

## Review

# Understanding the nature of apraxia of speech: Theory, analysis, and treatment

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### Abstract

Researchers have interpreted the behaviours of individuals with acquired apraxia of speech (AOS) as impairment of linguistic phonological processing, motor control, or both. Acoustic, kinematic, and perceptual studies of speech in more recent years have led to significant advances in our understanding of the disorder and wide acceptance that it affects phonetic-motoric planning of speech. However, newly developed methods for studying nonspeech motor control are providing new insights, indicating that the motor control impairment of AOS extends beyond speech and is manifest in nonspeech movements of the oral structures. We present the most recent developments in theory and methods to examine and define the nature of AOS. Theories of the disorder are then related to existing treatment approaches and the efficacy of these approaches is examined. Directions for development of new treatments are posited. It is proposed that treatment programmes driven by a principled account of how the motor system learns to produce skilled actions will provide the most efficient and effective framework for treating motor-based speech disorders. In turn, well controlled and theoretically motivated studies of treatment efficacy promise to stimulate further development of theoretical accounts and contribute to our understanding of AOS.

The debate over the precise characterization of acquired apraxia of speech (AOS) has been active for many years. Researchers have interpreted the behaviours of individuals with AOS as an impairment of linguistic phonological processing, motor control, or both. While the notion of AOS as a phonetic-motoric disorder is now generally accepted, it frequently co-occurs with aphasia and differentiating between the respective phonetic-motoric and linguistic impairments has proven difficult. This paper presents the most recent developments in theory, methods, and treatment to examine and define the nature of AOS.

The term apraxia was coined by Steinthal (1871, cited by Roy 1978) and elaborated upon by Liepmann (1900, 1905, 1913). These early researchers regarded apraxia as a

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disorder of purposeful (voluntary) movement not attributable to the loss of strength, co-ordination, or mental faculty and restricted to certain body parts and functional activities. Darley et al. (1975) explored the syndrome of AOS, describing it as an impairment of volitional speech production in the face of preserved linguistic and motor execution abilities. Specifically, they identified the clinical features of (a) effortful groping for articulatory postures, (b) consonant phonemes more affected than vowels, (c) inconsistent, or variable, errors across productions, (d) errors that increase complexity of articulation across a word rather than simplify, (e) errors that approximate the target within one to two features, (f) errors that represent perseveration, anticipation, and transposition of phonemes, (g) schwa insertion in consonant clusters, and (h) awareness of errors. Kent and Rosenbek (1983) also included such features as (a) slowed speech rate with prolongation of segments and transitions between segments, (b) impaired co-ordination of voicing with movement of other articulators, and (c) difficulty with initiation of utterances. Although many of these characteristics have not been rigorously tested, they have survived as diagnostic indicators of AOS and are widely utilized in clinical settings. Furthermore, there has been a focus on the clinical characteristics with less attention given to theorizing about the underlying nature of the disorder.

#### Theoretical accounts of apraxia of speech

A handful of theories have been offered to explain the impairment in AOS (e.g. Mlcoch and Noll 1980, Kelso and Tuller 1981, Kent and McNeil 1987, Kent and Adams 1989, Dogil et al. 1994, Van der Merwe 1997, Clark and Robin 1998, Whiteside and Varley 1998, Rogers and Storkel 1999). To place these theories in a broader perspective, it is beneficial to have an understanding of models of normal language production. Most language production models that have been proposed (e.g. Garrett 1975, Bock 1982, Levelt 1989) posit several stages from formulation to articulation of a message. Although aspects of these models are strictly serial in nature, subunits of a message are processed in parallel (e.g. Bock and Irwin 1980, Bock 1982). Initially, a thought is formulated and then this thought is converted into abstract semantic units. From semantic information, a syntactic frame can be generated that represents the syntactic structure to be used in expressing the thought. Following these semantic and syntactic stages, a phonological representation of the message must be developed followed by a phonetic representation. According to Levelt (1989), at the phonological level the morphological and metrical composition of words is spelled out, followed by the segmental composition (i.e. consonants and vowels). Finally, phonetic syllable programmes are derived from the string of segments and these programmes specify articulatory gestures and vocal tract configuration.

The prevailing theoretical approach to AOS claims that the processes that build the phonological representation of a message are intact but the phonetic-motoric level of production is disrupted (e.g. McNeil et al. 1997, Shriberg et al. 1997a, Van der Merwe 1997). Shriberg et al. (1997a) summarized these approaches by referring to AOS as a deficit in sequencing the spatiotemporal aspects of movement at a prearticulatory level. McNeil et al. (1997) provided a more refined definition of AOS based on a growing body of experimental data. They stated that it is a phonetic-motoric disorder that affects the translation of an intact phonological representation of a message into the learned kinematic parameters for an intended movement.

Van der Merwe (1997) developed a model for considering diagnosis and management of motor speech disorders, including AOS and dysarthrias. This model includes linguistic-symbolic, motor planning, motor programming, and execution levels and relates these to neural substrates. It has numerous components in common with previous models of production (e.g. Levelt 1989) and theories dealing specifically with motor programming (e.g. Schmidt, 1975), but is unique in being specifically applied to accounting for speech and language pathologies. Initially, there is an intention to communicate, driven by the fronto-limbic system. Following this, semantic, syntactic, and morphological planning occurs and finally selection and combination of phonemes to form a phonological representation. These processes are driven by the traditional language areas—temporo-parietal cortex and Broca's area. It is from this point on that the model expands into more detail. Following generation of a phonological representation, the motor planning phase is implemented. Extensive connections between Broca's area, Wernicke's area, prefrontal cortex, premotor cortex (lateral area 6), the supplementary motor area (medial area 6), and somatosensory cortex (areas 5 and 7) underlie a complex association between stored representations and motor and sensory information for planning the speech string. Core motor plans, or the invariant spatial and temporal specifications of phones, are retrieved from a sensorimotor memory store and the consecutive movements involved in fulfilling these specifications are planned at the level of the articulator and interarticulator synchronization. At this point, variance in the realization of temporal and spatial aspects is directly related to factors such as phonetic context and potential for coarticulation, linguistic influences on segmental duration, frequency and familiarity of the motor goal, interarticulatory synchronization, overall speech rate, and awareness of initial conditions of the articulators such as physical perturbations. Although response feedback can have no role in motor planning, it is possible that the process can be monitored centrally. That is, the adapted plan is compared to an internal stored representation that specifies permitted variance. When the limits to adaptation of spatial and temporal features of the core motor plan to the aforementioned variables are overstepped, the listener will perceive distortions and substitutions of sounds. The cerebellum and/or cortical sensorimotor areas may subserve this capacity. Finally, the articulator-specific subroutines that comprise a motor plan are temporally organized and fed forward to the motor programming level.

In Van der Merwe's model (1997), the motor programming phase involves specification of muscle-specific spatiotemporal and force parameters such as muscle tone, resistance, and absolute force, direction, range, and rate of movement. These programmes can be modified in response to sensory feedback as the movement unfolds. Neural substrates for programming include the supplementary motor area, basal ganglia, lateral cerebellum, frontolimbic system, and the primary motor cortex. The final phase in the model is execution, where the actual articulation is controlled and performed via the final common pathway. The primary motor cortex, lower motor neurones, peripheral nerves and motor units are the neural structures directly involved in executing movement. However, other structures such as the supplementary motor area, cerebellum, basal ganglia, and thalamus may also be involved in processing ongoing feedback during motor execution.

Van der Merwe proposed that the acquired motor speech disorders of AOS and the dysarthrias can be related directly to impairments in motor planning, programming, or execution. The behavioural profile of AOS may be explained by disturbances at the motor planning level—retrieval of core motor plans for phones, sequential organization of movements for a single phone or a series of phones, adaptation to phonetic context,

interarticulatory co-ordination, central monitoring, and relaying the motor plans to the motor programming level. It seems that motor speech disorders such as spastic, hypokinetic, hyperkinetic, and ataxic dysarthria may arise from damage affecting programming alone or both programming and execution. Flaccid dysarthria is thought to arise from damage affecting the motor execution level only. This model is possibly the most detailed and comprehensive attempt to explain impairments in the speech production process, relating subcomponents to underlying neural structures, diagnosis of motor speech disorders, and principled development of treatment strategies for such disorders. It provides a series of testable hypotheses for examining the nature of AOS and, thus, for developing treatment goals.

Kelso and Tuller (1981) proposed the coalitional theory of AOS which is consistent with, though less extensive than, the model of Van der Merwe (1997). They viewed AOS as a breakdown in the interaction between an individual and the environment that results in failure to meet behavioural goals. Kelso and Tuller argue that, for skilled actions to be co-ordinated, the neuromuscular system must be organized into functional units also known as coordinative structures (e.g. Easton 1972, Fowler 1977, Turvey 1977, Kelso et al. 1979). In the case of speech, these functional units govern the spatiotemporal relations between articulators during speech production. When a group of muscles is recruited as a functional unit, the relative timing of neuromuscular events between muscles within the group remains constant with changes in the absolute timing and magnitude of the activity (Turvey et al. 1978). Numerous studies have now demonstrated that this finely tuned spatiotemporal co-ordination between articulators is disrupted in AOS (e.g. Freeman et al. 1978, Itoh et al. 1979a,b, Kent and Rosenbek 1983, Ziegler and von Cramon 1986, Kent and McNeil 1987, but see Seddoh et al. 1996). Collectively, these studies have supported the interpretation that AOS is a disorder affecting the phonetic-motoric level of speech production.

Kent and Adams (1989) also refer to a breakdown in co-ordination of articulator movements in AOS. They argue that, when the integrity of the motor system is compromised by AOS, the correlation between articulator movements diminishes and variability in production of target movement patterns increases. They comment that a neurologically based temporal co-ordination disorder can account for many of the behavioural characteristics of AOS, although compensatory techniques or processes that code the contextual articulatory requirements for speech segments may play a role.

Two alternative theoretical approaches to AOS include theories of attentional resource allocation or resource capacity limitations (Kent and McNeil 1987, Clark and Robin 1998, Whiteside and Varley 1998, Rogers and Storkel 1999) and linguistic-based accounts (Dogil et al. 1994, Dogil and Mayer 1998). To explain the frequently noted dysprosody of AOS, Kent and McNeil (1987) invoked the notion of a resource allocation problem within a motor control-based approach to speech production. They claimed that the phonetic representation codes information on syllable and segment structure separately so that these two classes of information may be differentially affected. This vulnerability to error at the phonetic-motor programming level forces the speaker to allot more resources to the task of ordering segmental and syllabic information. Kent and McNeil deduce that this increased resource demand results in lengthening of syllables and intersyllabic pauses and so gives rise to the secondary characteristics of dysprosody. Levelt (1989) argued that it is at this level of developing a phonetic plan that output of processes becomes available for prearticulatory editing, which is a more controlled process requiring allocation of resources.

Whiteside and Varley (1998) proposed a 'cognitive-based' account of AOS that posits two routes for phonetic encoding. The direct route accesses stored 'verbo-motor patterns' which specify the relative timing and force of the components of coordinative structures. It appears that a verbo-motor pattern is similar to the concept of a motor programme (e.g. Schmidt 1975) or motor plan (Van der Merwe 1997). The direct route is used for retrieving frequently used phone sequences or syllables from a store and utilizes minimal computational resources. The indirect route is used for encoding very low frequency or novel syllables and words and involves computing the phonetic representation of a word anew on a phone by phone basis each time it is used. This route is demanding of computational resources. Whiteside and Varley claimed that individuals with AOS have lost access to verbo-motor patterns, or motor programmes, via the direct route and must compute phonetic representations phone by phone. This process predicts the reduced coarticulation seen in AOS (Zeigler and von Cramon 1985, McNeil et al. 1994, Mayer 1995). Furthermore, the authors proposed that the indirect route is not used efficiently in compensating for the loss of the direct route. This poor compensation is thought to result in the reported characteristics of articulatory groping (e.g. Darley et al. 1975), increased segmental and intersegmental durations (Bauman 1978, Freeman et al. 1978, Ryalls 1981, 1987, Collins et al. 1983, Kent and Rosenbek 1983, Mercaitis 1983, Strand 1987, Strand and McNeil 1996), and interarticulatory discoordination (Freeman et al. 1978, Itoh et al. 1979a,b, Kent and Rosenbek 1983, Zeigler and von Cramon 1986, Kent and McNeil 1987). This inefficiency in coping apparently may be due to at least two factors. The indirect route may not be effective in isolation, or individuals with AOS may have a coincident deficit in allocating processing resources that has a detrimental effect on their ability to utilize the more resource demanding indirect encoding route.

Rogers and Storkel (1999) proposed that AOS represents a strict resource capacity limitation. They claimed that the phonological output buffer, holding the output of speech programming processes, is limited to one syllable. In a parameter re-mapping task, normal controls and subjects with either aphasia or AOS plus aphasia were presented with a pair of printed words that differed in the feature-based similarity of the initial phoneme—shared voicing and manner, shared place and manner, or no shared features. Subjects were required to repeat the word pair as quickly and as accurately as possible. Only correct productions were analysed for the latency between the offset of the initial consonant of the first word and the onset of the initial consonant in the second word (CC interval) and the latency from offset of the final consonant of the first word and onset of the initial consonant in the second word (IP interval). For both dependent variables, there was no difference between conditions or groups for normal controls and subjects with aphasia alone. In the AOS plus aphasia group, the CC interval was longer across all conditions compared to the other groups. This slowed production of word pairs was interpreted as a phonological similarity effect. That is, when two consecutive words are phonologically similar, production of the second word is slowed (see Rogers and Storkel 1998). Rogers and Storkel (1998) proposed that this effect arises during programming of the second word. That study involved programming one word at a time, whereas the task used by Rogers and Storkel (1999) encouraged subjects to programme two words at a time. Thus, in the latter study, slowed production was taken as evidence that individuals with AOS cannot programme two words (or syllables) at a time for holding in the phonological buffer. Some support for this explanation is provided in a study by Rochon et al. (1990). These authors note that maintaining verbal

material in working memory requires a phonological store, or buffer, and sub-vocal articulatory rehearsal (e.g. Baddeley and Hitch 1974). They present evidence supporting a short-term memory impairment in AOS that results from reduced ability to perform articulatory rehearsal rather than impaired access to phonological representations. However, using a different task to that of Rogers and Storkel (1999), Rochon et al. based their conclusions on the finding that the phonological similarity effect was absent in their subjects with AOS.

The representational account of Dogil et al. (1994) and Dogil and Mayor (1998) takes the opposing stance to the theories just described, positing that the disorder of AOS can be explained as a purely linguistic, or phonological, impairment. These authors examined AOS cross-linguistically and argued that it reflects impaired implementation of phonological representations at the phonology–phonetics interface. The deficit manifests as phonological (e.g. substitution) and phonetic (e.g. distortion) errors. Normal phonological representations are thought to differ in degree of ‘specification’, depending on their role in the phonological structure of the language. Permanent under-specification maintains that phoneme features that do not appear at any stage of derivation or representation are not specified for segments. These features represent sound properties that are phonologically irrelevant in a system (Trubetzkoy 1939) and are associated with laryngeal consonants (e.g. /h/, which are placeless), schwa-like vowels (which are targetless), and plain coronals (which are [-lateral]). Dogil and Mayor cite several studies indicating that under-specification of features seems to persist from phonology into phonetics with permanently under-specified segments being strongly influenced by coarticulation at the phonetic-motoric level. On the other hand, features that are contrastive in the language are fully specified and less coloured by coarticulation. Such segments include multiply articulated stops, implosives, and clicks (as in the Xhosa language). Dogil and Mayor note that some phonologists argue for eliminating the concept of under-specification in phonological theory (e.g. Mohanan 1991, Broe 1993).

Dogil and colleagues suggested that AOS represents over-specification of phonetic representations, rather than under-specification. This leads to reduced coarticulation and impaired production of under-specified speech sounds (e.g. laryngeal consonants and schwa-like vowels), but does not affect production of highly specified sounds (e.g. clicks). The authors presented data on coarticulation and laryngeal segment production in German individuals with AOS and on production of clicks in individuals with AOS who speak Xhosa. Consistent with prediction, spectrographic analysis revealed considerably reduced or absent coarticulation, which they interpreted as full specification of features for each sound. Furthermore, production of typically under-specified laryngeals in German speakers was impaired while production of typically over-specified clicks in Xhosa speakers was intact. These data were interpreted as support for a linguistically based theory of AOS whereby phonological and phonetic units are fully specified for each distinct articulatory gesture. The authors claimed that this has the cascading effect of severely disrupting coarticulation, which leads to disruptions in speed of articulation and precise timing of articulatory gestures. This representational account of AOS is relatively new and further experimentation is required to determine its explanatory power. However, it cannot explain the observation by Robin and colleagues (e.g. Hageman et al. 1994, Clark and Robin 1998, Robin et al. submitted) that individuals with pure AOS demonstrate nonspeech motoric impairments when tested with motor planning tasks that are more sensitive than the standard clinical battery (see later).

## Experimental analyses with speech tasks

Although theoretical characterizations of the disorder of AOS appear quite clearly to identify specific behavioural manifestations of the disorder, clinical descriptions have lacked diagnostic power, failing to clearly differentiate between certain aphasic syndromes and AOS (Buckingham 1979, Duffy 1995, McNeil et al. 1977). Profiles of speech behaviours observed in individuals with AOS and patients with fluent aphasias, such as conduction aphasia (CA), show considerable overlap (Kent and McNeil 1987). For example, CA has been described as an impairment in (a) phonological encoding that affects generating and maintaining phonological codes (Friedrich et al. 1984), (b) phonemic encoding (Brown 1975), (c) a stage in motor encoding (Yamadori and Ikumura 1975), or (d) pre-articulatory programming (Kohn 1984). Blumstein (1981) observed that descriptions of the speech error patterns of individuals with posterior aphasia are remarkably similar to those in AOS: (a) more frequent consonant errors than vowel errors, (b) more frequent substitutions than distortions or omissions of phones, (c) more errors on initiation of words than in word-final position, (d) consonant cluster reductions, and (e) substitutions that closely approximate the target phoneme.

One reason for the confusion between disorders may be the vague definitions provided for frequently used terminology. For example, although various descriptions of AOS, nonfluent aphasia, and CA have included the phrase 'effortful speech production', no operational definitions are provided that might distinguish the disorder groups. That is, there are no well defined, objective criteria by which to judge the effortfulness of speech in subjects with AOS and differentiate it from that seen in the aphasias. Duffy (1995) suggested that, in AOS, effortfulness may reflect visible and audible grouping of the articulators while McNeil et al. (1997) suggested it reflects inconsistent articulatory errors with successive attempts at the target. In CA, effortfulness may reflect word-finding blocks, perhaps due to inefficient access to a phonological buffer, with persistent attempts at the word level to correct erroneous productions (Buckingham 1979, McNeil et al. 1997). However, objectively and successfully teasing apart these underlying causes of effortfulness may not be possible with the tests available in most clinical settings.

McNeil et al. (1995) set out to describe and differentiate the speech errors of subjects with AOS, ataxic dysarthria, and CA. They postulated that consistency of error location, variability in error type, and successive approximation towards a speech target are factors that may be used to differentiate these subject populations. Subjects produced a set of stimulus words, repeating each word three times following the clinician's model. Notably, the findings contradicted accepted clinical descriptions of the disorders. Subjects with AOS were found to have high consistency of error location within a word and low error type variation within a word. Subjects with CA demonstrated the opposite pattern: low consistency of error location and high error type variation. However, the subjects with ataxic dysarthria performed as expected and were similar to the AOS group. Other findings differentiating CA from AOS were that subjects with CA produced fewer attempts at targets across successive trials, produced most aborted attempts at the word level, and tended to achieve targets after successive attempts. The individuals with ataxic dysarthria produced very few attempts and starters and, therefore, were not included in this part of the analysis. The authors claim that, as individuals with AOS produced most aborted attempts at the single sound level and those with CA produced most aborted attempts at the word level, AOS represents a phonetic-motoric impairment and CA a linguistic impairment.

Other researchers have sought support for the hypothesis that AOS represents errors at the phonetic level while CA represents errors at the phonological level. As Code (1998) points out, phonetic-level errors will have implications for the phonological level particularly in perception by the listener. Code (1998) reviewed a series of studies that presented detailed analyses of the phonetic and phonological errors of these subject groups. Essentially, individuals with AOS tend to simplify articulation of individual phones by producing targets in motorically easier, or unmarked, forms. One specific example of this is the voicing contrast. Voiced consonants represent the marked form of a sound while voiceless consonants represent the unmarked form. Individuals with AOS tend to have difficulty producing voiced consonants (Fry 1958, DeRenzi et al. 1966, MacNeilage 1982). Code and Ball (1982) presented evidence from a single case of AOS that supports the hypothesis that these voicing errors represent phonetic-level impairment rather than a phoneme substitution process. They noted that their subject was unable to generate voicing in voiced fricatives but was sensitive to, and able to manipulate, other phonetic-level features that signal a voiced fricative—duration of the preceding vowel and duration of the friction on the consonant. The individual's errors clearly did not represent impairment at the phonological level.

Examining disturbance of temporal characteristics of speech has proved fruitful in distinguishing motoric from linguistic speech disorders. Seddoh et al. (1996) argued that temporal abnormalities may arise from impairment to either the linguistic or motoric systems but that the nature of the abnormalities will differ. That is, specific time-based information serves linguistic functions. For example, in English syllable timing rules exist that serve to vary the duration of unstressed syllables as a function of the sequential relationship with stressed syllables (Lehiste 1972, Bolinger 1976, Klatt 1976, Cooper et al. 1977). Seddoh et al. (1996) argued that it should be possible to differentiate temporal abnormalities due to motoric versus linguistic impairments. However, we are unaware of any studies that have attempted to do this systematically in AOS.

One frequently cited temporal characteristic of apraxic speech is a reduction in overall speech rate. In detailed analyses of this phenomenon, AOS speakers have been compared to individuals with normal language or aphasia and found to demonstrate discoordination of voice onset time (Freeman et al. 1978, Itoh et al. 1979a, Kent and Rosenbek 1983), increased vowel durations (Freeman et al. 1978, Collins et al. 1983), and increased consonant durations (Kent and Rosenbek 1983). These effects have been observed for multisyllabic words and phrases, and multisyllabic nonsense words and phrases (Bauman 1978, Ryalls 1981, 1987, Collins et al. 1983, Kent and Rosenbek 1983, Mercaitis 1983, Strand 1987, Strand and McNeil 1996). Furthermore, AOS speakers have protracted intersegment durations and transition durations (Kent and Rosenbek 1983, Mercaitis 1983). Other studies have demonstrated abnormal temporal features of speech on acoustic (Collins et al. 1983, Kent and Rosenbek 1983, McNeil et al. 1990a), kinematic (McNeil and Adams 1991), and perceptual (Odell et al. 1990, 1991) measures. These abnormalities all affect the realization of phonemes at a segmental, intersegmental, or word level and may serve to decrease speech rate.

The study by Kent and McNeil (1987) was also designed to examine the nature of the observed slow speech rate of individuals with AOS. They found that the speech of individuals with AOS was slower than individuals with CA and normal subjects, with increased segment and intersegment durations. Individuals with AOS also were unable to effectively increase speech rate on command, suggesting motoric inflexibility. This is contrary to McNeil et al. (1997), who stated that AOS speakers are able to increase their rate of speech but at the expense of phonemic integrity. Kent and McNeil (1987) noted

that, although subjects with CA also demonstrated pauses, these were not as pervasive and their speech attempts demonstrated periods of normal prosody. Although they were able to increase speech rate more effectively than subjects with AOS, indicative of greater motoric flexibility, the number of phonemic and phonetic errors increased at the faster speaking rate. Kent and McNeil (1987) concluded that both disorders represent some degree of phonetic-motoric impairment: (a) CA primarily affects phonetic coding with secondary effects on working memory for phonetic coding and motor control processes, (b) AOS primarily affects motor control processes with a secondary effect on phonetic coding.

Robin et al. (1989) examined the rate of articulator movements during speech as well as inter-articulator temporal co-ordination in five individuals with AOS. They measured peak articulatory velocity in the lower lip as well as co-ordination of articulatory movements in the lower and upper lip in relation to perceptual errors in speech. They found that the subjects were able to generate high peak articulatory velocities during speech and did not demonstrate disco-ordination in the relative timing of upper and lower lip movements. The latter finding is in contrast to Itoh et al. (1979a) who studied co-ordination of lingual and velar movements, although Robin et al. commented that temporal co-ordination between the lips may be less sensitive to disruption (Konno et al. 1987). Furthermore, Robin et al. reported that peak articulatory velocity and co-ordination of relative timing of the lips were not systematically related to speech rate or presence of phonetic errors in speech in the individuals tested. Consistent with previous studies (e.g. Kent and Rosenbek 1983, Kent and McNeil 1987), these subjects had difficulty manipulating rate of speech for syllable- and sentence-level material and demonstrated increased duration of segment and intersegmental components. Robin et al. concluded that while speakers with AOS tend to have a slowed rate of speech with increases in durational elements, these characteristics do not arise from slowed velocity of articulatory movements.

Square-Storer and Appeldoorn (1991) presented an interesting study of the acoustic characteristics of speech in three individuals with pure AOS and related these results to site of lesion. Subjects were diagnosed according to criteria developed at the Mayo Clinic (Darley et al. 1975). Notably, this system does not include dysprosody as a defining characteristic. One subject demonstrated a left basal ganglia lesion, another a left parietal and subcortical lesion, and the third bilateral parietal lesions. One- to four-syllable words were elicited two to three times by having subjects read, repeat, or name objects. Duration of segments, syllables, words, pauses, and additions and amplitude of the syllable nuclei were measured and related to the prosodic features of speech rate and stress patterns. Based on their results, Square-Storer and Appeldoorn suggested that the description of AOS as having unrelieved periods of dysprosody (Kent and Rosenbek 1983, Robin et al. 1989) may be relevant only for more severe cases, as their subject with milder AOS did not demonstrate this feature. Furthermore, the features of slowed speech rate and abnormal stress patterns were demonstrated in the two subjects with subcortical involvement and not in the subject with bilateral parietal damage. Given that the two former subjects demonstrated hemiplegia, the authors suggested that disruptions in rate and stress patterns may be signs of a concomitant unilateral upper motor neurone dysarthria. This study certainly highlights the problems of strictly diagnosing subjects *a priori* when clinical tools that adequately differentiate AOS from other motor speech disorders and some linguistic disorders are not yet available.

Another avenue researchers have employed in attempts to differentiate AOS and CA is degree of variability. The features traditionally used to define AOS serve to give

individuals with AOS the appearance of being variable in their attempts to produce speech. Although increased variability between and within subjects has been used to describe individuals with aphasia and speech motor disorders (e.g. Kent and McNeil 1987), increased levels of variability on temporal measures during speech have been taken to signify instability in motor control systems (DiSimoni 1974a,b, Tingley and Allen 1975, Kent and Forner 1980, Sharkey and Folkins 1985, Janssen and Wieneke 1987, Wieneke and Janssen 1987, Smith 1992, 1994, Smith and Kenney 1994). That is, variability may reveal reduced control in reaching intended motor targets due to impairment. Also, age is a factor with variability decreasing through childhood and increasing again in normal older adults, likely due to maturation and deterioration of the motor control system, respectively (DiSimoni 1974a,b, Tingley and Allen 1975, Kent and Forner 1980, Smith 1992, 1994, Smith and Kenney 1994, Ballard et al. 1999b). With increased variability, AOS speakers are bound to produce erroneous movement patterns, or patterns outside the acceptable range of normal, with greater frequency than normal subjects. Folkins (1985) argues that some of this increased variability may also reflect increased flexibility in dealing with an unstable motor control system to achieve perceptually acceptable tokens.

Several researchers have considered the variability of temporal characteristics of speech in relation to durational measures (Kent and McNeil 1987, McNeil et al. 1989, Robin et al. 1989, Seddoh et al. 1996). Kent and McNeil (1987) considered the temporal characteristics of sentence production in normal subjects ( $n = 3$ ) and individuals with AOS ( $n = 3$ ) or CA ( $n = 2$ ). Subjects produced two sentences at a comfortable rate and a faster rate. Segment durations were significantly longer in the speech of the AOS and CA subjects compared to the normal subjects. However, the AOS subjects demonstrated greatest variability in performance. At the fast speaking rate, the AOS and CA subjects performed similarly in terms of segment duration values and variability. Intersegment durations were longer and more variable in the AOS individuals than the CA and normal individuals, especially at the faster speaking rate. On measurements of voice-onset time, all subjects with AOS and one subject with CA demonstrated longer than normal times. The second subject with CA had voice-onset times close to or briefer than normal. Similarly, both AOS and CA subjects demonstrated more variability in second format transitions than the normal subjects. It seems that some temporal parameters of speech may be affected in both AOS and CA, although the sample sizes in this study were small.

Seddoh et al. (1996) studied four individuals with CA, five with AOS, and normal age-matched controls. They measured accuracy and variability of stop gap duration, voice-onset time, second formant transition duration for a vowel, steady state vowel duration, and consonant-vowel duration in perceptually accurate repetitions of a given phrase (i.e. 'That's a pop/pea/Bob/bee a day'). They found that speakers with AOS and CA were not clearly differentiated by measures of mean duration of segmental and intersegmental components of speech but were differentiated by the amount of variability on these measures. Contrary to Kent and McNeil (1987), Seddoh et al. found that both AOS and CA demonstrated extended segment and intersegment durations indicative of abnormal temporal control during speech. On stop gap duration, AOS had significantly longer mean duration than the CA and normal groups, who did not differ. This was taken to support a motoric deficit in AOS and absence of motor involvement in CA. On vowel duration, both AOS and CA subjects demonstrated longer mean durations than normal, although for the CA subjects this was across all target words and for AOS subjects it was only for words with the tense vowel /i/. The AOS subjects demonstrated greater token-to-token variability as individuals and also significantly greater variability as a group on stop gap duration, vowel duration, and consonant-

vowel duration compared to age-matched normal controls. Compared to speakers with CA, AOS speakers were more variable on stop gap duration and consonant-vowel duration. However, relatively normal levels of variability on voice-onset time and second formant transitions in individuals with AOS prompted the authors to conclude that some aspects of temporal control are preserved. Subjects judged to have a more severe speech impairment demonstrated the greatest token-to-token variability. In comparing the CA speakers and age-matched normal subjects, there was no difference in variability of stop gap duration but CA speakers showed significantly greater variability for consonant-vowel duration. CA speakers did not differ from AOS or age-matched controls on variability of vowel duration. From these data, the authors concluded that both AOS and CA subjects present with abnormal temporal characteristics but the greater variability of the AOS subjects indicates a motoric deficit in AOS and a phonological deficit in CA. However, the results lend support to Kent and McNeil's (1987) conclusion that CA may also involve a subtle sensorimotor deficit. Furthermore, it is possible that some temporal abnormalities reflect cascading effects of higher level phonological impairment (e.g. Dogil et al. 1994, Dogil and Mayor 1998).

Seddoh et al. (1996) also reflected on the issue of motoric flexibility. All tokens analysed in their experimental task were perceptually accurate. However, differences in acoustic characteristics between AOS, CA, and normal subject groups were still detectable. The authors suggested that subjects with AOS demonstrate a greater flexibility in compensating for their motoric instability so that they still achieve perceptual accuracy in a constrained task. This interpretation is consonant with that of Folkins (1985). However, the observations by Kent and McNeil (1987), that the inability of their AOS subjects to manipulate speech rate reflected motoric inflexibility, suggest that the ability to compensate is limited.

Further support for increased variability in AOS comes from McNeil et al. (1989) who studied variability of peak articulatory velocities of the lower lip in four subjects. They found that, in word targets, the subjects with AOS were more variable than normal but did not differ on mean velocity.

Taken together, the studies just reviewed best support theoretical explanations of AOS that place the locus of the deficit in motor planning processes affecting the translation of an intact phonological representation of a message into the phonetic-motoric representation prior to execution by the articulators (Kelso and Tuller 1981, Kent and Adams 1989, McNeil et al. 1997, Van der Merwe 1997, Whiteside and Varley 1998). This disruption would affect retrieval of motor plans and/or specification of the spatiotemporal parameters of movements. These disruptions would manifest as trial and error groping as the individual attempts to retrieve a motor plan or sequence motor plans, distortion of phones, segmental and intersegmental durational changes with reduced speech rate, and loss of interarticulator co-ordination. The framework of Van der Merwe is the most explicit and can account for all of these disruptions. Capacity limitation theories, such as Rogers and Storkel (1999) provide an alternative perspective but may only account for some of these characteristics. However, none of these theories addresses the now robust literature demonstrating that the motor control impairments of AOS extend to nonspeech movements.

#### Experimental analyses with nonspeech tasks

Although it is necessary to consider the impairment of AOS in the context of speech production tasks, also studying nonspeech behaviours has the potential to disambiguate which characteristics are a result of the underlying motor impairment and which are

related to the interaction between the motor and linguistic systems (see Folkins and Bliede 1990, Robin et al. 1997 for details supporting this argument). Several studies have reported that individuals with AOS demonstrate impaired movement of the articulators during both speech and nonspeech tasks (De Renzi et al. 1966, Itoh et al. 1979a, McNeil and Kent 1990, McNeil et al. 1990b, Square-Storer et al. 1990, Hageman et al. 1994). Pursuing this line of research promises to reveal the motoric disturbances that give rise to the profile of intact and disrupted perceptual and acoustic characteristics that have been detailed (see McNeil et al. 1997 for an extensive review). While there is a widely held view that speech and nonspeech oral movements are controlled by separate systems, as indicated by dissociations between oral apraxia and AOS (e.g. Wertz et al. 1984, Duffy 1995), this is probably due to most of these studies using traditional clinically based oral movement tasks that create substantially less motoric demands than does speech.

Nonspeech tasks that better capture the complexity of speech production show breakdowns in both speech and nonspeech movements in AOS. As such, the motoric disturbances underlying AOS can be revealed in nonspeech tasks provided that the measure is sensitive to such disturbances. It is likely that standard clinical measures of oral and speech motor programming often lack the sensitivity to detect these disturbances, particularly in cases of milder impairment. This may be demonstrated in the study by DeRenzi et al. (1966). They attempted to examine the correlation between the presence of oral apraxia and 'phonemic-articulatory disorder' in right, left, and non-brain-damaged individuals. Oral apraxia was examined using traditional clinical tasks such as protruding the tongue, whistling, and chattering teeth. They found that oral apraxia was present in most subjects with Broca's aphasia, and presumably AOS. DeRenzi et al. concluded that the same cortical areas or mechanisms subserved speech and nonspeech volitional movements such that disorders of both must coexist. However, in three cases, severe Broca's aphasia was not accompanied by oral apraxia. Here DeRenzi et al. argued that, with recovery of the underlying system, performance of less complex nonverbal movements had returned but the more complex speech movements had not. At least two alternative explanations exist. First, it is possible that the expressive impairment in these three subjects was not 'articulatory' in nature, and so AOS was not present. Alternatively, subtle nonspeech impairments may have been present but not detectable with the clinical tasks utilized.

A number of more sophisticated methods to examine nonspeech motor control have been developed which are thought to be sensitive to the demands on the motor system during speech. These include tasks requiring subjects to control static position or isometric force of articulators (Barlow and Abbs 1986, McNeil et al. 1990b) and pursuit tracking of a visual signal with the articulators (McClellan et al. 1987, Moon et al. 1993, Hageman et al. 1994, Clark and Robin 1998, Ballard et al. 1999b, Robin et al. submitted). In these tasks, a target level of force or position or a target movement pattern is displayed visually, for example on an oscillographic screen. A transducer is placed on the articulator of interest and the transduced signal is overlaid on the visual display. The subject is instructed to match the two signals and so achieve a given level of force or a target position for a given period of time or, in the case of visuomotor tracking, follow a given movement pattern. These techniques have clear application to assessment of static and dynamic aspects of motor control of the articulators.

Barlow and Abbs (1986) examined the stability of force in the lip, jaw, and tongue in subjects with spastic dysarthria and related these nonspeech measurements to perceptual accuracy of speech. Control of muscle force was more variable, or less stable, than in normal controls and demonstrated a significant correlation with perceptual judgements

of speech intelligibility. McNeil et al. (1990b) compared subjects with AOS, ataxic dysarthria, CA, and normal controls on isometric force and static position control. According to the traditional view of AOS, one would not predict impairments in muscle force. They reported significantly less stable control of both static position and force in AOS and ataxic dysarthria compared to control subjects. It is noted that the mechanism underlying the similarity in performance between AOS and dysarthria is not necessarily the same. Performance of individuals with CA fell between normal controls and the apraxic and dysarthric groups. Consistent with previous studies by McNeil and Colleagues, McNeil et al. (1990b) suggested that CA involves some degree of orofacial sensorimotor impairment coexisting with the primary linguistic deficit.

The visuomotor tracking (VMT) has long been utilized by researchers studying normal motor control and motor skill learning in the limbs (see Poulton 1974) and impaired motor control of upper extremities in Parkinson's Disease (e.g. Flowers 1978). It was first applied to the oromotor system by McClean et al. (1987) who examined normal subjects and individuals with acquired dysarthria. Applying the schema theory of motor control (e.g. Schmidt 1975, Schmidt and Lee 1999), this paradigm assumes that a specific 'programme' or stored plan of movement is implemented during, for example, jaw closure regardless of the behavioural context. However, the parameters of that motor programme, such as the absolute speed and amplitude of the movement, differ with context. That is, a single motor programme that drives jaw closure may be implemented during nonspeech VMT and speech but the absolute speed and amplitude of the movement (i.e. parameters of the motor programme) will differ for these two activities.

In visuomotor tracking with the articulators, subjects gaze at an oscilloscope screen and use their lower lip, jaw, or larynx (i.e.  $F_0$ ) to track the movement of a horizontal bar (i.e. the target signal) with a cursor (i.e. the tracker signal). With laryngeal tracking, subjects prolong the spoken vowel /a/ and track the target by changing pitch. Three predictable target signals (i.e. 0.3, 0.6, and 0.9 Hz) and an unpredictable target signal (i.e. a complex signal composed of ten equal amplitude frequencies from 0.1 to 1 Hz) have been used. The velocity and amplitude of movements required by the task are within the range used during speech and, to some degree, approximate movements that occur during speech.

Hageman et al. (1994) tested the jaw, lower lip, and voice tracking abilities of normal subjects and individuals with AOS. They reported that normal subjects' accuracy in tracking declined as the frequency of predictable signals increased and tracking was poorest for the unpredictable signal. On predictable signals, AOS subjects were consistently poorer than normal controls but demonstrated a similar decline in accuracy with increasing frequency. While the normal controls tracked the predictable targets with smooth articulator movements, the subjects with AOS produced 'jerky' movements. Notably, subjects with AOS tracked unpredictable target patterns more accurately than predictable targets and at a level of accuracy similar to normal subjects. The authors argued that, for predictable targets, normal subjects were able to formulate and follow an internal model, or motor programme, of the target signal (Hageman et al. 1994). That is, subjects initially perform in a reactive, or feedback, mode, having to closely monitor the target in order to extract a pattern and develop a motor programme to execute the task. Once the programme is developed, subjects move into a predictive, or feedforward, mode, implementing the programme with only occasional sampling of the target to ensure accuracy and execute corrections if accuracy is deficient. Subjects with AOS, on the other hand, seemed unable to develop such an internal model or

programme. They appeared to remain in a reactive mode, relying solely on feedback to execute the task. This interpretation is supported by the 'normal' tracking of the unpredictable signal by subjects with AOS. In unpredictable target tracking, it is not possible to develop a programme of movement and so both subject groups are forced to use feedback mechanisms to execute the task. Hageman et al. concluded that AOS reflects an impairment in developing or implementing motor programmes for articulator movements both in speech and nonspeech tasks.

Hageman et al. (1993) compared subjects with AOS, ataxic dysarthria, and normal controls. They reported that, contrary to individuals with AOS, those with ataxia demonstrated poorer correlations between target and tracker than normal subjects for both predictable and unpredictable signals. These results indicate that ataxic dysarthria is a motor execution disorder, rather than a motor programming disorder, so that tracking is affected for both target types.

A recent study by Robin et al. (submitted) considered the relation between nonspeech VMT ability and perceptual measures of speech accuracy in individuals with pure AOS and CA. Subjects tracked both predictable and unpredictable targets with the lower lip and jaw. Consistent with previous work (Hageman et al. 1993, 1994), tracking performance for individuals with AOS was poorer than normal for predictable signals only, and individuals with CA were not differentiated from normal subjects in either condition. For only those subjects with AOS, performance on tracking predictable signals was highly correlated with measures of speech accuracy. Given that other behaviours such as peak articulatory velocity (Robin et al. 1989), temporal co-ordination of upper and lower lip movements during speech (Robin et al. 1989), and accuracy and variability of segment and intersegment durations (Seddoh et al. 1996) have not proven useful in predicting speech accuracy, these findings support the use of the tracking paradigm in assessment of AOS. Furthermore, these findings strongly support the notion that AOS is a disorder of motor planning that affects articulator movements in both speech and nonspeech tasks, whereas CA is primarily a linguistic impairment.

Clark and Robin (1998) further explored the concept of a motor programming impairment in AOS by using VMT to examine the formation of motor programmes and the setting of temporal and amplitude parameters in normal controls and individuals with AOS or CA. They predicted that all subjects with AOS would show reduced accuracy of the motor programme and that some may show reduced temporal and/or amplitude parameterization accuracy. Furthermore, they predicted that individuals with CA would perform similarly to normal controls, as they do not have a motor programming impairment. A variation on the VMT task was employed with the jaw only. Each subject was required to gaze at a monitor, study a given movement pattern (i.e. a waveform) that appeared on the monitor, and then attempt to replicate the pattern by opening and closing the jaw after the pattern had disappeared from the screen. Two seconds later, feedback was provided by displaying the subject's production superimposed on the target waveform. Accuracy of the motor programme was determined by calculating the residual or relative difference between the subject's production and the target waveform, after correcting for absolute temporal and amplitude differences. Accuracy of temporal and amplitude parameters was determined by calculating absolute differences between the duration and amplitude of the subject's production and the target waveform.

Contrary to prediction, Clark and Robin (1998) found dissociation between motor programme accuracy and parameterization accuracy among their AOS subjects. Either motor programme formation or parameterization was impaired, but not both. This

dissociation was also observed within-subject, where individuals with AOS were able to improve parameterization accuracy but only at the expense of motor programme accuracy or vice versa. The authors suggested that these findings may reflect individual differences in resource allocation strategies. It appears that the process of developing, recalling, and/or implementing a reasonably accurate GMP or of manipulating parameter values may be more demanding of attentional resources in the individual with AOS. Therefore, in a finite system, fewer resources are available for one or the other, and performance deteriorates along one dimension with the individual being forced to choose which of the two processes to execute more accurately.

In summary, nonspeech physiological research into AOS has indicated that subjects with AOS have difficulty with developing or implementing movement programmes and/or setting the absolute timing and amplitude parameters of those programmes. It appears that this impairment also may have an impact on the availability and allocation of attentional resources for performing and adapting actions (Clark and Robin 1998, Rogers and Storkel 1999). The difficulty with motor control for nonspeech tasks appears to be one of the most consistent findings in the literature and is not wholly unexpected (see later). These data demand that models of AOS focusing predominantly or entirely on speech production need substantial revision. As is the case with the dysarthrias (e.g. Darley et al. 1975, Duffy 1995), where the basic nonspeech condition and movement control define the speech anomaly, we propose that AOS must be defined at multiple levels and the fundamental impairment needs to be considered at the level of nonspeech motor control.

The nonspeech motor control problems of AOS can be accounted for from a number of different theoretical postures. For instance, our work has largely been guided by theories of motor programming and, in particular, the concepts of generalized motor programmes and the parameters that set absolute aspects of programmes (e.g. Schmidt and Lee 1999). The nonspeech data can also be interpreted within recent models of dynamic systems (e.g. Thelen and Smith 1994, Kelso 1995). In the following discussion we present these two models of motor control and propose a broader view of AOS that encompasses both nonspeech and speech motor control difficulties.

#### A broader view of acquired apraxia of speech

Although current theories of AOS account for the effects of the impaired motor system on speech with varying degrees of specificity, they do not explain the effects on nonspeech actions. We propose that, in order to incorporate findings from nonspeech tasks, we need to conceptualize AOS as a disorder of motor control that also may have an impact on the availability and allocation of attentional resources for performing and adapting actions (Clark and Robin 1998, Rogers and Storkel 1999). We rely on such theoretical stances as that presented by Folkins and Bliede (1990) and Saltzman (1986) which claim that the motor system is not necessarily organised around presumed units of language or speech. Rather, it is assumed that the motor system has its own cognitive architecture that is activated and monitored, in part, by the language system.

We propose, on a theoretical basis and from empirical data, that AOS is a motor control disorder that has its basis in nonspeech anomalies. When these motor control anomalies are such that they interfere with the production of speech, a phonetic-motoric disorder emerges that is called AOS. In our view, the issue that confronts scientists and clinicians is how to model the underlying motor anomalies in AOS. Once this has been accomplished, extension of such modelling to include speech can occur. Two opposing

theories of motor control have explanatory power in understanding both the speech and nonspeech profiles of AOS—schema theory (e.g. Schmidt 1975, Schmidt and Lee 1999) and dynamic systems theory (e.g. Kelso 1995, Thelen & Smith 1994).

Schema theory (Schmidt 1975, Schmidt and Lee 1999) has been one of the most influential theoretical approaches to motor control and learning, and was developed through the study of normal individuals learning novel limb movements or performing skilled actions. Briefly, this theory assumes that learning results from developing and refining a representation of an action that prescribes the relation between how we move and the demands of a given task. Two primary concepts in schema theory are generalized motor programmes (GMP) and parameters. GMPs contain an abstract code about the relative timing of actions and the relative force with which actions are to be produced. Parameters specify details about how a GMP will be expressed in terms of absolute duration of movement, absolute force of muscle contractions, and the muscles or limb used to make the movement. Through manipulation of parameters, numerous and even novel actions can be completed using a single GMP. These two concepts in combination serve to reduce storage demands for representations of action and explain the relatively invariant features across different productions of an action. As with most fields, there is terminological confusion when applying this work to the area of speech motor control. For example, in the model of Van der Merwe (1997) described earlier, the term 'motor plan' seems to correspond rather directly to Schmidt's concept of GMPs and the term 'motor programme' to Schmidt's concept of parameters.

AOS has traditionally been defined as a disorder of motor programming (e.g. Darley et al. 1975). Within the framework of schema theory, the speech errors are considered to be the result of disruption to the programmes that drive sound production. There is a historical precedent to using schema theory to explain the speech impairment in AOS (e.g. Mlcoch and Noll 1980, Kent and Rosenbek 1983). The issue of defining what constitutes a motor programme in speech production is far from resolved (e.g. Smith et al. 1995). However, if one assumes that there are GMPs that need to be parameterized for speech production at some level, then AOS can be thought of as a breakdown in activation and selection of GMPs, in specification of the parameters for those programmes, or both. Importantly, this model can also account for the nonspeech findings in AOS. Specifically, Clark and Robin (1998, see earlier) reported that individuals with AOS were unable to execute a model jaw movement pattern accurately and appeared to trade-off accuracy of GMP execution for parameterization accuracy, or vice versa. We interpret the tracking data described earlier within this model. Normal subjects are able to develop a GMP rapidly in order to perform visuomotor tracking of predictable targets in a predictive mode. They are also able to manipulate the temporal parameter adeptly so as to track predictable signals of increasing frequencies with high accuracy. Individuals with AOS have difficulty in either developing or implementing the GMP. They are also unable to manipulate the temporal parameter to produce a given speed of movement in the context of the visuomotor tracking task. Tracking of unpredictable targets does not permit development of a GMP and performance on this condition does not distinguish individuals with intact motor control and those with AOS.

An alternative approach to motor control is based on dynamic systems (e.g. Thelen & Smith 1994, Kelso 1995). Rather than rely on concepts of motor programmes and parameters, proponents of the dynamic systems approach argue that the 'invariant' features in movement are represented as 'attractor states'. In relation to motor skills, these states are action patterns that emerge through (a) the interaction of the parts of a system with each other and with the external environment, (b) inherent constraints on

the system, and (c) the available supply of energy. In essence, each time one performs an act, it emerges as a new form. Some aspects of the act will be stable and predictable over repeated performances while others will reflect the variable, flexible, and adaptive aspects that take their form according to changes in the stimuli that were present at the time of a given action. As a given set of conditions reoccurs, the emergent behavioural patterns increase in stability and develop into an attractor state, and the order and complexity in the system may increase. The nature of dynamic systems permits great plasticity to adapt and reorganize in response to new conditions and to learn new acts. These phenomena are demonstrated in the developing human as new behaviours are learned and adaptations are made to accommodate the changing cognitive and neuromuscular systems (Thelen and Smith 1994). Similarly, as changes in cognitive and neuromuscular systems occur with ageing or with impairment, stability is disturbed and patterns of behaviour are disrupted or lost.

Speech sounds can be viewed as emergent properties, with production of speech sounds in the adult representing attractor states with the behavioural goal of perceptual adequacy. In this model, AOS can be viewed as a breakdown in some aspect of attractor state formation. It is possible that, in the impaired individual, the system cannot self-organize so that stable behavioural patterns do not emerge. Alternatively, some states may be pathologically stable, or inflexible (Folkins 1985), so that the system remains in that state when it is no longer appropriate (e.g. perseveration, reduced ability to manipulate speech rate). Furthermore, the system may achieve attractor states but be overly sensitive to perturbation or feedback, or be unable to adapt effectively to such perturbation or feedback. Kelso and Tuller's (1981) coalitional theory of AOS is based on early formulations of dynamic systems theory within the field of speech motor control. The dynamic systems approach accounts for the nonspeech data described earlier. The data of Clark and Robin (1998) appear to implicate a system that demonstrates either reduced adaptation to stimulus variations or adequate sensitivity to such changes but difficulty self-organizing into an appropriate attractor state. Data from the VMT studies (Hageman et al. 1993, 1994, Robin et al. submitted) suggest that, when tracking predictable signals, individuals with AOS have difficulty achieving or remaining in a stable state. The unpredictable tracking condition, however, does not permit a behavioural pattern to emerge and, again, normal and impaired individuals perform similarly.

Although quite speculative at this point in time, these theories provide a framework for research on clinical applications addressing description, diagnosis, and treatment of motor speech disorders. Specifically, we present three clinical issues that bear directly on the relation between speech and nonspeech motor control in AOS. First, it is known that there are individuals with AOS who perform within normal limits on routine oral motor examinations that include tasks such as alternating labial protrusion and retraction. Our contention (see Robin et al. 1997) is that most nonspeech motoric tasks used in clinical settings are too far removed from speech so that they permit detection of only fairly severe apraxic disturbances. Given that even patients with very mild AOS perform abnormally on the visuomotor tracking task described earlier, this task appears to be a sensitive measure of praxis for nonspeech oral motor tasks. Considering the converse situation, we predict that any individual who performs poorly on the relatively less sensitive routine oral motor exam will show some degree of AOS.

A second issue relates to development of a diagnostic marker for AOS. As is the case with most disorders of speech and language, a systematic approach to accurate diagnosis of AOS will most likely stem from a detailed and sensitive range of both nonspeech and

speech tasks. Data from our laboratory (e.g. Hageman et al. 1994, Clark and Robin 1998, Robin et al. submitted) are intriguing to consider in this light. Although preliminary in nature, our studies of visuomotor tracking ability have demonstrated that every individual with AOS that has been tested to date has an impaired ability to track predictable targets in the face of 'normal' tracking of unpredictable signals. These data lead to the hypothesis that nonspeech visuomotor tracking performance is a diagnostic marker for AOS. If this hypothesis holds, then the presence of impaired tracking of predictable targets but normal tracking of unpredictable targets, regardless of other cognitive or linguistic (i.e. aphasia) problems, will support a diagnosis of apraxia. Note that Shriberg et al. (1997b,c) have argued that prosodic abnormalities may be diagnostic markers for developmental apraxia of speech. However, like many models of AOS, their model fails to account for the nonspeech findings in the childhood apraxia literature and does not consider the fact that prosodic anomalies are robust throughout all motor speech disorders including the dysarthrias.

The final clinical issue that has driven our work with AOS is the development of a principled approach to treatment. Our findings over a range of experimental contexts have led us to the area of motor control and learning (Schmidt and Bjork 1992, Schmidt and Lee 1999) as one that can provide a model-driven approach to treatment of motor speech disorders. We review approaches to treatment and their efficacy next, and present some preliminary data supporting the application of specific principles of motor learning (Schmidt and Lee 1999) developed and tested in other motor systems.

### Treatment

Taking into account the findings from the studies of both speech and nonspeech tasks described earlier, well motivated and principled approaches to treatment can be developed based on broader theories of motor control and learning. To date, surprisingly few studies exploring treatment approaches to AOS and efficacy of these approaches have been published (Wambaugh and Doyle 1994). The majority of these studies have proposed methods that presume to alter the organization of the neuromotor system, and thus bring about relearning of motor speech skills. Specifically, they focus on developing motor plans or programmes by retraining the production of spatiotemporal aspects of sounds and sound sequences. Many do not directly provide practice in independently recalling these newly developed, or relearned, motor plans—an approach that would likely facilitate long-term retention and transfer to novel contexts (see later). Many typically do not focus on varying the phonetic context in which sounds are practised with the explicit goal of affecting coarticulation or interarticulatory co-ordination. References to available theories of motor control and learning are rare in these studies and treatment goals typically are not overtly motivated by theoretical accounts (but see Wambaugh et al. 1996, 1999). In the context of Van der Merwe's framework (1996), AOS may represent an impairment in one or more of the following processes: (a) retrieval of motor plans for phones, (b) sequential organization of movements for a single phone or a series of phones, (c) adaptation to phonetic context in the form of coarticulation, (d) interarticulatory co-ordination, (e) central monitoring by comparison with an internal stored representation of the plan, and (f) relaying the motor plans to the motor programming level for specification of parameters of movement. Although some of these processes may not be amenable to intervention, it seems relevant to determine which are affected for a given individual and structure therapy accordingly.

In direct contrast to the speech motor learning literature, numerous studies have considered motor learning of limb movements in normal children and adults. Many of these studies are based on the schema theory of motor control and learning (Schmidt 1975, Schmidt and Lee 1999). In reviewing this body of literature, it appears that development of flexible and skilful actions is influenced by several principles of motor learning that pertain to the structure of practice and the nature of feedback (see Schmidt and Lee 1999 for an extensive review). These principles are listed in table 1. The critical point that can be derived from this literature is that, in training motor skills, performance during practice is not a good indicator of learning. Schmidt and Bjork (1992) observed that conditions present during practice could facilitate acquisition of a skill, as measured during the practice session. However, these same conditions may actually interfere with, or minimize, long-term retention and transfer to novel contexts and novel responses. Obviously, retention cannot occur without acquisition, but this research indicates that retention is the more informative measure of a training programme's efficacy.

Although these findings have not yet been rigorously tested in the motor speech system, they have serious implications for treatment of motorically based speech disorders such as dysarthrias and acquired and developmental apraxia of speech. It is reasonable to expect that principles of motor learning will apply equally to the speech motor system. A few specific principles, such as performing a high number of trials, have been applied in many treatment programmes for AOS (e.g. Phonetic Placement: Van Riper and Irwin 1958, Eight-Step Task Continuum: Rosenbek et al. 1973, Melodic

Table 1. Principles of motor learning that enhance acquisition or retention of motor skills.

Principle	Enhances Acquisition	Enhances Long-Term Retention
<b>Structure of Practice</b>		
Order of stimulus presentation		
Blocked	✓	✗
Random	✗	✓
Number of trials in a practice session		
Low (5–20)	✗	✗
High (≥ 50)	✓	✓
<b>Structure of Feedback</b>		
Frequency of feedback		
High frequency (100% of trials)	✓	✗
Low frequency (30–60% of trials)	✗	✓
Type of feedback		
Knowledge of performance <sup>1</sup>	✓	✗
Knowledge of results <sup>2</sup>	✗	✓
Timing of feedback		
Immediate	✗	✗
Delayed 3–4 seconds	✓	✓
Structure of feedback delay		
Filled with extraneous movement	✗	✗
Silent	✓	✓

<sup>1</sup> Knowledge of performance feedback provides information on specific aspects of how a response was performed.

<sup>2</sup> Knowledge of results feedback provides information only on correctness of a response.

Intonation Therapy: Sparks et al. 1974, Prompts for Restructuring Oral Muscular Phonetic Targets [PROMPT]: Chumpelik 1984, Square et al. 1985, Phonetic Derivation and Phonetic Approximation: see Square-Storer 1989, Phonetic Contrasts or Minimal Pairs Treatments: e.g. Hill 1978, Wambaugh et al. 1998). Furthermore, several programmes encourage varied or random practice on several targets in order to prevent perseveration (e.g. Melodic Intonation Therapy: Sparks et al. 1974, Minimal Pairs Treatment: Hill 1978) and facilitate retention (Wambaugh et al. 1999). However, little empirical evidence has been amassed to support or refute the application of these and other motor learning principles in treating the speech production impairments of AOS. It is also the case that some treatment programmes that purport to be 'motor-based' actually have procedures that are in direct antithesis to the principles of motor learning. For example, although not explicitly stated, many treatments are provided with immediate high-frequency feedback that gives information about how an action was performed (i.e. knowledge-of-performance feedback, see table 1). Others include drill practice where single speech targets are practised in blocked order (e.g. Darley et al. 1975). Finally, the focus of the majority of studies has been on either acquisition or short-term retention performance during treatment rather than retention of skills post-treatment and transfer of treatment effects (Wambaugh and Doyle 1994).

Wambaugh and Doyle (1994) reviewed 28 studies reporting on the efficacy of treatment for AOS. Of the 26 studies that treated speech production, as opposed to alternative communication modalities, the focus was on performance during the treatment phase. Only eight studies reported on post-treatment retention of targeted skills (Warren 1977, Simmons 1980, Lane and Samples 1981, Thompson and Young 1983, LaPointe 1984, Square et al. 1985, Dworkin et al. 1988, Raymer and Thompson 1991), with four of these claiming positive results. Six studies reported on transfer to untreated responses (Thompson and Young 1983, LaPointe 1984, Square et al. 1986, Dworkin et al. 1988, Square-Storer and Hayden 1989, Raymer and Thompson 1991), with one study claiming positive results (Square et al. 1986) and three only limited transfer. Three studies systematically tested transfer to untreated stimuli (Southwood 1987, Dworkin et al. 1988, Raymer and Thompson 1991), with one claiming positive results (Dworkin et al. 1988). Notably, four of the eleven studies reporting on long-term retention and/or transfer did not utilize a controlled experimental design (Lane and Samples 1981; Square et al. 1985, 1986, Square-Storer and Hayden 1989) so that results from these studies must be interpreted with caution. The studies by Square and colleagues (Square et al. 1985, 1986, Square-Storer and Hayden 1989) applied the PROMPT method. However, a subsequent single-case study reported by Freed et al. (1997) demonstrated positive retention effects for PROMPT using a well controlled multiple baseline across behaviours design. This study was not designed to examine transfer across responses and stimuli. Of the remaining seven reports, the majority employed drill-type activities with high numbers of trials. No studies systematically tested the effects of principles of motor learning on acquisition of treated skills and retention and transfer of treatment effects.

Since the review of Wambaugh and Doyle (1994), Wambaugh et al. (1998) have published a treatment efficacy study using a minimal pairs treatment approach. They employed multiple baseline designs across speakers and behaviours targeting one minimal pair phoneme contrast (e.g. / $\int$ /-/s/), at word level, in each phase of treatment. In experimental probes given throughout the treatment phase, the three subjects demonstrated short-term retention of all or most of the targeted phonemes in trained words and demonstrated transfer to production of the target phonemes in untrained words. Treated behaviours that reached criterion levels during treatment were retained

at testing six weeks post-treatment, but transfer to novel responses and novel phrase- and sentence-level stimuli was limited. This treatment protocol involved presentation of targeted behaviours in blocks with high-frequency verbal feedback. It is possible that retention may have been facilitated through application of principles of motor learning such as low-frequency delayed feedback or training several phoneme contrasts in parallel (Wambaugh et al. 1999, see table 1).

Ballard et al. (1999a) presented the first study to test the influence of principles of motor learning on treatment for AOS. They examined the effect of practising speech skills in random versus blocked order in the context of Phonetic Placement Therapy (Van Riper and Irwin 1958). In this preliminary study, multiple baseline designs across subjects and behaviours and an alternating treatments design (McReynolds and Kearns 1983) were combined to study two subjects with severe AOS and concomitant oral apraxia and aphasia. For one subject, the targeted behaviours were consonant-vowel (CV) syllables and, for the other subject, CVC words. Both subjects demonstrated less erratic performance during acquisition on behaviours treated in random order compared to those treated in the blocked condition. Consistent with prediction, retention performance was superior for the behaviours trained in the random practice condition. Neither subject demonstrated transfer to untrained responses. However, the first subject transferred the treatment effects to an untrained stimulus (i.e. a probe task), with retention of transfer effects post-treatment being greatest for those behaviours treated in the random condition. Although this study represents preliminary data, results are positive and warrant continued work in this area.

### Summary and conclusions

Significant advances have been made in recent years towards understanding the nature of the motor speech disorder of AOS. Acoustic, kinematic, and perceptual studies of speech have led to more refined definitions of the behavioural characteristics for use in differential diagnosis. New theoretical accounts have been proposed that are embedded within well known models of speech and language production. Development and application of methods for studying nonspeech motor control are providing new insights, indicating that the motor control impairment of AOS is more pervasive than previously thought. We have provided a broader account of AOS to accommodate these data and claim that the disorder involves a fundamental impairment of praxis in the articulatory motor system that crosses both speech and nonspeech motor tasks.

Many theoretical and analytical developments in this field have not yet been incorporated into treatment approaches for AOS. Furthermore, few treatment studies have taken advantage of the extensive body of literature supporting the use of specific principles that maximize learning of motor skills in other systems. The time is at hand to re-examine AOS with the goal of modifying existing approaches to treatment, developing new approaches, and rigorously comparing the efficacy of these manipulations. Current theoretical accounts now provide a stronger framework for setting specific treatment goals and examining transfer to related responses and stimuli. Treatment programmes that are driven by a principled account of how the motor system learns should provide the most efficient and effective framework for treating motor-based speech disorders. In turn, well controlled and theoretically motivated studies of treatment efficacy promise to stimulate development of these theoretical accounts and contribute to our understanding of this enigmatic disorder.

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