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Apraxia of Speech : A Phonologic or Motoric Disorder ?

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Abstract

Apraxia of speech has been considered as a phonologic disorder by some researchers and as a motoric disorder by others, and the debate continues. The current study attempted to further explore this issue using acoustic analysis of speech segments. Acoustic durations of high and low vowels were obtained from two apraxics and two normal speakers at two speaking rates and in two postvocalic consonantal voicing contexts. The results of the study showed that the apraxics had abnormally long vowel durations; failed to maintain a significant duration difference between high and low vowels; did not vary speaking rate and had poor motor precision. However, they did manifest longer vowel durations when followed by a voiced postvocalic consonant. These findings were used to infer that apraxia of speech is primarily a motoric disorder with an intact phonological system.

Introduction

Speech segments exhibit a great deal of variation in their timing, A major theoretical motivation forthe study of speech timing is to understand how much of this variation is random and how much of it is systematically controlled. Is any or all of the variation in their liming a mere random phenomenon? Or are there specific rules that systematically govern the observed variation in timing of speech segments? If there arc systematic patterns of influence are these influences controlled by the phonology of the language and thus linguistic in nature? Or are these influences a mere consequence of the physiology of the structures required to produce the segments? What is the significance of this variation in the timing of segments? Questions such as these have generated numerous studies of speech timing in an attempt to explore these issues and explicate the speech timing system (Crystal and House, 1988: Gay, 1978,1981; Gopal, 1987, 1990; House, 1961; Klatt, 1976;Lehistc, 1972; Lindblom, 1963,1967; Nootcboom, 1972; Peterson & Lehiste, 1960; Port, 1981 and others),

A number of factors influence the duration of speech segments. Some of these

factors are — vowel tensity, vowel height, manner and voicing of the following sound, number of syllables in a word, the position of the word in a phrase, the presence or absence of stress on the syllable, speaking rate, semantic novelty, etc. Most of these factors may be classified as intrasyllabic and extrasyllabic factors (see Crystal and House 1988; Klatt, 1976;Gopal, 1987,1990 for a detailed review).

Based on the numerous studies of speech timing in normals, the factors that influence segmental duration have been interpreted as being primarily phonologicallymotivatedorphysiologically based. Forexample, Klatt (1976) found that the pattern of durations of individual phonetic segments and pauses convey information about syntactic boundaries, lexical stress and emphasis, and semantic novelty. He claimed that these patterns were governed by phonological rules as they served the primarily purpose of conveying linguistic information. Thus these factors, in addition to vowel tensity, the influence of postvocalic consonantal voicing, etc., were interpreted as phonological factors.

In contrast, House (1961) claimed that some of the factors that influence segmental duration were physiologically based. He found that low vowels were longer in duration than high vowels in American English and attributed this increased vowel duration to the physiology of the mandible. The mandible being a heavy structure requires more time to be moved for a low (or open) vowel. Thus the factor vowel height, was interpreted as a physiologic one. The current view is that certain aspects of segmental duration (or certain factors) are phonologically based and certain other ones are physiologically based. Using studies of speech timing, it may be possible to elucidate the nature of the various factors that influence scgmental timing. Such information provides us with a better understanding of the human speech production system.

Speech being a highly co-ordinated act requires precise programming and sequencing of the various muscles involved in its production. This fine level of control is provided by the central nervous system, Regardless of thephonologicorphysiologic basis of a factor that results in a particular duration pattern in speech, the timing of various speech gestures requires mediation by the central nervous system. This neuromuscular programing of timing and sequencing is referred to as the motor control of speech. Studies of speech timing provide valuable information regarding the complexity of motor control of speech.

One such application of this line of inquiry is the study of speech timing in clinical populations, particularly in the neurologically impaired populations such as aphasia, apraxia, dysathria, etc. If one can sustain the premise that certain aspects of speech timing are phonologically based, then those neurological impairments that affect the phonologic / linguistic compo-

nents of language ought to manifest deviations only in the timing of segments that are phonologically determined. Similarly, those neurological impairments that do not affect the phonologic components of a language ought not to show any aberrancy in the timing of phonological factors of speech. Thus studies of speech timing may help in understanding the nature of a neurological impairment and provide information that may be useful in differential diagnostic of sorts. Among the various neurologically impaired populations that have been subjected to studies of speech timing, apraxia of speech is one such.

The most commonly cited defini lion of apraxia of speech is the one provided by Darley in 1969, wherein he states that apraxia of speech is

"...an articulatory disorder resultingfrom impairment as a result of brain damage, of the capacity to program the positioning of speech musculature and the sequencing of muscle movements for the production of phonemes. The speech musculature does not show significant weakness, slowness, or incoordination when used for reflex and automatic acts" (cited in Collins, Rosenbek, and Wertz, 1983; p. 224).

These deficits in programming and sequencing are usually manifested as abnormalities in the timing of speech in apraxics. For example, many studies have shown that apraxics have abnormally long vowel duration and abnormally large standard deviations (Caligiuri & Till, 1983; Collins, Rosenbek & Wertz, 1983; Kent & Rosenbek, 1983; Skencs, 1987; Weismer& Fenncll, 1985; Ziegler & Von Cramon, 1986). While there have been a number of studies on apraxia of speech, there has been an on-going debate as to whether apraxia of speech is a motor disorderor a phonological disorder.

On the one hand are several studies that claim that apraxia of speech is a motoric disorder and not a phonologic one (Caligiuri & Till, 1983; Collins, Rosenbek & Wertz, 1983; Duffy & Gawle, 1984; Kent & Rosenbek, 1983; Skenes, 1987; Weismer& Fennell, 1985; Ziegler & Von Cramon, 1986). For example, Collins et al. (1983) reported that inspite of very long vowel durations, apraxics manifested a reduction vowel duration of the stem (or base) word when the number of syllables in the word increased. This seems to be an almost universal phonological rule in normals across differentlanguages(Lindblom, 1963, 1967; Nooteboom, 1972; Lehiste, 1970, 1972). Similarly, Duffy & Gawle (1984) reported that apraxics had highly variable vowel durations. They attributed this deviation from normal speakers as being a result of poor temporal control but accurate phonological selection. Ziegler and Von Cramon (1986) conducted a more direct study of the phase relations of individuals speech movements and found that patients with apraxia of speech have a basic problem in phasing individual speech gestures appropriately.

On the other hand are the findings of Klich, Ireland and Weidner(1979), Martin

and Rigrodsky (1974), and Kent and Rosenbek (1983). Klich et al. (1979) conducted a phonologic analysis of the errors in apraxia of speech, such as distinctive feature and markedness analysis, and found that the substitution errors in apraxia of speech arc very similar to those seen in phonologically disordered children. These findings led them to conclude that apraxia of speech is a phonological and not a motoric disorder. Kent and Rosenbek (1983) found a variety of segmcntal and prosodic abnormalities in the apraxic speakers and concluded that while some of the liming deficits were probably motoric in nature, others were due to phonological rule deficits. Thus the controversy remains unresolved.

The question also arises as to how pervasive is this speech motor deficit in apraxics. If apraxia of speech is indeed a motor disorder, docs the impairment spare all of the phonologic factors that influence segmental timing and affect only those that are physiologically based? Or does the impairment selectively affect certain physiological factors and not others? Answers to questions such as these will help *us* further understand the nature of the disorder and the workings of the speech production system. Only future studies of various aspects of speech timing in apraxia may help provide answers to such questions.

Based on the findings of several researchers described above, it is evident that vowel duration is function of both, physiological and phonological factors. It is also a commonly held premise that the duration difference between high and low vowels is primarily due to physiological factors. If apraxics indeed have a physiological timing deficit, then it is expected that they will not exhibit the characteristic time difference between high and low vowels. The purpose of the current study was to explore the pattern of high and low vowel durations in apraxics and interpret the findings within this framework.

For the purposes of the present study, three null hypothesis were tested:

1) there will be no di (Terence in the duration of high vowels between apraxics and normal speakers.

2) there will be no di (Terence in the duration of low vowels between apraxics and normal speakers.

3) there will be no difference in duration between high and low vowels in apraxia of speech.

Methodology

Subject :

Four male General American English (GAE) speakers were used as subjects. Two of these speakers had apraxia of speech and varying amounts of coexisting aphasia. Both apraxic subjects had a single thromboembolic cerebrovascular lesion (CVA) involving the left hemisphere. Diagnosis of apraxia of speech was made on the basis of performance on the Western Aphasia Battery (WAB) and the Motor Speech Evaluation tests by three professional

speech-language pathologists in a hospital in Northern California. The apraxic subjects were required to have a similar score of 75 on the repetition subtest of the WAB to be included in the study. The control group consisted of two normal speakers, matched for age, years of education, handedness (as best as possible) and with no history of neurological disease and no clinical evidence or history of speech-language deficits.

Speech Stimuli:

The stimuli elicited consisted of sentences containing words that included the target high or low vowels. There were two pairs of high and low vowels. These consisted of the front high-low vowel pari / i / and / ae / and the back highlow vowel pair / u / and / a /. These vowels were placed in a monosyllabic test word. The initial consonant of the test word was either a / p / or a / b / and the final consonant was either a / l/or a/d/. These test words were embedded in the sentence "Put the_____away" or "Take the_____away". Each speaker produced each of these sentence twice at each of two speaking rates, fast and slow. Therefore, each subject produced a total of 128 sentences (4 vowels x 4 consonants x 2 phrases x 2 repetitions x 2 rates). Fourlists were made with each list containing a randomized order of the 128 sentences. A total of 512 sentences were recorded from the 4 speakers.

Procedure :

The procedure consisted of one of the researchers reading each stimulus sentence and simultaneously presenting the same sentence written on an index card to the subject. The subject was then asked to repeat the sentence. Each subject was provided with simple oral instruction regarding the procedure, in addition to a short demonstration and practice of the procedure. Sentences were recorded for the slow speaking rate first and then for the fast speaking rate. All subjects were requested to speak slowly in the slow rate, and they were told that they would later be asked to talk as fast as possible. A maximum of three attempts were allowed before moving on to the next sentence. Subjects were provided rest periods whenever they opted, and often times the researcher instigated the break between repetitions. All the sentences and all repetitions were recorded on the same day for each subject.

Recording :

The subjects were asked to produce the sentences at a comfortable voice (intensity) level. Spoken sentences were tape recorded on a calibrated Marantz 420 cassette recorder for later computeranalysis. A condenser microphone (Realistic Model 33 - 984 B) was placed at a distance of 6 - 8 inches from the subject's lips and the subject produced the sentences directly into the microphone. The apraxics were recorded in their homes in a quiet room. The normal subjects were recorded in a double-walled IAC sound booth in the Speech Sciences Lab of the University of California at Santa Barbara.

Acoustic Analysis and Measurement:
The recorded sentences were first low pass filtered (9.6 KHz) and then digitized using a 12-bit Analog-to-Digital board with a sampling frequency of 20 KHz (DT2281 -F by Data Translations, Inc.) on to an IBM-AT computer. The digitised sentences were analyzed for vowel duration measurements using the Interactive Laboratory System (ILS from Signal Technology, Inc.) speech analysis programs. Three different temporal measurements were made in each sentence recorded (vowel duration, syllable duration, and sentence duration). The duration of the vowel was computed by measuring the interval of the periodic portion of the waveform in the corresponding region of the test word by selective listening of portion of the signal (see Gopal, 1987,1990 for details).

Statistical Analysis:

A Multifactorial ANOVA was used to analyze the vowel duration data. The dependent variable was vowel duration (in milliseconds) and the four grouping variables or factors used were: vowel type (high or low vowels), subject type (apraxia or normal), Speaking Rate (fast or slow), and postvocalic consonant type (voiced or voiceless). The design of the study was intended to help analyze the mean vowel duration differences under the influence of four factors; namely, type of speaker (normal or apraxic), type of vowel (high or low), rate of speech (fast or slow), and postvocalic consonant (/ t / or / d /). The Statistical Analysis Software (SAS) on a PC was used to analyze the data. Significant differences were analyzed using the Tukey post-hoc multiple comparison procedure (Zar, 1984).

Results

The results of the ANOVA showed significant effects of subject type on vowel duration ($F = 428.17$, $df=3$, $p <= 0.0001$), vowel type on vowel duration ($F=18.29$, $df=l$, $p <= 0.0001$), speaking rate on vowel duration ($F=130.48$, $df=l$, $p <= 0.0001$), and poslvocalic consonant on vowel duration ($F=55.13$, $df=l$, $p <= 0.0001$). Interaction effects were significant only between subject type and vowel type ($F=l . 79$, $df=3$, $p <= 0.0001$), and between subject type and speaking rate ($F=9.49$, $df=3$, $p <= 0.0001$), and between subject type, vowel type, and speaking rate on vowel duration ($F=5.35$, $df=3$, $p <= 0.0012$). Since there were significant interaction effects, a post-hoc Tukey's multiple comparison test was undertaken to make comparisons between specific means within the interaction effects (Zar, 1984).

The mean vowel durations and the standard deviations for the high and low vowels for each of the subjects in each of the rate, postvocalic consonantal conditions is listed separately in Table I. Figure 1 is a plot of the mean vowel durations, shown on the ordinate, as a function of the various rate and consonantal conditions, shown on the abscissa. The four different curves are for the four different subjects.

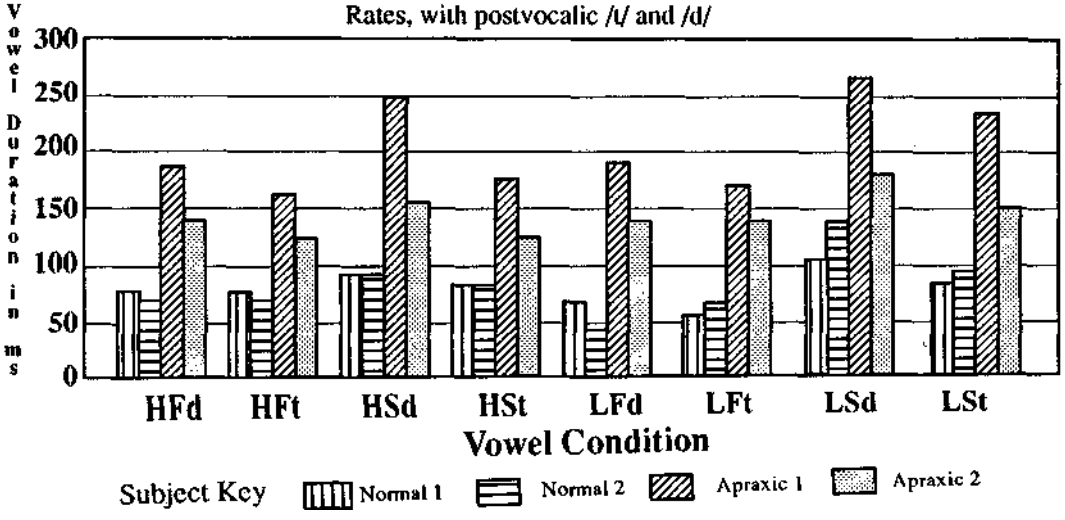
The first hypothesis tested was the null hypothesis that there will be no difference in the high vowel durations between apraxics and normal speakers. The results of the Tukey test showed that there was a significant difference in the mean vowel durations of high vowels (averaged across rate and postvocalic consonant) between apraxics and normals ($q=31.8113$, $p< 0.05$). The mean high vowel duration forthe apraxic speakers was 123.6 ms, and for the normal subjects it was 46.27 ms. The first null hypothesis was therefore rejected since a signi ficant difference in duration was found for the high vowels between apraxics and normals. As can be seen in figure 1 and Table I, this difference is found in each of the speaking rate and postvocalic consonantal conditions.

Table I : Mean durations in milliseconds (X), Standard Deviations (SD), for Apraxic (A) and normal (N) subjects, for High (H) and Low (L) vowels, at Slow (S) and Fast (F) speacking rates, with Poslvocalic Consonants /d/ and /t/.

		Normals			Apraxics	
		X	SD		X	SD
HFd	Nl	68.04	24.28	Al	183.31	46.81
	N2	60.66	18.89	A2	143.80	44.14
HFl	Nl	68.23	20.11	Al	159.40	50.47
	N2	57.44	14.45	Al	123.60	39.47
HSd	Nl	90.64	48.94	Al	249.18	50.32
	N2	90.00	43.02	A2	155.95	44.35
HSt	Nl	74.84	46.72	Al	178.16	24.98
	N2	74.64	25.57	A2	128.85	41.50
LFd	Nl	61.31	27.68	Al	190.72	52.61
	N2	46.27	29.76	A2	144.72	42.53
LFt	Nl	52.79	30.96	Al	170.23	48.40
	N2	56.56	31.34	A2	143.36	32.40
LSD	Nl	106.45	68.31	Al	266.96	71.00
	N2	139.79	49.07	A2	176.58	44.35
LSt	Nl	86.18	56.22	Al	238.44	105.80
	N2	96.48	39.50	Al	150.71	31.07

The second hypothesis tested was that there will be no difference in the duration of low vowels between apraxics and normal speakers. The results of the Tukey test showed that there was a significant difference in the duration of low vowels between

Fig. 1. Mean Vowel durations for High (H) Low (L) vowels in Fast (F) an Slow (S) Rates, with postvocalic /t/ and /d/



apraxics and normals (q=24.0593, $p < 0.05$), and thus this hypothesis was also rejected. The mean low vowel duration for the apraxic group was 266.96 ms and for the normal subjects it was 139.72 ms. Again, as can be seen in figure 1 and Table I, this difference is found in each of the speaking rale and postvocalic consonantal conditions.

Since the significant difference that was found for both high and low vowels between apraxics and normals was pooled across the two rates and two postvocalic consonants, it was necessary to perform the post-hoc multiple comparison for each of the vowel groups separately at each of the two rates and each of the postvocalic consonants. The results revealed that there was a significant difference in duration in all cases between apraxics and normals forlow and high vowels.

The results of these two hypotheses taken together indicated that the vowel duration for both high and low vowels were consistently longer in the subjects with apraxia of speech when compared with normals.

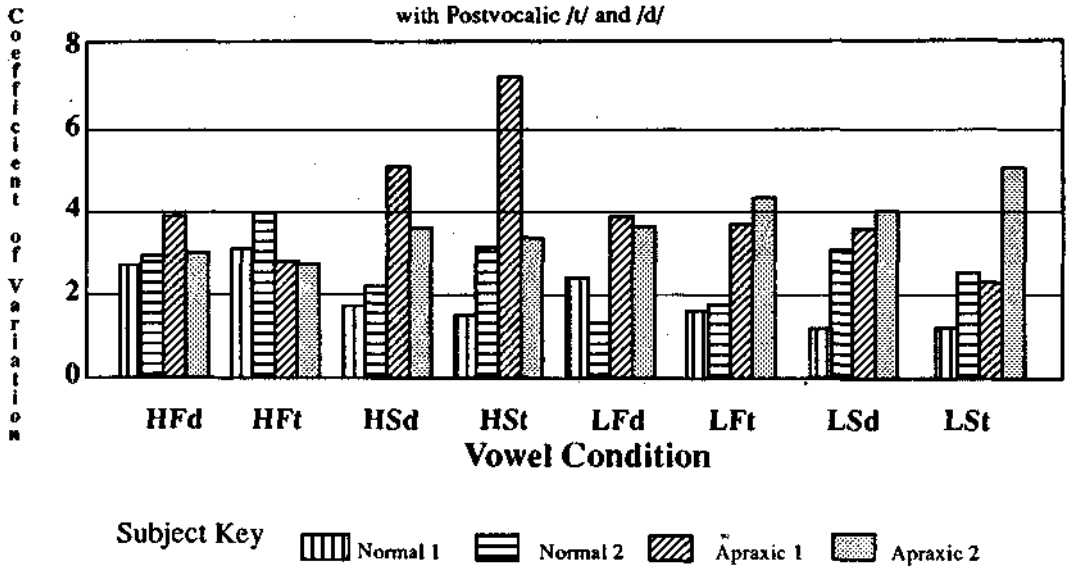
The third null hypothesis tested was that there will be no significant difference in duration between high and low vowels in apraxia of speech. The Tukey test revealed that there was no significant difference between apraxic high and low vowels (pooled across both consonants) in the fast rale (q = 1.3714, $p < 0.05$) but that there was a significant difference between apraxic high and low vowels in the slow rate (q = 7.1887, $p < 0.05$). Further multiple comparisons between mean durations for high and low vowels in the slow rate for each of the postvocalic consonants revealed thatapraxic high and low vowels differed significantly only for the postvocalic consonant A/ (q = 5.9886, $p < 0.05$). this data is provided in Table I for each of the rate and consonantal conditions separately. Therefore, the null hypothesis was rejected for the slow rate not for the fast rate.

Table II : coefficient for Variation (C V) (Standard Deviation divided by mean) and average CVs for each condition (xCV) for Apraxic (A) and Normal (N) subjects, for High (II) and , Low (L) vowels, at Slow (S) and I-asl (1') speaking rates, with Posivocalic consonants /d/and A/.

		Normals			Apraxica	
		CV	xCV		CV	xCV
HFd	N1	2.80	3.00	Al	3.91	3.58
	N2	3.21		A 2	3.26	
HFt	Nl	3.39	3.68	Al	3.16.	3.14
	N2	3.97		Al	3.13	
HSd	Nl	1.85	1.97	Al	4.95	4.23
	N2	2.09		A2	3.52	
HSt	Nl	1.60	2.26	Al	7.13	5.11
	N2	2.92		A 2	3.10	
LF1	Nl	2.21	1.88	Al	3.62	3.51
	N2	1.55		A 2	3.40	
lLR	Nl	1.70	1.75	Al	3.52	3.97
	N2	1.80		A2	4.2	
I.Sd	Nl	1.56	2.20	Al	3.76	3.87
	N2	2.85		A 2	3.98	
I.St	Nl	1.53	1.98	Al	2.25	3.55
	N2	2.44		A 2	4.85	

Additionally, it was found that across both rates and postvocalic consonants, the standard deviations of the apraxic speakers were larger and overlapped considerably with the normal speakers. This data is also provided in Table I. Table II

Fig. 2. Co-efficient of varation (CV) for High (H) and Low (L) Vowels at Slow (S) and Fast (F) rates with Postvocalic /t/ and /d/



lists the Coefficient of Variation (CV) for all conditions separately for the apraxics and normals. Figure 2 is a plot of the CV (shown on the ordinate) as a function of the various consonantal and rate conditions (shown on the abscissa). The four curves are for the subjects. These CVs were determined by dividing the standard deviation by the respective mean values. As can be gathered from figure 2 and Table II, the CVs for the apraxics tended to be higher than those of the normals in majority of instances.

Discussion

The results of the current study showed that the apraxic subjects had vowel durations which were abnormally long and highly variable compared to the normals. These findings are consistent with the other studies of apraxia of speech (Caligiuri & Till, 1986 ; Collins et al., 1983 ; Kent & Rosenbek, 1983 ; Ziegler & von Cramon, 1986). Although each of these researchers were examining different phenomena, they all reported abnormally long speech segment duration in apraxics. However, these results are inconsistent with those reported by Duffy and Gawle (1984) wherein their apraxic subjects manifested shorter than normal vowel durations. They did report that the apraxic vowel durations were highly variable which is consistent with the findings of the current study.

The acoustic analysis of high and low vowel durations undertaken in this study

was an attempt to contribute to the resolution of the phonologic vs. motoric nature of apraxia of speech. The durational difference between high and low vowels (low vowels being longer than high) seen in normals was originally thought to be motorically motivated (House, 1961). The results of this study indicated that the high-low duration difference was not maintained in apraxia of speech, except in the case of high and low vowels preceding the voiceless consonant /t/ in the slow speaking rate. At first glance, this finding may be taken to support the notion that apraxia of speech is indeed a motoric disorder as there is a disruption of the influence of the physiologic factor of vowel height. However, the findings of McNeilage (1970)using pipe speech may provide an interesting alternative explanation.

McNeilage (1970) found that normal subjects with a pipe in their mouth could still produce an intelligible distinction between high and low vowels without moving their jaws. He argued that while the durational difference between high and low vowels may have originally been motoricall y motivated, it had now been incorporated into the phonology of the language (also see Port, 1981). Thus the factorof vowel height need not be strictly construed as a physiologic one. therefore, the finding that apraxics do not maintain a distinction between high and low vowels may, in and of itself, not provide us with very much information in order to resolve this debate. But this finding coupled with the fact that the apraxic subjects did consistently produce longer vowels before the voiced consonant /d/ than before the voiceless consonant /t/, provides some evidence that their phonological system was unimpaired. The influence of postvocalic consonantal voic-' ing on vowel duration has been interpreted as being phonologically motivated. The finding of longer vowels before voiced stop consonants that before voiceless ones is consistent with reports in normals (Crystal & House, 1988 ; House, 1961) as well as in apraxia of speech (Caligiuri & Till, 1983 ; Duffy &Gawle, 1983). thus, the combination of these two findings in the present study may be taken as evidence to refute the claim that apraxia of speech is a phonologic disorder (Klich, Ireland & Wcidner, 1979 ; Martin & Rigrodsky, 1974 ; Kent and Rosenbck, 1983).

In some of the previous studies that had suggested that apraxia was a phonological disorder, the methods used to study apraxia of speech as well as the terminology used to describe it were largely linguistic (Klich et al., 1979 ; Martin & Rigrodsky, 1974). As pointed out by Kent and Rosenbek (1983), some linguistic variable s are very closely associated with motoric variables and so it is entirely possible for the disruption of a motoric variable to bring about a change in a corresponding (or closely associated) linguistic variable. Thus one needs to exercise caution in making strict dichotomous distinctions of apraxia of speech be-

ing a phonologic or motoric disorder. It is important to consider the possibility that apraxia of speech cannot be treated as a purely motoric or a purely phonologic disorder, but rather a combination of both. Thus it could be potentially difficult to separate the disruption of motor deficits from phonologic deficits in apraxia of speech when using such isolated variables.

The finding of a larger coefficient of variation in apraxics when compared with normals in the current study provides further evidence that there is a motoric disruption in apraxia of speech. The CV, which is obtained by dividing the standard deviation scores by the mean values was suggested by Duffy & Gawle (1984) as a measure of motor precision, they viewed the mean as the "target" while considering the standard deviation as the "attempt to produce the target". Duffy & Gawle (1984) found that their apraxic subjects had greater CVs than normals and concluded that this was an indication of poor motor precision. The results of this study revealed that by using CV as an index of motor precision, the apraxic subjects in the study did have poor motor precision because they consistently had larger CVs than normals. This indication of poor motor precision was supported by the observable groping behavior demonstrated by the two apraxic subjects in the study.

The results of this study also showed that in the apraxics, speaking rate did not cause a significant effect on the duration of high and low vowels. That is, apraxics had difficulty in varying speaking rate when compared with the normals. The literature on the effects of speaking rate on duration of high and low vowels shows that there is a decrease in the duration of vowels as speaking rate increases (Crystal and House, 1988 ; Gay, 1981 ; Gopal, 19900. these findings are consistent with those of Skenes (1987) who reported that her apraxic subjects were unable to make changes in speaking rate even upon instruction.

Utilizing the cluster of findings from this study and those from literature, it may be argued that there is a fair amount of evidence to suggest that the phonological system is preserved in apraxia of speech, .whereas there is a disruption of the motor control mechanism.

Summary and Conclusions

The findings of the current study showed that the apraxic subjects had abnormally long vowel durations; did not manifest a duration difference between the high and low vowels in seven out of eight conditions ; and had difficulty manipulating speaking rale. These findings clearly suggest that the apraxics do exhibit motor control deficits in their speech timing. This was further supported by the fact that their motor control precision was not as good as the normal subjects as indicated by larger than normal Coefficient of Variation. The finding that they maintained longer vowel durations before voiced consonants than before

voiceless consonants is suggestive of the fact that their phonological system was not impaired. The results of this study, then, provide some evidence that apraxia of speech is a disorder in which the phonological system was minimally, if at all impaired, but the motoric system was largely impaired.

The findings of this study arc limited in their generalizability because of a very small subject sample. These subjects may not be representative of the apraxic population at large. However, given the agreement of the current findings with the other growing number of studies, it seems likely that this sample was fairly representative of the apraxic population. The results of this study and other studies of apraxia of speech may have eventual implications in the treatment of apraxia of speech. If indeed apraxia of speech is a motoric and not a phonologic disorder, then the choice of treatment may be more motoric in nature. The utility of such an implication awaits future evaluation.

References

Caligiuri, M. P. & 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. C, & Wertz, R. T. (1983). Spectrographic analysis of vowel and word duration in apraxia o f speech. *Journal of Speech and Hearing Research, 26,* 224-230.

Crystal, T. H. & House, A. S. (1982). Segmental durations in connected speech signals : Preliminary results. *Journal of the Acoustical Society ofAmerica, 72* (3), 705-716.

Crystal, T. H. & House, A. S. (1988). The duration of American English vowels: An overview. *Journal of Phonetics, 16,* 263-284.

Duffy, J. R. & Gawle, C. A. (1984). Apraxic speakers' vowel duration in consonant-vowel-consonant syllables. In J.C. Rosenbek, M. R. McNeil & A. E. Aronson (Eds.), *Apraxia of speech : Physiology, Acoustics, and Management.* San Diego : College-Hill Press, pp. 167-196.

Gay, T. (1978). Effect of speaking rate on vowel form ant movements. *Journal of the Acoustical Society of America, 63* (1), 223-230.

Gay, T. (1981). Mechanisms in the control of speech rate. *Phonetica, 38,* 148-158.

Gopal, H. S. (1987). Temporal aspects of tense and lax vowels in American English: Effects of speaking rale and postvocalic consonantal voicing. Unpublished dissertation. University of Texas at Dallas.

Gopal, H. S. (1990). Speaking rate and its effect on the durations of tense and lax vowels. *Journal of Phonetics, 18,* 497-518.

House, A. s. (1961). On vowel duration in English. *Journal; of the Acoustical Society of America, 33, 11*74-1178.

Johns, D. & Darley, F. L. (1970). Phonemic variability in apraxia of speech. *Journal of Speech and Hearing Research, 13,* 556-583.

Kent, R. D. & Rosenbek, J. C. (1983). Acoustic patterns of Apraxia of Speech. *Journal of Speech and Hearing Research, 26,* 231-249.

Klatt, D. H. (1976). Linguistic uses of segmental duration n English ; Acoustic and Perceptual evidence. *Journal of the Acoustical Society of America, 59(5), 1200-1221.*

Klich, R. J. Ireland, J. v., & Weidncr, W.E (1979). Articulatory and phonological aspects of consonant substitutions in apraxia of speech. *Cortex, 15,* 451-470.

Lehiste, I. *Suprasegmenlals.* MIT Press, Cambridge, Mass., (1970).

Lehiste, I. (1972). the timing of utterances and linguistic boundaries. *Journal of the Acoustical Society of America, 51,* 2018-2024.

Lindblom, B.(1963). spcctrographic study of vowel reduction. *Journal of the Acoustical Society of America, 35,* 1773-1781.

Lindblom, B. (1967). *Vowel duration and a model of lipmandible co-ordination.* Speech Transmission Lab., Royal Institute of Technology, QPSR 4/1967, pp. 1-29.

Martin, A. d. & Rigrodsky, S. (1974). An investigation of phonological impairment in aphasia, part 1. *Cortex, 49,* 317-328.

McNeilage, P. F. (1973). Motor control of serial ordering in speech. *Psychology Review, 77,*182-196.

Nooteboom, S. G. (1972). *Some timing factors in the production and perception of vowels.* Occasional papers, University of Essex, Language Centre, Colchester, September 1972, 1, pp. 25.

Peterson, G. E. & Lehiste, I. (1960). Duration of syllable nuclei in English. *Journal of the Acoustical Society of America, 32,* 693-703.

Port, R. F. (1981). Linguistic timing factors in 000combination. *Journal of the Acoustical Society of America,* 69(1), 263-274.

Rosenbek, J. C.: (1980). Apraxia of speech - Relationship to stuttering. *Journal of Fluency Disorders, 5,* 223-253.

Skenes, L. L. (1987). Durational changes of apraxic speakers. *Journal of Communication Disorders, 20,* 61-71.

Weismer, G. & Fennel, A. M. (1985). Constancy of (acoustic) relative timing measures in phrase-level utterances. *Journal of the Acoustical Society of America,* 78(1), 49-57.

Wertz, R. T. (1978). Neruopathologies of speech and language : An introduction to patient management. In D. Johns (Ed.), *"Clinical Management of Neurogenic Communicative Disorders".* Boston : Little Brown & Company (1978).

Zar, J. J. *Biostatistical Analysis.* Prentice-Hall, Inc. New Jersey. Second Edition (1984).

Ziegler, W. & von Cramon, D. (1986). Timing deficits in apraxia of speech. *European Archives of Psychiatric Neurological Science, 236,* 44-49.