Generalized motor programme and parameterization accuracy in apraxia of speech and conduction aphasia

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Abstract

The present study examined three aspects of motor programming (generalized motor programme (GMP) accuracy, temporal parameterization accuracy, and amplitude parameterization accuracy) in subjects with apraxia of speech (AOS) or conduction aphasia (CA) and normal speaking participants. Subjects were presented with a movement pattern on a monitor that they were required to produce with the jaw, after the target pattern had been removed from view. Analyses examined differences in relative (parameterization) and absolute (GMP) timing and amplitude between the target and actual movement. Examination of individual subject performance revealed inter-subject variability within the AOS group, with two of the four subjects demonstrating unimpaired GMP accuracy but poor parameterization accuracy, while the other two subjects exhibited the opposite pattern, impaired GMP accuracy but normal parameterization. No clear pattern of deficit was noted for the subjects with CA. Results are discussed with respect to motor control theories of AOS and CA.

Introduction

Apraxia of speech (AOS) has been the subject of controversy for the past several decades. Although historically the debate focused on whether AOS was primarily linguistic or motoric in nature, more recently the focus of research has been on characterizing the nature of the underlying motoric impairment (e.g. McNeil *et al.* 1990a, Robin *et al.* 1989). A predominant conceptualization of AOS is that the behavioural characteristics stem from a 'motor programming' impairment (Darley 1969, Kent and Rosenbek 1983). From this perspective, individuals with AOS have difficulty developing and/or executing motor programmes that specify the temporal and/or spatial goals of the articulators for perceptually adequate speech. However, an operational definition of the 'motor programme' has been absent and the systematic examination of motor programming in subjects with AOS has been non-existent.

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Schmidt (1975, 1988) developed a motor programming model for learned movement patterns which includes the notion of *generalized motor programmes* (GMP). GMPs are open-loop controlled movement structures which form the basis for related groups of actions. Schmidt operationally defined the GMP as the relative timing and/or forces of an action which can be examined by measuring the time or amplitude relations among kinematic landmarks of a movement pattern (e.g. Young and Schmidt 1991). Schmidt defines *parameters* as the absolute timing and forces of actions that serve to scale GMPs for individual movement patterns. Because a single GMP may be activated for several related actions, a finite number of GMPs are necessary to complete an infinite number of potential actions, as different parameters are assigned for each variation of the movement pattern. For example, within this framework, a single GMP may be activated during all instances of a jaw-closing gesture. However, the speed and amplitude of the gesture is varied by assigning different parameters to the GMP for each specific action.

Within Schmidt's model, different aspects of motor programming might be disrupted in AOS. For example, individuals with AOS may have difficulty developing or executing the GMP. Alternatively, their impairment may stem from an inability accurately to parameterize an intact GMP. Finally, a combination of these two factors may underlie their motor control difficulties. Based on definitions of AOS provided by Darley *et al.* (1975) and current data, a breakdown in GMP appears to be a promising explanation for AOS, since the extant literature provides examples of potential disruptions of relative timing during speech tasks (e.g. Kent and McNeil 1987, Kent and Rosenbek 1983, Seddoh *et al.* 1996), as well as inaccurate motor planning during non-speech tasks (Hageman *et al.* 1994). However, preliminary evidence that parameterization may also be disrupted in AOS was provided by Hageman *et al.* (1994).

In recent studies, the production of speech and non-speech movements of subjects with AOS has been compared to that of subjects with conduction aphasia (CA) (McNeil and Adams 1991, McNeil *et al.* 1990a, 1994, Odell *et al.* 1991a, b, Robin *et al.* 1998, Seddoh *et al.* 1996). Individuals with CA demonstrate speech sound errors similar to those displayed by individuals with AOS (McNeil and Kent 1990, Odell *et al.* 1991a, b), but the errors produced by these individuals are generally believed to result from an underlying phonological impairment associated with aphasia. McNeil and Kent (1990) proposed that some of the speech errors observed in CA may also be attributed to breakdowns in motor control. This argument is based primarily on observations of the acoustic signal or articulator movements obtained during speech samples (McNeil and Adams 1991, McNeil *et al.* 1990b, 1994) during which subjects with AOS and CA exhibit performance deficits implicating breakdowns in motor control. However, during non-speech motor control tasks, subjects with CA do not reliably show performance deficits (Hageman *et al.* 1994, McNeil *et al.* 1990a).

The present study represents a preliminary examination of GMP and parameterization accuracy in subjects with AOS or CA during a non-speech oral motor task. It was predicted that all subjects with AOS would exhibit impaired GMP accuracy (although some subjects with AOS might also exhibit reduced parameterization accuracy). It was further predicted that subjects with CA would exhibit GMP and parameterization accuracy similar to that of non-brain-damaged controls.

Method

Subjects

Two experimental subject groups were recruited, AOS and CA. A subject was included in the AOS group if he/she exhibited effortful trial-and-error positioning of the articulators, dysprosody, error inconsistency across repetitions, and notable difficulty in initiating utterances (Kent and Rosenbek 1983). In addition, each subject had to exhibit these symptoms as a result of a single left-hemisphere lesion and to be at least 1 year post-onset (see table 1). All subjects exhibited some level of concomitant aphasia as measured by the *Boston Diagnostic Aphasia Examination* (BDAE) (Goodglass and Kaplan 1983), the *Western Aphasia Battery* (WAB) (Kertesz 1982), or the *Multilingual Aphasia Examination* (MAE) (Benton and Hamsher 1989), but not to the extent that it interfered with their ability to complete the task. Subjects who exhibited oral weakness or incoordination were not included in this experimental group. Four subjects met these inclusion criteria. The mean age of the subjects with AOS was 69·25 years (range 60–77). All subjects in the AOS group exhibited non-fluent speech.

The CA group included subjects who were at least 1 year post-onset of left-hemisphere stroke and exhibited frequent sound substitutions in the presence of fluent speech. These subjects exhibited aphasia as measured by standardized aphasia batteries including the BDAE and the WAB or the MAE. Subjects with CA all demonstrated inordinate impairment of repetition with the relative prevalence of phonemic paraphasias. Subjects with aphasia who exhibited weakness, incoordination, or groping movements of the articulators were not included in this experimental group. Four subjects met the inclusion criteria. The mean age of the subjects with CA was 54·25 years (range 25–71). All of the subjects with CA exhibited fluent speech.

In addition to the two experimental groups, four non-brain-damaged subjects were recruited to serve as controls. The mean age of the control subjects was 67·25 years (range 57–75). Although the control group appears to better match the AOS group than the CA group, it should be noted that the mean age of the CA group was greatly decreased by a 25 year old subject. Also, the mean performance of the control subjects is similar to that of 40 non-brain-damaged subjects who completed a similar experiment (Clark and Robin 1998). Thus, the apparent age differences between the control group were not deemed critical for the purposes of this experiment. All subjects passed a vision screening, during which they were asked to identify a letter and a set of numbers displayed on the screen used during the experimental task (see below). One non-verbal subject with AOS responded to yes/no questions about the visual display to verify adequate vision.

Apparatus

Subjects were seated 24 inches (610 mm) from a computer monitor on which the movement target patterns were displayed. Subjects' heads were stabilized in all planes with a wall-mounted cephalostat to reduce whole-head movement artefact using well standardized methodology (Muller and Abbs 1979). Inferior–superior labiomandibular movements were transduced with a strain gauge. The amplified signal was digitized at 200 Hz by a Metrabyte Dash 16 analogue-to-digital converter and stored directly on the hard drive of a personal computer.

Table 1. Subject information

Subject information

Subject	Age	Sex	Time post-onset	Site of lesion	Spontaneous speech	Auditory comprehension	Repetition	Naming	Reading comprehension
AOS1	60	M	5 years	L MCA	Non-fluent	1	4	1	1
AOS2	77	M	15 months	L basal ganglia	Non-fluent	3	4	NA	3
AOS3	69	M	3 years	L hemisphere	Non-fluent	2	4	3	2
AOS4	71	M	21 years	L basal ganglia and insula	Non-fluent	2	4	NA	3
CA1	70	M	6 years	L inferior half of precentral gyrus and superior half of inferior frontal gyrus	Fluent	2	3	3	2
CA2	69	M	7 years	L basal ganglia and insula	Fluent	2	4	2	2
CA3	53	M	9 years	L inferior parietal lobe, insula	Fluent	3	4	3	2
CA4	25	M	2 years	L hemisphere	Fluent	2	3	2	2
C1	69	M	n/a	n/a	n/a	n/a	n/a	n/a	n/a
C2	75	M	n/a	n/a	n/a	n/a	n/a	n/a	n/a
C3	68	F	n/a	n/a	n/a	n/a	n/a	n/a	n/a
C4	57	M	n/a	n/a	n/a	n/a	n/a	n/a	n/a

1 = normal, 2 = mild impairment, 3 = moderate impairment, 4 = severe impairment, NA = not able to be assessed, n/a = not applicable. Lesion information was obtained from chart review or from the data reported by Seddoh et al. (1996) for subjects AOS4, CA1, CA2, and CA3, who participated in both experiments. Severity ratings were determined by a speech–language pathologist and a board certified neuropsychologist based on the test results, clinical interview, chart review, and perceptual judgements of speech.
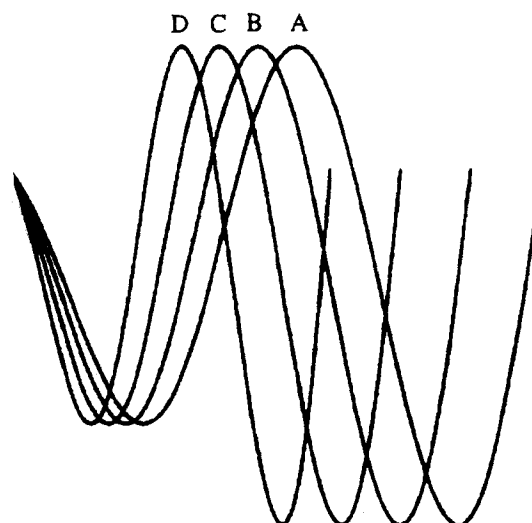
Figure 1. Target movement patterns.

Task

The experimental task was modelled after Wulf *et al.* (1993), with the modification that labiomandibular movements were targeted instead of upper limb movements. Subjects were required to produce labiomandibular opening–closing movements with specific spatiotemporal goal movement patterns. Four target movement patterns were utilized, each with the same relative timing and amplitudes, but with different absolute movement times (see figure 1). The total target movement times for targets A, B, C, and D were 1238, 1073, 908, and 743 ms respectively.

Before each trial, one of the target movement patterns was displayed for 4 s. The specific target wave was identified by a letter (A, B, C, or D) displayed in the upper left corner of the screen. When the target was removed, the subject began with the lower lip in a slightly opened position, then produced a sequence of opening–closing–opening–closing movements to produce the target pattern, attempting to match the target in both space and time. During the movement, the screen remained blank.

After a 2 s interval, during which the subject's movements were transduced, digitized, and recorded, knowledge of results (KR) was presented by super-imposing the subject's actual movement trace (displacement over time) with that of the target pattern. The target pattern was displayed in white (on a black background) and the subject's pattern was displayed in blue. The root-mean square deviation (RMS error) between the two patterns was calculated and displayed in the upper right hand corner of the screen. KR remained on the screen for 5 s.

Procedure

Subjects were provided with verbal and written instructions for the task. Following the instructions, the subjects performed 10 familiarization trials using version B. The subject's performance was discussed after each familiarization trial to ensure that the subject knew how to interpret the KR.

Practice trials of versions A, B, and D were presented in blocks of six trials, in which each version was performed six times before a switch to another version. The order of task versions was randomized, with the restriction that each version appeared once in each three-block sequence. The relative KR frequency was

systematically reduced across practice trials. Specifically, in the first three blocks, KR was presented on five of the six trials; during the next six blocks, KR was presented on four of the six trials; and the during the final six blocks, KR was presented on three of the six trials. This yielded an average KR frequency of 63%.

Each subject completed a total of 90 practice trials. Following a rest period and recalibration, the subjects completed the experimental task, which consisted of 10 retention trials of each practiced version A, B, and D, as well as 10 transfer trials on version C, with the order of the task versions randomized. No KR was provided during these trials. Version C differed from the other target patterns only in absolute movement time (908 ms), which was between the movement times of the practice targets. The entire experiment, including familiarization trials and the rest period, ranged from 90 to 110 minutes.

Data analysis

In the manner described by Wulf *et al.* (1993), temporal and amplitude scaling were conducted to determine the accuracy of GMP and amplitude and timing parameters. For each trial, the target wave was temporally rescaled (compressed or expanded) from 0·4 to 1·74 in increments of 0·02 with RMS error re-calculated at each increment. The scaling which resulted in the smallest RMS error was termed the 'temporal scaling factor' and was the measure of temporal parameterization accuracy. Within this scheme, movement patterns produced too slowly, resulting in longer movement durations, result in temporal scaling factors of less than 1·0, as the pattern must be compressed (i.e. multiplied by a scaling factor less than 1·0) to best match the target pattern. Conversely, movement patterns produced too quickly produce temporal scaling factors greater than 1·0, as these patterns must be expanded to best match the target. A scaling factor of 1·0 would indicate that the subject produced a movement of the exact total duration of the target pattern.

Next, the amplitude scaling factor was obtained. The variance in amplitudes of the position–time samples in the target pattern was divided by the variance in amplitudes of the temporally rescaled trace. The square root of this ratio was deemed the amplitude scaling factor and was the measure of amplitude parameterization accuracy. An amplitude scaling factor greater than 1·0 indicated that the subject produced the movement with amplitudes smaller than the target pattern, while an amplitude scaling factor less than 1·0 indicated that the subject produced the movement with amplitudes greater than the target pattern.

Once the temporal and amplitude scaling factors were computed, the factors were applied to the movement trace and RMS error was again calculated. This residual RMS error was taken as the measure of GMP accuracy. That is, after correcting for errors in *absolute* timing and amplitude (by applying the scaling factors), any difference remaining between the target and the actual movement trace results from differences in *relative* timing and amplitude, which is specified by the GMP. Thus, GMP accuracy was defined as the residual RMS error calculated after the temporal and amplitude scaling factors were applied.

In order to justify the use of the analysis protocol described above, it was necessary to verify that the movements produced during the task were programmed with a single GMP (Wulf *et al.* 1993). This was achieved by computing correlations among temporal landmarks selected from the velocity–time function of the movement traces (Young and Schmidt 1991). The assumption is that since

movements programmed with the same GMP differ only in absolute timing, but not relative timing, the correlation between the landmarks of a single structural unit (GMP) should be near 1·0. However, if a movement is governed by two or more GMPs, the correlations between landmarks which fall under the governance of separate GMPs should fall towards zero.

Average correlations between contiguous landmarks averaged from 0·51 to 0·97, except for the correlation between landmark 1 and landmark 2, for which the average correlation was 0·157. The observed pattern of correlations was consistent with the interpretation that the lip movement was generated by one GMP (with the exception of the first landmark, which is from movement onset to the first velocity peak). Data analysis proceeded based on several factors. First, Schmidt (personal communication) proposed that this pattern of correlations probably represents 'start-up' variability associated with initiating a movement. That is, the motor delays (Heuer 1988, Heuer *et al.* 1995) during the initiation of a movement may be more variable than those associated with the remainder of the movement. Although it might be expected that subjects with AOS would exhibit significant start-up variability, given the difficulty these subjects exhibit initiating utterances, the unit structure exhibited by the AOS subjects was not different from that of the other groups, or that of young normal subjects (Clark and Robin 1998).

Second, because the displacement trace corresponding to the start of the movement to the first landmark was a very small portion of the entire movement (less than 10% of the total movement time or approximately 50 ms), it was reasoned that even if this portion of the movement was governed by a different GMP, to include that portion in the analysis of the remainder of the movement would minimally affect the results of the analysis procedure.

Results

GMP accuracy

Figure 2 depicts the residual RMS error for the individual experimental subjects, as well as the mean performance of the control subjects. The error bars reflect intra-subject variability for the experimental subjects and inter-subject variability for the control subjects. Note that the ordinate represents error, so greater accuracy is reflected by lower RMS error values. Subjects AOS1 and AOS2 exhibited highly accurate GMPs, particularly in the retention and transfer conditions. In contrast, the remaining subjects with AOS exhibited lower GMP accuracy across conditions. Across subjects, the subjects with CA exhibited more consistent GMP accuracy, as well as accuracy levels similar to the controls, particularly in the retention and transfer conditions.

Temporal parameterization accuracy

Figure 3 illustrates temporal parameterization accuracy. Recall that a scaling factor of 1·0 reflects perfect temporal parameterization accuracy, while a scaling factor greater than 1·0 indicates that the subject performed the movement more quickly than the target, and a scaling factor less than 1·0 indicates that the subject performed the movement more slowly than the target pattern. Subjects AOS3 and AOS4 exhibited temporal parameterization accuracy equal to or greater than that
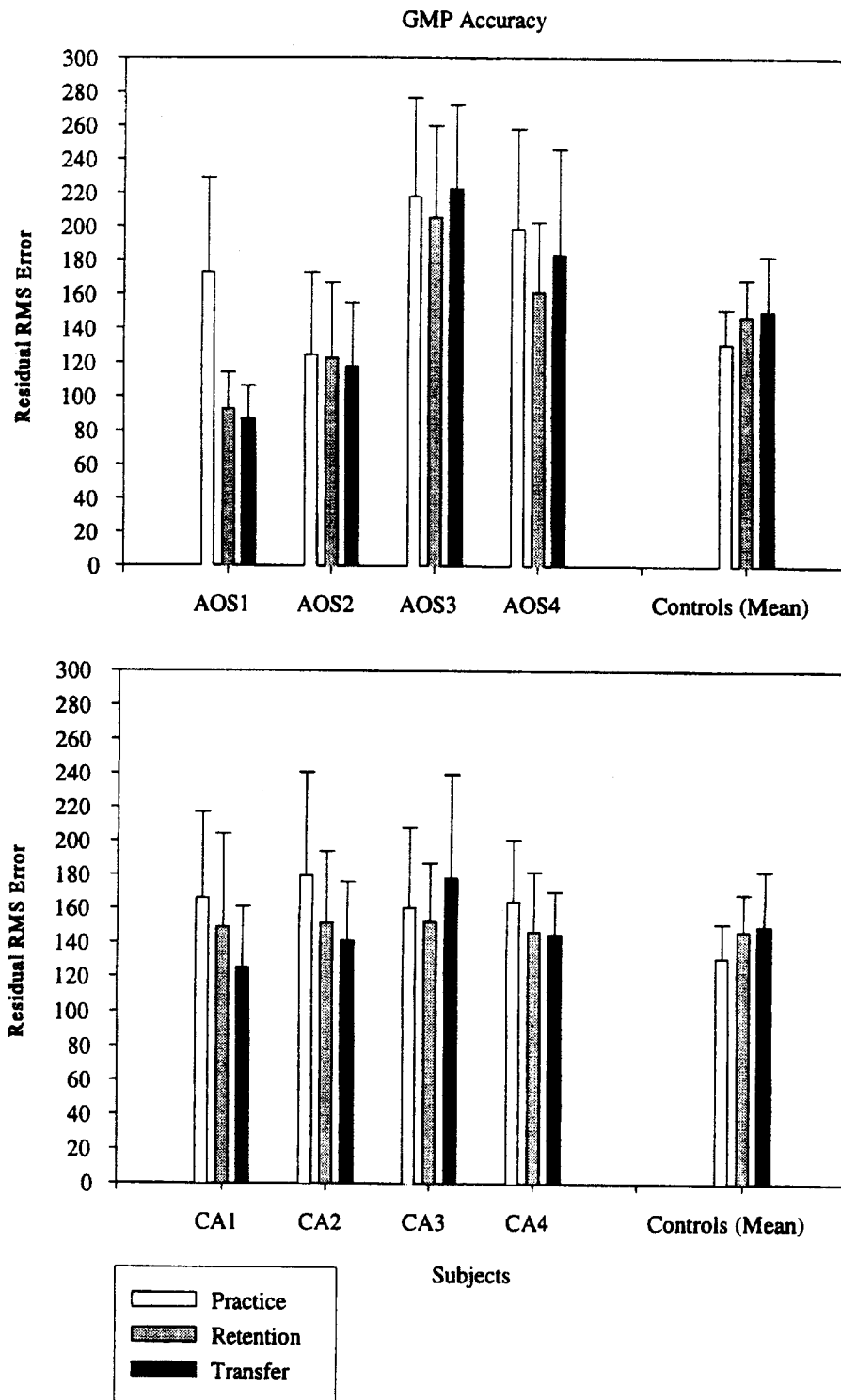
Figure 2. GMP accuracy for individual subjects. Mean and standard deviation for individual subjects. The mean and standard deviation of the control group are provided for comparison.

of the controls in most conditions. Subjects AOS1 and AOS2 exhibited high performance variability across conditions: subject AOS1 exhibited normal temporal parameterization accuracy only in the practice condition, while subject AOS2 was inaccurate only in the practice condition.

The subjects with CA all tended to err to the same degree although not

Temporal Parameterization Accuracy
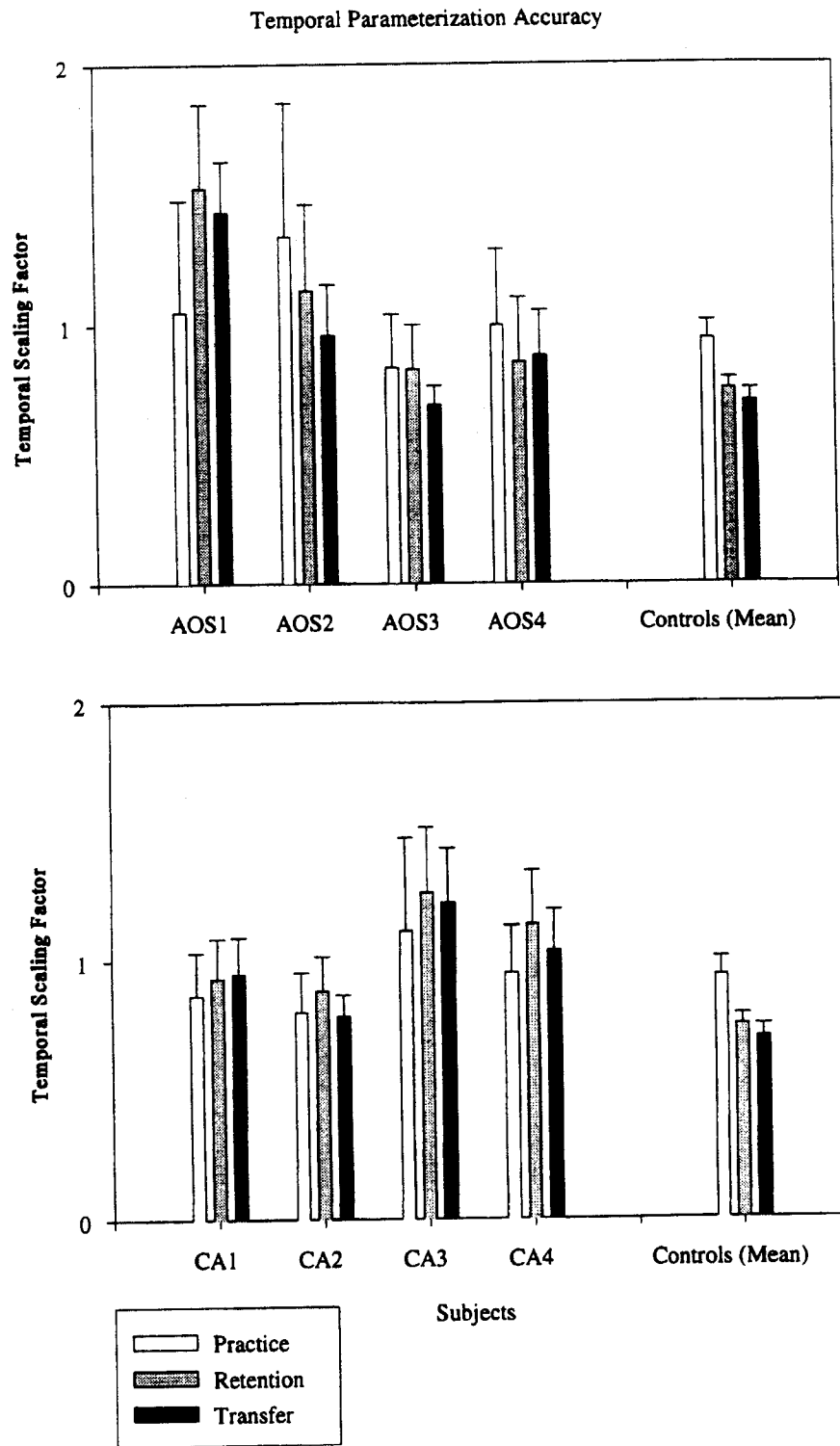
Figure 3.   Mean temporal parameterization accuracy for individual subjects. Mean and standard deviation of temporal scaling factor for individual subjects.

necessarily in the same direction. Subjects CA1 and CA2 tended to produce the movements too slowly, as evidenced by temporal scaling factors less than 1·0. This pattern was also demonstrated by the control subjects. In contrast, temporal scaling factors greater than 1·0 indicate that subjects CA3 and CA4 tended to produce the movements more quickly than the targets.
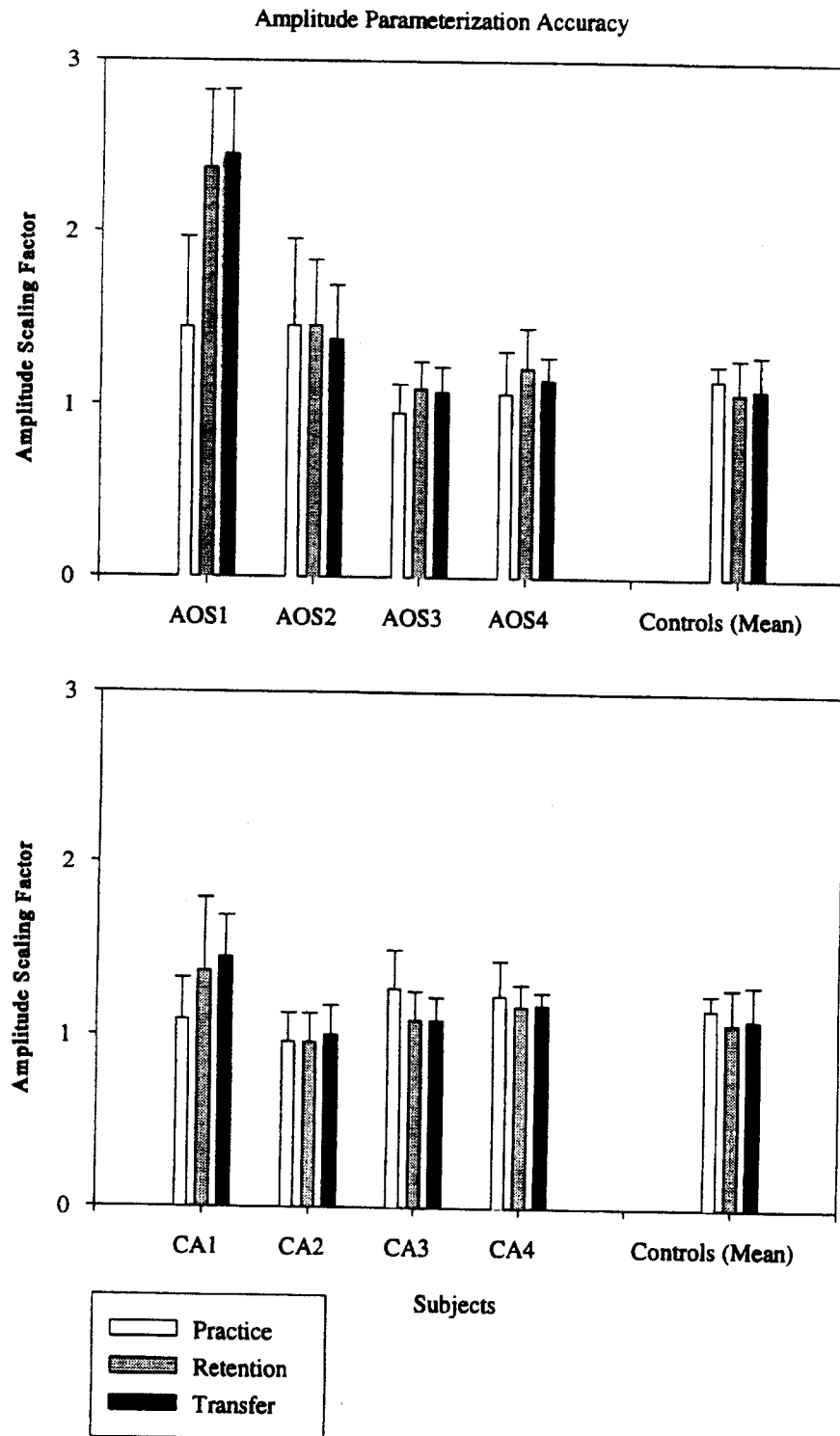
Amplitude Parameterization Accuracy

Figure 4.   Mean amplitude parameterization accuracy for individual subjects. Mean and standard
deviation of amplitude scaling factor for individual subjects.

Amplitude parameterization accuracy

Amplitude scaling factors greater than 1·0 indicate that the subject produced movements smaller in amplitude than the target pattern, while scaling factors greater than 1·0 indicate that the subject produced movements greater in amplitude

than the target pattern. Movements perfectly matching the target in amplitude resulted in a scaling factor of 1·0. It is clear from figure 4 that subject AOS1 exhibited very poor amplitude parameterization accuracy. This subject consistently produced movements much smaller in amplitude than the target pattern, particularly in the retention and transfer conditions. Subject AOS2 also produced movements smaller than the target, while the remaining subjects exhibited amplitude parameterization accuracy similar to that of the controls.

Subject CA1 also produced movements which were smaller in amplitude than the target patterns, particularly in the retention and transfer conditions. Each of the remaining subjects with CA exhibited amplitude parameterization accuracy in the range exhibited by the controls.

Discussion

The present experiment examined GMP and parameterization accuracy of subjects with AOS or CA during a non-speech oral motor task. Based on the prominent conceptualization of AOS as a motor programming disorder and previous evidence of disruption in relative timing of movements, it was predicted that all subjects with AOS would exhibit reduced GMP accuracy. Further, it was predicted that, in addition to GMP inaccuracy, subjects with AOS might also exhibit impaired parameterization. Subjects with CA were predicted to perform similarly to controls on all of the measures. *None* of these predictions were realized in the current study.

Performance of subjects with AOS

While three subjects with AOS exhibited reduced GMP accuracy in at least one condition, subjects AOS1 and AOS2 exhibited high GMP accuracy in at least two conditions. Interestingly, it was only these two subjects who exhibited reduced parameterization accuracy. Thus, even though some subjects with AOS exhibited reduced GMP accuracy (AOS prediction 1), no subjects simultaneously exhibited GMP and parameterization inaccuracy (AOS prediction 2).

Thus, an apparent dissociation was observed: subjects with AOS exhibited impairments in GMP or parameterization, but not both. Several potential explanations arise for this finding. First, it is possible that the observed patterns reflect performance trade-offs. That is, within the context of an impaired motor programming system, subjects may have only enough processing resources correctly to programme either the GMP or the parameters, but not both. Thus, the different performance patterns may simply reflect different resource allocation strategies in which subjects with AOS, reaching the limits of their capacity, were forced to choose some aspects of motor programming to which they would attend, but not all programming processes. Since neither GMP nor parameterization accuracy was emphasized in the present study, subjects may have been equally likely to prioritize either of these aspects of control. However, given that the GMP defines the shape of the movement, it may be inefficient for subjects to attend more to the parameter than the GMP. Thus, those subjects who had accurate parameterization but inaccurate GMPs may have been utilizing a maladaptive strategy in that changing the parameter is relatively trivial compared to modification of the programme that defines the shape of skilled actions. It is interesting to note that young non-brain-damaged subjects may also adopt this

strategy (Clark and Robin 1998), suggesting that while this strategy appears maladaptive, it may be preferred by many subjects, irrespective of the presence of apraxia. However, in the person with AOS, this strategy may be particularly disruptive since these subjects may not have the flexibility to maintain overall performance accuracy in the face of impaired motor programming.

Important support for viewing the data as reflective of a performance trade-off across conditions is provided by the performance of subject AOS1 (see figures 2–4). During the practice trials, AOS1 exhibited reduced GMP accuracy with relatively accurate amplitude parameterization (note that temporal parameterization accuracy was relatively stable across conditions). In contrast, during the retention and transfer conditions, GMP accuracy improved but at the cost of decreased amplitude parameterization accuracy. It is remarkable that within a single subject, performance trade-offs were observed. These trade-offs suggest that control strategies were utilized to cope with an impaired motor control system.

It may also be the case that performance differences within the AOS group represent subtypes of AOS. Subtypes of AOS have been proposed, based on site of lesion (Kertesz 1984, Square-Storer and Apeldoorn 1991) and behavioural characteristics (Hough and DeMarco 1996). Unfortunately, inadequate lesion data for the subjects studied here precludes differentiation based on specific site of lesion. Likewise, Hough and DeMarco developed their subtypes based on the ability of subjects to maintain phonological representations, skills that were not addressed in the present study.

It is clear from these data that the conceptualization of AOS as a deficit in the 'motor programme' or the GMP only is not accurate. High level motor programming is a complex process in which the GMP needs to be realized and then set according to specific parameters. AOS appears to involve the entire process of motor programming and not only one process (the GMP) within the programming of events. Such an impairment requires speakers with AOS to produce skilled movement patterns under great resource demand, resulting in an increased susceptibility to breakdown.

Performance of subjects with CA

The final prediction, that subjects with CA would perform normally on all the measures, was nullified by the performance of subject CA1. This subject exhibited reduced amplitude parameterization accuracy in the retention and transfer conditions. This finding suggests that while GMP or parameterization impairments do not appear to be characteristic of CA, such deficits may be present in some patients. Our findings suggest that the present paradigm may be sensitive to detection of these subtle motor control deficits. Thus, notions about motor control anomalies underlying the speech characteristics of CA appear inaccurate. Rather, one might view the motor control deficits reported in some patients with CA as concomitant to the linguistic disorder and not necessarily as a core part of the problem.

Conclusions

The current study is the first to define operationally and then examine motor programming ability in subjects with AOS, even though Darley (1969) defined the disorder over 25 years ago. Interestingly, not all subjects with AOS exhibited a

defective GMP, as defined operationally in this study. However, before dismissing Darley's assertions, it is necessary to explore how the current experiment's operational definition relates to Darley's original conceptualizations.

Darley's definition focused on the programming of 'positioning' of articulators and 'sequencing of muscle movements' (p. 639). This particular definition relates most closely to Schmidt's (1975, 1988) concept of GMP *plus* amplitude parameterization, both part of the high level programming process. Thus, the present view of AOS refines Darley's notion and does not focus on the GMP only but suggests that the GMP and/or parameterization may be impaired in AOS. It is also clear that amplitude parameterization as well as temporal parameterization, to which Darley does not refer, may also be impaired.

Several questions are raised by the present findings. One of the most prominent issues is whether and how impairments of GMP and/or parameterization differentially affect speech production. That is, how does the speech of subjects who exhibit primarily GMP errors differ from the speech of subjects with primarily parameterization errors? This is a particularly relevant question since researchers disagree about the degree to which speech and non-speech oral movements are related (e.g. Folkins *et al.* 1995, Weismer and Liss 1981). Additional information about the relationships among GMP and parameterization accuracy of speech and non-speech movements and speech accuracy would contribute greatly to our understanding of oral motor control.

Other questions which warrant addressing include: are GMP/parameterization impairments differentially associated with specific sites of lesion? Do subjects with different underlying impairments (GMP vs. parameterization) respond differently to speech treatment? Further, are different types of treatment more efficacious when applied to subjects who exhibit specific underlying impairments and, if so, what is the nature of that treatment? And finally, what are the implications for each of the above questions if differences in GMP and parameterization accuracy primarily reflect resource allocation strategies rather than actual differences in competence?

Replication of the current study with a larger number of subjects with a broader range of severity levels, while controlling for potential confounders (e.g. visual memory deficits) is needed. Precise lesion information as well as exacting speech performance measures will aid in the understanding of the contribution of specific impairments of GMP and/or parameterization to speech performance, as well as assist in the possible development of more comprehensive classifications of subgroups of AOS. Treatment efficacy studies which address the issue of underlying impairment would also contribute to our understanding of how motor control is affected in AOS. Finally, additional study of the GMP and parameterization accuracy in subjects with CA will lead to a better understanding of the underlying impairment of this disorder as well and allow for estimations of how frequently motor control anomalies co-exist with linguistic disorders in these patients.

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