APHASIOLOGY, 1997, VOL. 11, NO. 4/5, 433–445

The vowel lengthening exaggeration effect in speakers with apraxia of speech: compensation, artifact, or primary deficit?

MARGARET A. ROGERS

University of Washington, Seattle, WA, USA

Abstract

Vowel duration functions contrastively in English to signal the voicing feature of syllable-final stop consonants. This study examines three hypotheses posited to explain why speakers with apraxia of speech and a concomitant aphasia exhibit an exaggerated vowel lengthening effect relative to speakers with dysarthria, aphasia without apraxia and controls. The investigation addresses the hypotheses that the vowel lengthening exaggeration effect is attributable to: (1) a compensatory strategy, (2) an artifact of slow speaking rate, (3) the concomitant language impairment, or (4) a primary deficit reflecting the underlying nature of the apraxia disorder. The results do not support the first three of these hypotheses. It is hypothesized that the temporal measures most likely to reveal abnormalities which are uniquely characteristic of speakers with apraxia of speech are those which are relational in nature, either with respect to inter-articulator timing or contrastive durations.

Introduction

Apraxia of speech (AOS) is a difficult disorder to define objectively, to diagnose accurately with exclusive criteria, and to quantify satisfactorily in terms of clinical severity. However, the notion that the disorder entails disturbed motor programming for speech production resulting in impaired articulation and prosody has garnered support (e.g. Kent and Rosenbek 1983, McNeil and Kent 1990). There is also some consensus that AOS is distinct from dysarthria (although some similarities with ataxic dysarthria have been discussed, e.g. Kent and Rosenbek 1982) and even though it usually manifests with a concomitant aphasia, AOS is certainly not a necessary feature of aphasia. According to Lebrun *et al.* (1973), AOS 'stands between true aphasia and genuine dysarthria'. There are many reasons why empirically based definitions, diagnostic criteria, and indices of severity have not been firmly established, but central to the variety of potential explanations are two overriding problems. The first stems from the difficulty of studying AOS in a distilled manner, isolated from the confounding effects that a disrupted language system unavoidably introduces, perhaps regardless of the nature of the dependent variable being examined. The second problem concerns the difficulty of disambiguating the primary deficits of the disorder (i.e. those behaviours directly stemming from the impairment itself), from compensatory behaviours. Appropriate methods designed to partial out the contribution of the apraxic component from the language impairment and compensatory modifications are

Address for correspondence: Margaret Rogers, PhD, Department of Speech and Hearing Sciences, University of Washington, 1417 N.E. 42nd Street, Seattle, WA 98105-6246, USA.

needed in order to increase our understanding of the nature of AOS. Resolution of this problem is viewed as a precursory step in the endeavour to develop more exacting definitions, diagnostic criteria, and severity indices for AOS. Additionally, it is anticipated that more precise specification of AOS along these lines may enable improved utilization of persons with AOS to further research concerning speech motor control. This paper describes a method that has the potential to help circumvent both of the aforementioned problems, and also contributes to our understanding of how speakers with AOS implement phonologically driven durational contrasts.

Vowel durations produced in polysyllabic words and phrases by speakers with AOS have generally been characterized as being longer than those produced by normal speakers (Baum *et al.* 1990, Collins *et al.* 1983, Kent and Rosenbeck 1983, Ryals 1981, Strand 1987). Kent and Rosenbek (1983) reported that speakers with AOS exhibit slower than normal speaking rates with prolonged transitions, segment durations, steady states, interword pauses, and the insertion of inter-syllabic pauses. Johns and Darley (1970) reported that speakers with AOS acknowledged that they were attempting to slow down. Darley *et al.* (1975) posit that the decreased speaking rate occurs 'in compensation for the continuing articulation difficulty' (p. 262). Other authors have suggested that temporal slowing is not compensatory, but rather reflects the nature of the disorder and should be considered a primary deficit of AOS. Baum *et al.* (1990) hypothesized that the longer vowel durations produced by speakers with AOS might be attributed to a general slowness of articulation. However, DiSimoni and Darley (1977) found that /p/ transition durations in one speaker with AOS were faster than normal in the intrasyllabic position. Robin *et al.* (1989) studied the velocity of the lower lip in five speakers with AOS and found that these speakers were all able to generate high peak articulatory velocities, regardless of the perceptual adequacy of the tokens. McNeil *et al.* (1986) reported that even though speakers with AOS produced longer vowel durations, peak velocities of their lip and jaw movements were similar to those produced by normal-speaking subjects. This evidence was interpreted to suggest that the longer vowel durations produced by speakers with AOS are not due to a generalized slowing of articulatory movements. Even though the articulatory movements of individuals with AOS were found to be within normal limits, McNeil *et al.* posited that the decreased speaking rate may be a direct consequence of the primary deficit in that they attributed the slowness to a disturbance of spatiotemporal motor programming, not to compensation.

Rogers *et al.* (1996) investigated whether the vowel prolongation noted among persons with AOS could be attributed to the disordered speaker's attempt to monitor vowel accuracy (i.e. as a compensatory strategy). Vowel durations obtained under conditions of white noise masking were compared to those obtained under normal listening conditions in order to ascertain whether the speakers with AOS would continue to prolong the steady states even when on-line monitoring of target vowel accuracy was no longer possible via the auditory modality. Since vowel durations increased under the masking condition for both the normal and AOS groups, the authors concluded that the vowel prolongation phenomenon is unlikely to be a consequence of compensatory attempts to monitor auditory information.

An unexpected finding was reported by Rogers *et al.* (1994) concerning the degree of vowel lengthening produced by speakers with AOS to signal the voicing

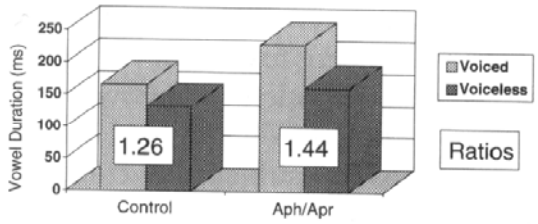

Figure 1. Ratios of vowel durations from voiced and voiceless post-vocalic contexts for normal speakers and speakers with apraxia of speech and a concomitant aphasia. (Rogers *et al.* 1994.)

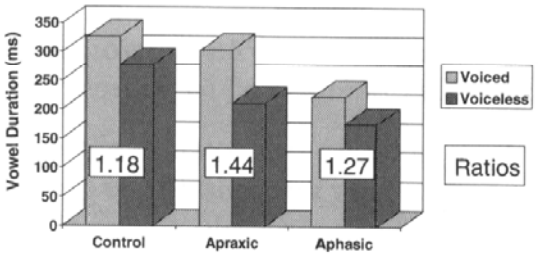

Figure 2. Ratios of vowel durations from voiced and voiceless post-vocalic contexts from one normal speaker, one speaker with apraxia of speech, and one speaker with aphasia. (Data derived from Caligiuri and Till 1983).

contrast of syllable-final stop consonants. In their study, participants produced CVC minimal pairs which differed in terms of the voicing feature of the post-vocalic consonant (e.g. [bvd] and [bVt]). In English, the voicing feature of the post-vocalic consonant determines the relative length of a vowel as noted by the following phonological rule:

$$V \rightarrow V: / \_\_ C_{[+\text{voice}]}$$

Thus, a vowel preceding a voiced consonant is relatively longer than when it precedes its voiceless counterpart with the same manner and place of articulation (House and Fairbanks 1953, Umeda 1975). Investigators have found that differences in vowel duration signalling the voicing contrast of syllable-final stop consonants are generally preserved in speakers with AOS and/or aphasia (Freeman *et al.* 1978, Caligiuri and Till 1983, Duffy and Gawle 1984, Baum *et al.* 1990). This finding has been interpreted to support the notion that AOS is not a phonological or linguistic deficit since compliance with this rule should indicate intact abilities at the level of phonological encoding. The unexpected finding reported by Rogers *et al.* (1994) was that speakers with AOS and a concomitant aphasia exaggerate the vowel lengthening rule relative to the durational differences obtained from the normal controls (see Figure 1). While this finding has not been explicitly reported elsewhere, the data reported by Caligiuri and Till (1983) were examined in light of this question, and also show an exaggeration effect for their patient with AOS (see Figure 2).[1]

The present study addresses the issue of whether the exaggeration of the vowel lengthening effect preceding voiced final stops in speakers with AOS is: (1) attributable to compensation, (2) a simple artifact of slow speaking rate, (3) attributable to the concomitant language impairment, or (4) whether it reflects the nature of the underlying impairment stemming from the apraxic or the aphasic component of this typically mixed disorder. In order to test these possible

[1] It is also interesting to note that Ryalls (1986) reported that the 'motor aphasic' speakers in this study exaggerated the durational difference between tense and lax vowels.

explanations, vowel duration measurements were obtained using three minimal pairs of words in which only the voicing feature of the final consonant differed. The dependent variable employed was the ratio of the vowel durations (RVD) obtained from the voiced (V+) and voiceless (V−) final consonant word pairs (i.e. RVD = V+/V−). Four groups of subjects participated in this experiment: subjects with dysarthria (DA), subjects with aphasia but without AOS (APH), subjects with AOS and a concomitant aphasia (AOS), and control subjects with normal speech (CON). The following experimental questions were addressed.

Is there a significant difference among the four groups relative to ratio of the vowel durations (RVD) obtained from voiced and voiceless post-vocalic contexts? If there is, then the analysis will be directed to address the following:

1. Do speakers with AOS exaggerate the vowel lengthening effect?
2. Do relative differences in speaking rate among the groups predict the respective RVDs of each group? (In other words, do speakers with dysarthria and slow speaking rates also exhibit exaggerated RVDs? Are exaggerated RVDs simply an artifact of slow speaking rates?)
3. Do individuals with reduced intelligibility exaggerate the vowel lengthening effect? (In other words, is exaggerated vowel lengthening attributable to an individual's attempt to compensate for decreased speech intelligibility?)
4. Do subjects with aphasia but without AOS also exhibit exaggerated vowel lengthening? (In other words, is there a significant RVD difference between the two groups of subjects with aphasia? Is exaggerated vowel lengthening attributable to the apraxic component of the mixed motor speech and language disorder, or do the aphasic subjects without apraxia also exaggerate RVDs thereby, increasing the likelihood that it is the language impairment which accounts for the exaggerated vowel lengthening effect observed in the subjects with both aphasia and AOS?)

Method

Subjects

Seventeen subjects participated: three with aphasia but no evidence of AOS (APH); four with AOS and aphasia (AOS); four with dysarthria (DA); and six subjects with normal speech and no history of speech, language or neurological problems (CON). Descriptive information concerning the ages, dialect, diagnoses, and time post-onset of the subjects are listed in Table 1. Table 2 displays selected test results obtained from the subjects with dysarthria, aphasia, and AOS. Subjects were classified as 'aphasic' based on the presence of language impairments in the areas of auditory comprehension and verbal expression. All the individuals comprising the purely 'aphasic' and the 'AOS with aphasia' groups exhibited a non-fluent aphasia with respect to phrase length and low-frequency usage of function words. The purely 'aphasic' subjects did not exhibit signs of AOS, such as laboured difficulty in articulation or an inconsistent pattern of phonemic errors across multiple attempts to articulate the same target. The presence of a concomitant dysarthria was ruled out for both groups of subjects with aphasia based on structural and functional oral mechanism examinations. Subjects were classified as 'apraxic' based on convergent diagnoses of AOS by three ASHA-certified speech–language pathologists and the presence of laboured difficulty in

Table 1. Subject characteristics

Subject	Group	Age (years)	Regional dialect	Diagnosis/aetiology	Time post-onset
1	Apraxic	71	Northwest	Primary progressive aphasia	4 years, 6 months
2	Apraxic	47	Northwest	CVA, occlusion of left MCA	12 months
3	Apraxic	39	Northwest	Multiple, CVAs, brainstem & MCA	5 years, 2 months
4	Aphasic	59	West coast	Primary progressive aphasia	1 year
5	Aphasic	68	Northeast	CVA, left hemisphere	2 years
6	Aphasic	54	Northwest	Multiple CVAs	3 years, 2 months
7	Aphasic	53	Northeast	CVA, left parietal/occipital lobe	9 months
8	Dysarthric	28	Northwest	TBI	6 years, 9 months
9	Dysarthric	40	Northwest	ALS	2 years, 6 months
10	Dysarthric	55	Northwest	ALS	12 years
11	Dysarthric	72	Northwest	Parkinson's	12 years
12	Control	29	Midwest	n/a	n/a
13	Control	29	Northwest	n/a	n/a
14	Control	29	West coast	n/a	n/a
15	Control	70	Northwest	n/a	n/a
16	Control	48	Northwest	n/a	n/a
17	Control	40	Northwest	n/a	n/a

CVA = Cerebral Vascular Accident; MCA = Middle Cerebral Artery; TBI, Traumatic Brain Injury; ALS = Amyotrophic Lateral Sclerosis.

Table 2. Descriptive data for experimental subjects

Subject	Group (Gender)	ALSSS	Auditory Comprehension	Verbal Expression	Reading Comprehension
1	Apraxic (male)		SVTT AS = 21·5 WAB Seq. Commands 80/80 WAB Spelled Wd. Recog 2/6 WAB Sp-wr. Wd Recog 4/4	MTDDA Sentence Prod. 1/6 MTDDA Definitions 8/10 WAB Reading Commands 10/10 BNT TS = 51	RCBA IV Functional Reading 100% RCBA VI Sentence Pic. 100% RCBA VII Paragraph Pic. 40%/80% RCBA VIII Paragraph–Factual 100%
2	Apraxic (male)		SVTT AS = 20·5 ACTS 20/21	BNT TS = 40 MTDDA Sentence Prod. 3/6	RCBA IV Functional Reading 100% RCBA VI Sentence Pic. 100% RCBA VII Paragraph Pic. 80% RCBA VIII Paragraph–Factual 100%
3	Apraxic (male)		SVTT AS = 10 MTDDA Understanding Sents. 13/16 ACTS 18/21	BDAE Rep. Phrases 3/8(hp), 2/8(lp) MTDDA Naming Pictures 18/20	RCBA I World–Visual 90% RCBA IV Functional Reading 60% RCBA V Synonyms 70% RCBA VI Sentence Pic. 90%
4	Aphasic (male)		SVTT AS = 30 ACTS 20/21	Sentence Repetion 23/30 Verbal Fluency 24/1 min	RCBA VIII Paragraph–Factual 100% RCBA IX Paragraph-Inferential 100% RCBA X Morphosyntax 90%
5	Aphasic (female)		SVTT AS = 24 ACTS 16/21 BDAE Complex Ideational 9/12	BNT no ceiling obtained 34/36 BDAE Cookie Theft 2·9 CU/min	RCBA VIII Paragraph–Factual 100% RCBA IX Paragraph-Inferential 100% RCBA X Morphosyntax 80%
6	Aphasic (female)		SVTT AS = 33 MTDDA Quicksand passg. 8/16 IUs	BNT TS = 58 BDAE Cookie Theft 10·2 CU/min	RCBA I Word–Visual 100% RCBA IV Functional Reading 90% RCBA VIII Paragraph–Factual 100%
7	Aphasic (male)		SVTT AS = 18 MTDDA Quicksand passg. 6/16 IUs BDAE Complex Ideational 8/12	BNT TS = 60 COWAT AS = 18 BDAE Cookie Theft 12·3 CU/min	Nelson–Denny AS = 42
8	Dysarthric (male)		Normal	Not formally assessed No word finding problems. Normal syntax.	Not assessed.
9	Dysarthric (male)	SP: 5 SW: 7 UE: 6 LE: 8	Normal	Not formally assessed. No word finding problems. Normal syntax.	Not assessed.
10	Dysarthric (male)	SP: 6 SW: 8 UE: 9 LE: 8	Normal	Not formally assessed. No word finding problems. Normal syntax.	Not assessed.
11	Dysarthric (male)		Normal	Not formally assessed. No word finding problems. Normal syntax.	Not assessed.

ALSS = Amyotrophic Lateral Sclerosis Severity Scale; ACTS = Auditory Comprehension Test for Sentences; BDAE = Boston Diagnostic Aphasia Examination; BNT = Boston Naming Test; COWAT = Controlled Oral Word Association Test; MTDDA = Minnesota Test of Differential Diagnosis of Aphasia; RCBA = Reading Comprehension Battery for Aphasia; SVTT = Short Version Token Test; SP = Speech; SW = Swallowing; UE = Upper Extremity; LE = Lower Extremity.

articulation, and an error inconsistency across multiple attempts to articulate the same target. Subject selection for the APH group was also guided by the intention to have the severity of the language impairment match closely with that of the AOS group. The test of auditory comprehension employed, the Shortened Version of the Token Test (DeRenzi and Faglioni 1978), reveals that this goal was not entirely achieved. The AOS group's scores range from 10 to 21·5, while the APH group's scores range from 18 to 33. In general, subjects from both groups are classified as mildly to moderately impaired, as none of the subjects fell into a severely impaired category.

The subjects with dysarthria were selected based on the following criteria: (1) having at least a 1-year history of dysarthria (to ensure adequate time to develop compensatory behaviours); (2) exhibiting a slow rate of speech; and (3) intelligibility compromised but no more than moderately impaired. Slow speaking rate was determined by subjective judgement and later confirmed by acoustic analyses of steady-state durations. Moderately impaired intelligibility was determined by either the Speech Severity Index of the Amyotrophic Lateral Sclerosis Scale or by subjective ratings of intelligibility during spontaneous speaking contexts performed by two speech–language pathologists. The normal controls were selected based on their ages and regional dialect. All subjects were right-handed and had at least 12 years of education.

Stimuli and procedure

A set of six target words (bet, bed, bat, bad, butt, and bud) were produced in the carrier phrase 'Say ___ for me' until a total of five accurate productions were obtained for each word. The carrier phrase and target words were presented in written form on index cards and ordered so that each consecutive target contained a different vowel from the previous production. Subjects were instructed to speak as naturally as possible and were asked to repeat the target if it sounded unnatural or was perceived by the experimenter as erred. Only those productions which were judged to be phonemically accurate and sounded natural were used in the analyses.

Audio-recordings were obtained using a high-quality headset microphone (AKG C410) positioned 15 cm from the right corner of the subject's mouth in a quiet room. The speech samples were recorded by a Sony DAT (TDC-D10 Pro II) and later digitized at 22 kHz sampling rate and digitally filtered at 11k using the IBM M-Audio Capture and Playback Adapter into CSpeech (Milenkovic 1994).

Temporal measures of vowel duration were obtained using both the time waveform and wideband spectrograms to determine the onset and offset of periodicity. The initial and final striation which spanned across both the second and third formants of the vowel segment in the spectrogram served to demarcate the boundaries. Fifteen per cent of the tokens from each subject were remeasured in order to assess the reliability of the measurement procedure.

Results

Sample exclusion

It was deemed necessary to exclude certain productions from the analysis either because they were perceptually incorrect or due to the perception that they did not sound natural. Specifically, two of the control subjects, (subjects 14 and 16) tended

Table 3. Absolute vowel durations (ms) from voiced and voiceless post-vocalic contexts

Final consonant	Control	Aphasic	Dysarthric	Apraxic
Voiced context	172	178	291	227
SD	51	55	92	56
Voiceless context	131	137	221	158
SD	33	34	65	45

to over-articulate during the initial segment of the experiment. They were instructed to 'speak more naturally' and were both able to resume a speaking style that was judged to sound more natural. Only those productions that were recorded after they had ceased over-articulating were used in the analysis. All three of the disordered groups produced tokens that were judged to be incorrect. However, the percentage of erred productions relative to the number of total correct productions analysed was relatively low for each group (AOS: 8/90 = 8%; APH: 6/120 = 5%; DYS: 13/120 = 11%). Only natural-sounding and perceptually correct productions were used in the analysis, in order to maintain as much consistency as possible in the quality of the productions across the four groups of subjects.

Absolute vowel durations

Reliability of the measurement procedure was tested by comparing two experimenters' vowel duration measurements made on 15% of each subject's productions. Inter-observer reliability was assessed using Pearson's correlation coefficients. Overall, the reliability was found to be high across all groups ($r >$ 0·974). The largest differences between observers was 6·6 ms and no single subject or group mean difference yielded a Pearson's correlation coefficient under $r >$ 0·962.

The results of the absolute vowel duration comparisons by groups showed that the DA group produced significantly longer vowels than all the other groups. The AOS group was significantly slower that the APH and CON groups, but there was no significant difference between the APH and CON groups. All comparisons were conducted using ANOVA ($p < 0{\cdot}05$) and Bonferroni *post-hoc* procedures. The absolute vowel durations by groups are displayed in Table 3.

Ratios of vowel duration

The mean ratios of vowel duration (RVD) are displayed in Table 4 by group across the three sets of minimal pairs. The ratios were calculated by dividing the vowel durations from the final voiceless context into their voiced counterpart. The pairs were established based on the order in which the tokens were produced, such that the first /e/ word was paired with the first production of its minimal pair counterpart. Ratio means were then calculated by collapsing across all productions for each paired comparison within subjects. Lastly, the group ratio means were calculated by collapsing across subjects within groups. The ratios of vowel duration for each minimal pair are displayed in Figure 3 by group.

To determine whether the RVDs differed among the groups or by minimal pairs,

Table 4. Ratios of vowel duration by minimal pair set and group

Stimulus pair	Control	Aphasic	Dysarthric	Apraxic
Bad/bat				
Mean	1·340	1·262	1·356	1·433
SEM	0·038	0·056	0·022	0·081
Bed/bet				
Mean	1·248	1·271	1·264	1·462
SEM	0·035	0·035	0·038	0·115
Bud/butt				
Mean	1·369	1·351	1·321	1·451
SEM	0·072	0·089	0·038	0·089
Collapsed across tokens				
Mean	1·31	1·30	1·32	1·44
SD	0·25	0·15	0·25	0·28

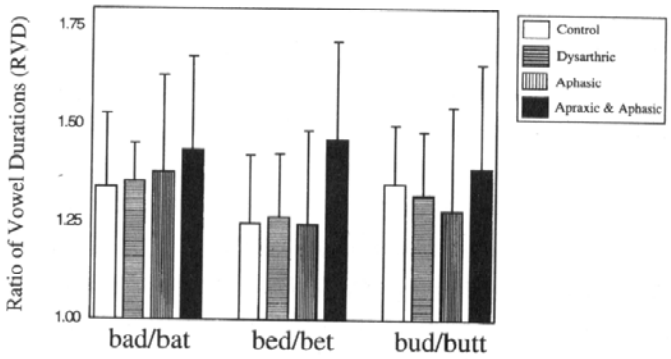

Figure 3. Ratios of vowel durations from voiced and voiceless post-vocalic contexts for each of three minimal pairs by group.

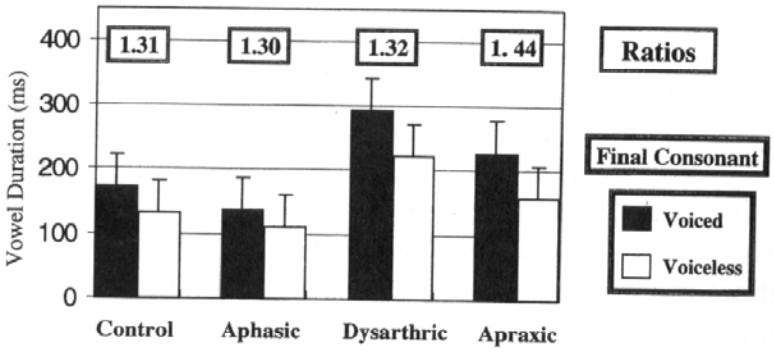

Figure 4. Mean absolute vowel durations from the voiced and voiceless post-vocalic contexts by group. The mean ratios of vowel durations for each group are also displayed in the boxes.

a two-factor analysis of variance (one between, one within) was performed. There was no significant main effect for the minimal pairs ($F(2,156) = 1{\cdot}26$; $p = 0{\cdot}29$). The main effect for group was significant ($F(3,156) = 27{\cdot}31$; $p < 0{\cdot}001$), with no significant interaction ($F(6,156) = 1{\cdot}48$; $p = 0{\cdot}19$). The minimal pairs were then collapsed and the results were subjected to Bonferroni *post-hoc* procedures and included six pairwise comparisons. The analysis revealed that the AOS group exhibited significantly greater RVD values than all the other groups and there were no other significant differences. The mean overall RVDs are listed by group in Table 4. Figure 4 shows this same data graphically.

Conclusions

The finding that only the AOS group exhibited greater than normal RVD values is interpreted to support the hypothesis that the vowel lengthening exaggeration effect stems from the underlying motoric impairment in AOS. This interpretation is arrived at by a process of elimination in which three alternative hypotheses concerning the genesis of exaggerated vowel lengthening in speakers with AOS were tested: (1) that the exaggeration is secondary to compensation; (2) it is simply an artifact of slow speaking rate; and (3) that it stems from the concomitant language impairment.

Since vowel length is a contrastive cue, which signals voicing in syllable-final stops, it is conceivable that exaggerated durational differences could provide a compensatory modification. The finding that the RVD values of the DA group were not significantly different from the CON group suggests that the DA subjects did not exaggerate contrastive vowel durations to compensate for diminished intelligibility. However, the hypothesis that the greater RVD values of the AOS group are attributable to compensation still remains a logical possibility in that the nature of compensatory behaviours may vary across disorders based on the source of the speech disruption. Shinn *et al.* (1985) investigated the saliency of syllable-duration effects for the perception of the (b–w) contrast and concluded that the role of context-dependent cues, such as vowel duration, in speech perception may be overestimated. This suggestion brings into question the extent to which listeners use vowel duration cues to distinguish between cognates. Thus, while it is not possible, based on the current data, to reject the hypothesis that speakers with AOS compensatorily exaggerate the vowel lengthening contrast, the hypothesis would be severely weakened to the extent that listeners are found not to use these cues. Further research is required to determine the perceptual saliency of the vowel lengthening effect, especially within ranges such as those produced by the speakers with AOS in this study.

A similar argument is made relative to the artifact of slow speaking rate hypothesis. Since the RVD values from the slowest group (DA) were within normal limits, the exaggeration effect is unlikely to be a simple artifact of slow speaking rate.

The finding that only the AOS group, and not the APH group, exhibited significantly greater RVD values than the CON group suggests that the exaggeration effect more likely stems from the apraxia deficit itself than from the concomitant impairments in the language domain. It is argued that the results of this study support the hypothesis that while the phonological rule is clearly preserved in these subjects with AOS and aphasia, implementation of the rule is affected by the motoric impairment, and not the impaired language component of this mixed disorder.

Discussion

In general, the study of AOS has been constrained by the availability of patients exhibiting the pure form of this disorder (i.e. one that is uncontaminated by the concomitant presence of a language impairment). The present investigation attempted to not only discover the underlying cause of the exaggerated vowel lengthening observed in the productions of the three patients with AOS and

aphasia, but also to apply a research design which has the potential to allow AOS to be studied even in the context of a concomitant aphasia. This study entailed a comparison of four groups of subjects in order to disambiguate the separate contributions of slow speaking rate, reduced intelligibility (and the subsequent modifications to compensate), and the concomitant language impairment from the effects of the motoric impairment. The primary group of interest consisted of three individuals whose most salient problem was judged to be AOS, yet all three of these subjects also exhibited a concomitant aphasia. The comparison of the subjects with dysarthria allowed the contribution of slow speaking rate and compensatory modifications for decreased intelligibility to be examined. Since the dysarthric group exhibited vowel duration ratios similar to the normal control group, it was concluded that neither slow speaking rate nor attempts to compensate for decreased intelligibility appear to be sufficient conditions to cause contrastive vowel lengthening to become exaggerated. The comparison of the subjects with aphasia (but without AOS) allowed the contribution of the language impairment to be tested. In the final analysis it is argued that neither dysarthria nor aphasia appears to create sufficient conditions to cause a speaker to exaggerate vowel lengthening in the voiced post-vocalic context. This finding suggests that temporal measures designed to examine *relative timing* (such as the duration ratios obtained from minimally contrastive phonemic contexts), appear to be sensitive to the motoric impairment in speakers with AOS, but not to the deficits arising from dysarthria or aphasia.

Other temporal parameters of speech have been reported to be abnormally produced by speakers with AOS. Some of these studies have examined the *absolute* durations of various temporal parameters produced by subjects with AOS and typically compared these to age- and gender-matched controls. For example, absolute durations have been reported for consonant and vowel durations (e.g. Baum *et al.* 1990, Collins *et al.* 1983, Kent and Rosenbek 1983, Seddoh *et al.* 1996). In general these studies have demonstrated that the durations produced by speakers with AOS tend to be longer and more variable in comparison to the productions of the normal speakers. However, measures of *absolute* durations have not successfully differentiated the speakers with AOS from other disordered populations. For example, Seddoh *et al.* (1996) reported that the group of speakers with conduction aphasia and the group of speakers with AOS both tended to produce longer than normal vowel durations, but that they did not differ significantly from one another, except that the token-to-token variability was much greater for the speakers with AOS. On the other hand, measures that reflect temporal coordination among articulators, and are therefore considered to be measures of *relative timing*, did exhibit sensitivity to the differences between conduction aphasia and AOS (e.g. stop gap durations). In the present study the variable that differentiated the individuals with AOS from the other impaired speakers was a different kind of relative timing measure. The comparison of vowel lengths in minimally contrastive phonemic contexts does not reflect coordination among articulators, but does reflect the implementation of relative timing that is prescribed by the phonemic context. It is hypothesized that the temporal measures most likely to reveal abnormalities which are uniquely characteristic of speakers with AOS are those which are *relational* in nature, either with respect to inter-articulator timing or contrastive durations. Accordingly, measurements of relative timing, whether based on inter-articulator coordination or contrastive durations, may have the

potential to be a differentially sensitive measures of AOS, and therefore might be useful for improving the diagnostic accuracy as well as the sensitivity and reliability of the severity measures employed to assess and study patients with AOS.

The preliminary nature of this study, and the small number of subjects within each group, warrant a rather tentative explication of the conclusions. Whether most individuals with apraxia of speech exaggerate contrastive durations, such as the vowel lengthening rule, is an empirical question that needs to be followed up. The question of why speakers with AOS may exaggerate contrastive durations is entirely a matter of speculation at this point in time. However, even though the conclusions that can be drawn from the present investigation are tentative, the method employed is one that clearly has merit and potential. Given the rarity of cases presenting with a singular disorder of pure apraxia of speech, a method which may allow the apraxic component to be studied, even when accompanied by other concomitant impairments, should be viewed as a creative solution worth pursuing. The method presented arguably permits examination of how other concomitant impairments and accompanying compensatory modifications are contributing to the outcome of interest. The results of this investigation suggest that exaggeration of the vowel lengthening rule exhibited by the three individuals with AOS and a concomitant aphasia is not likely to be the consequence of slow speaking rate, compensatory modifications, or a consequence of the coexisting language impairment. Ruling these alternatives out is a preliminary, but important, step in understanding the effects that a disruption of motor programming has on speech production.

References

BAUM, S., BLUMSTEIN, S., NAESER, M. and PALUMBO, C. (1990) Temporal dimensions of consonant and vowel production: an acoustic and CT scan analysis of aphasic speech. *Brain and Language*, **39**, 33–56.

CALIGIURI, M. P. and TILL, J. A. (1983) Acoustical analysis of vowel duration in apraxia of speech: a case study. *Folia Phoniatrica*, **35**, 226–234.

COLLINS, M., ROSENBEK, J. and WERTZ, R. (1983) Spectrographic analysis of vowel and word duration in apraxia of speech. *Journal of Speech and Hearing Research*, **26**, 224–230.

DARLEY, F., ARONSON, A. and BROWN, J. (1975) *Motor Speech Disorders* (W. B. Saunders, Philadelphia, PA).

DERENZI, E. and FAGLIONI, P. (1978) Normative data and screening power of a shortened version of the Token Test. *Cortex*, **14**, 41–49.

DISIMONI, F. G. and DARLEY, F. L. (1977) Effects of phoneme duration control of three utterance length conditions in an apractic patient. *Journal of Speech and Hearing Disorders*, **42**, 257–264.

DUFFY, J. and GAWLE, C. (1984) Apraxic speakers' vowel duration in consonant-vowel-consonant syllables. In J. Rosenbek, M. McNeil and A. Aronson (Eds) *Apraxia of Speech: Physiology, acoustics, linguistics, management* (College-Hill Press, San Diego, CA), pp. 167–196.

FREEMAN, F. J., SANDS, E. S. and HARRIS, K. S. (1978) Temporal coordination of phonation and articulation in a case of verbal apraxia: a voice onset time study. *Brain and Language*, **6**, 106–111.

HOUSE, A. S. and FAIRBANKS, G. (1953) The influence of consonant environment upon secondary acoustical characteristics of vowels. *Journal of the Acoustical Society of America*, **25**, 105–113.

JOHNS, D. and DARLEY, F. (1970) Phonemic variability in apraxia of speech. *Journal of Speech and Hearing Research*, **13**, 556–583.

KENT, R. D. and ROSENBEK, J. C. (1982) Prosodic disturbance and neurologic lesion. *Brain and Language*, **15**, 259–291.

KENT, R. and ROSENBEK, J. (1983) Acoustic patterns of apraxia of speech. *Journal of Speech and Hearing Research*, **26**, 231–249.

LEBRUN, Y., BUYSSENS, E. and HENNEAUX, J. (1973) Phonetic aspects of anarthria. *Cortex*, **9**, 126–135.

McNeil, M. R. and Kent, R. D. (1990) Motoric characteristics of adult aphasic and apraxic speakers. In G. R. Hammond (Ed.) *Advances in Psychology: Cerebral control of speech and limb movements* (Elsevier/North-Holland, New York), pp. 349–386.

McNeil, M. R., Caliguiri, M., Weismer, G. and Rosenbek, J. (1986) Labio-mandibular kinematic durations, velocities and dysmetrias in apraxic adults. *Americal Speech and Hearing Association*, **28**, 64.

Milenkovic, P. (1994) Cspeech (Version 4.X) [computer software] (Department of Electrical and Computer Engineering, University of Wisconsin–Madison, Madison, WI).

Robin, D. A., Bean, C. and Folkins, J. W. (1989) Lip movement in apraxia of speech. *Journal of Speech and Hearing Research*, **32**, 512–523.

Rogers, M. A., Eyraud, R. and Strand, E. A. (1994) The effects of noise masking on vowel duration in apraxia of speech. Presentation at the Clinical Aphasiology Conference, Traverse City, MI.

Rogers, M. A., Eyraud, R., Strand, E. A. and Strokel, H. (1996) The effects of noise masking on vowel duration in three patients with apraxia of speech and a concomitant aphasia. *Clinical Aphasiology*, **24**, 83–96.

Ryalls, J. (1981) Motor aphasia: acoustic correlates of phonetic disintegration in vowels. *Neuropsychologia*, **19**, 365–374.

Ryalls, J. (1986) An acoustic study of vowel production in aphasia. *Brain and Language*, **29**, 48–67.

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

Shinn, P. C., Blumstein, S. E. and Jongman, A. (1985) Limitations of context conditioned effects in the perception of (b) and (w). *Perception and Psychophysics*, **38**, 397–407.

Strand, E. (1987) Acoustic and response time measures in utterance production: A comparison of apraxic and normal speakers. Unpublished doctoral dissertation. University of Wisconsin–Madison.

Umeda, N. (1975) Vowel duration in American English. *Journal of the Acoustical Society of America*, **58**, 434–445.