 1988, *Motor control and learning: A behavioral emphasis*, second edition (Champaign, IL: Human Kinetics).

Seddoh, S. A. K., Robin, D. A., Sim, H.-S., Hageman, C., Moon, J. B. and Folkins, J. W., 1996, Speech timing in apraxia of speech versus conduction aphasia. *Journal of Speech and Hearing Research*, **39**, 590–603.

Shattuck-Huffnagle, S., 1983, Sublexical units and suprasegmental structure in speech production planning. In P. F. MacNeilage (Ed.) *The production of speech* (New York, NY: Springer), pp. 109–136.

Shriberg, L. D., 1975, A response evocation program for /ɜ˞/. *Journal of Speech and Hearing Disorders*, **40**, 92–105.

Shriberg, L. D., 1980, An intervention procedure for children with persistent /r/ errors. *Language, Speech, and Hearing Services in Schools*, **11**, 102–110.

Shriberg, L. D., 1986, *PEPPER: Programs to examine phonetic and phonologic evaluation records* (Hillsdale, NJ: Lawrence Erlbaum).

Shriberg, L. D., 1993, Four new speech and prosody-voice measures for genetics research and other studies in developmental phonological disorders. *Journal of Speech and Hearing Research*, **36**, 105–140.

Shriberg, L. D., 1998, Management of Developmental Apraxia of Speech: Linguistic and motor perspectives: Convergence on nosology, etiology, and treatment. Discussion of papers presented at the Annual Convention of the American Speech-Language-Hearing Association, November, San Antonio, TX.

Shriberg, L. D., Aram, D. M. and Kwiatkowski, J., 1997a, Developmental apraxia of speech: I. Descriptive perspectives. *Journal of Speech, Language, and Hearing Research*, **40**, 273–285.

Shriberg, L. D., Aram, D. M. and Kwiatkowski, J., 1997b, Developmental apraxia of speech: II. Toward a diagnostic marker. *Journal of Speech, Language, and Hearing Research*, **40**, 286–312.

Shriberg, L. D., Aram, D. M. and Kwiatkowski, J., 1997c, Developmental apraxia of speech: III. A subtype marked by inappropriate stress. *Journal of Speech, Language, and Hearing Research*, **40**, 313–337.

Shriberg, L. D., Austin, D., Lewis, B. A., McSweeny, J. L. and Wilson, D. L., 1997a, The Percentage of Consonants Correct (PCC) metric: Extensions and reliability data. *Journal of Speech, Language, and Hearing Research*, **40**, 708–722.

Shriberg, L. D., Austin, D., Lewis, B. A., McSweeny, J. L., and Wilson, D. L., 1997b, The Speech Disorders Classification System (SDCS): Extensions and lifespan reference data. *Journal of Speech, Language, and Hearing Research*, **40**, 723–740.

Shriberg, L. D. and Kent, R. D., 1995, *Clinical phonetics*, second edition (Boston, MA: Allyn and Bacon).

Shriberg, L. D. and Kwiatkowski, J., 1980, *Natural Process Analysis: A procedure for phonological analysis of continuous speech samples* (New York, NY: Macmillan).

Shriberg, L. D. and Kwiatkowski, J., 1982, Phonological disorders III: A procedure for assessing severity of involvement. *Journal of Speech and Hearing Disorders*, **47**, 256–270.

Shriberg, L. D. and Kwiatkowski, J., 1994, Developmental phonological disorders I: A clinical profile. *Journal of Speech and Hearing Research*, **37**, 1100–1126.

Shriberg, L. D., Kwiatkowski, J. and Hoffmann, K. A., 1984, A procedure for phonetic transcription by consensus. *Journal of Speech and Hearing Research*, **27**, 456–465.

Shriberg, L. D., Kwiatkowski, J. and Rasmussen, C., 1990, *The Prosody-Voice Screening Profile* (Tucson, AZ: Communication Skill Builders).

Shriberg, L. D., Kwiatkowski, J., Rasmussen, C., Lof, G. L. and Miller, J. F., 1992, The Prosody-Voice Screening Profile (PVSP): Psychometric data and reference information for children (Tech. Rep. No. 1). Phonology Project, Waisman Center on Mental Retardation and Human Development, University of Wisconsin-Madison.

Shriberg, L. D. and Lof, G. L., 1991, Reliability studies in broad and narrow phonetic transcription. *Clinical Linguistics and Phonetics*, **5**, 225–279.

SHRIBERG, L. D. and OLSON, D., 1988, *PEPAGREE: A Program to Compute Transcription Reliability* (Waisman Center Research Computing Facility, University of Wisconsin-Madison).

SIEGEL, S. and CASTELLAN, N. J., 1988, *Nonparametric statistics for the behavioral sciences*, second edition (New York, NY: McGraw-Hill).

SKINDER, A., STRAND, E. A. and MIGNEREY, M., 1999, Perceptual and acoustic analysis of lexical and sentential stress in children with developmental apraxia of speech. *Journal of Medical Speech-Language Pathology*, **7**, 133–144.

SQUARE-STORER, P., DARLEY, F. and SOMMERS, R. T., 1988, Nonspeech and speech processing skills in patients with aphasia and apraxia of speech. *Brain and Language*, **33**, 65–85.

STACKHOUSE, J., 1992, Developmental verbal dyspraxia I: A review and critique. *European Journal of Disorders of Communication*, **27**, 19–34.

STRAND, E. A., 1998, Treatment of developmental apraxia of speech: Application of motor learning principles. *Language Learning and Education*, 16–21.

STRAND, E.A. and 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.

STRAND, E. A. and SKINDER, A., 1999, Treatment of developmental apraxia of speech: Integral stimulation methods. In A. Caruso and E. Strand (Eds) *Clinical management of motor speech disorders in children* (New York, NY: Thieme Medical Publishers, Inc.), pp. 109–148.

THOONEN, G., 1998, *Developmental apraxia of speech in children: Quantitative assessment of speech characteristics* (Enschede, the Netherlands: Print Partners Ipskamp).

THOONEN, G., MAASSEN, B., GABREELS, F., SCHREUDER, R. and DE SWART, B., 1997, Towards a standardised assessment procedure for developmental apraxia of speech. *European Journal of Disorders of Communication*, **32**, 37–60.

VAN DER MERWE, A., 1997, A theoretical framework for the characterization of pathological sensorimotor control. In M. R. McNeil (Ed.) *Clinical management of motor speech disorders* (New York, NY: Thieme Medical Publishers, Inc.), pp. 1–25.

VAN DER MERWE, A. and GRIMBEEK, R. J., 1990, A comparison of the influence of certain contextual factors on the symptoms of subjects with acquired apraxia of speech and developmental apraxia of speech. *The South African Journal of Communication Disorders*, **37**, 27–34.

VAN DER MERWE, A., MLOG, E. G. and GRIMBEEK, R. J., 1996, Acquired and developmental apraxia of speech: Perceptual and acoustic analysis of symptoms with reference to context sensitivity and variability. Paper presented at the Eighth Biennial Conference on Motor Speech, February.

VARGHA-KHADEM, F., WATKINS, K. E., ALCOCK, K. J., FLETCHER, P. and PASSINGHAM, R., 1995, Praxic and nonverbal cognitive deficits in a large family with a genetically transmitted speech and language disorder. *Proceedings of the National Academy of Sciences of the United States of America*, **92**, 930–933.

VARGHA-KHADEM, F., WATKINS, K. E., PRICE, C. J., ASHBURNER, J., ALCOCK, K. J., CONNELLY, A., FRACKOWIAK, R. S., FRISTON, K. J., PEMBREY, M. E., MISHKIN, M., GADIAN, D. G. and PASSINGHAM, R. E., 1998, Neural basis of an inherited speech and language disorder. *Proceedings of the National Academy of Sciences of the United States of America*, **95**, 12695–12700.

VELLEMAN, S. L., 1998, Lexical stress errors in developmental verbal dyspraxia: The problem and some possible solutions. *Language Learning and Education*, 12–16.

VELLEMAN, S. L. and SHRIBERG, L. D., 1999, Metrical analysis of children with suspected developmental apraxia of speech and inappropriate stress. *Journal of Speech, Language, and Hearing Research*, **42**, 1444–1460.

VELLEMAN, S. L. and STRAND, K., 1994, Developmental verbal dyspraxia. In J. E. Bernthal and N. W. Bankson (Eds) *Child phonology: Characteristics, assessment, and intervention with special populations* (New York, NY: Thieme Medical Publishers, Inc.), pp. 110–139.

WERTZ, R. T. and ROSENBEK, J. C., 1976, Structural-functional exam. Unpublished manuscript, Veteran's Administration Medical Center, Madison, WI.

WERTZ, R .T., LAPOINTE, L. L. and ROSENBEK, J. C., 1991, *Apraxia of speech in adults: The disorder and its management* (San Diego, CA: Singular Publishing Group, Inc.).

YOSS, K. A. and DARLEY, F. L., 1974, Developmental apraxia of speech in children with defective articulation. *Journal of Speech and Hearing Research*, **17**, 399–416.