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The Effects of Noise Masking on Vowel Duration in Three Patients with Apraxia of Speech and a Concomitant Aphasia

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A slow speaking rate has been reported with some consistency for persons exhibiting aphasia and/or apraxia of speech (AOS), (Ryalls, 1981; Collins, Rosenbek, and Wertz, 1983; Kent and Rosenbek, 1983; Strand, 1987; Baum, Blumstein, Naeser, and Palumbo, 1990; Seddoh, Robin, Sim, Hageman, Moon, and Folkins, 1996). Kent and Rosenbek (1983) noted that this slowness can be of two types: articulatory prolongation and syllable segregation. Syllable segregation entails the insertion of intersyllabic pauses, increased interword pauses and constitutes an increase in intervals of silence. Articulatory prolongation is characterized by prolonged transitions and steady states which manifests as lengthened segments and errors controlling relative segment durations. Articulatory prolongation is the type of slowness that the present study investigates. Kent and Rosenbek (1983) posit that articulatory prolongation may have adverse consequences on the process of producing speech as well as on the perceptual adequacy of the output.

> "The slow rate probably contributes significantly to the perceptual impression of apraxic speech as effortful and groping. Furthermore, slow rate may be predisposing to increased timing error—that is, errors in the timing of individual movements—given that slow speakers have larger timing errors than fast speakers."
>
> (Kent and Rosenbek, 1983, p. 243)

Slow speaking rate might be a contributing factor causing some of the speech abnormalities observed in patients with AOS and a concomitant aphasia. Thus, research is warranted to understand better why these patients' speech is slow and whether slowness itself should be targeted as a goal for remediation. It is also of theoretical import to determine whether the slow speaking rate is an intrinsic consequence of brain damage to the left perisylvian region (i.e., part of the impairment), or rather, whether it is a compensatory strategy that is commonly adopted by this patient population.

Investigations of articulatory prolongation have frequently examined the temporal parameter of vowel duration. The production of vowels in connected speech and polysyllabic utterances has been characterized as being significantly longer compared to normal controls in speakers with aphasia (Baum, Blumstein, Naeser, and Palumbo, 1990; Ryalls, 1986; Ryalls, 1981), as well as in speakers with AOS (Strand, 1987; Collins, Rosenbek, and Wertz, 1983; Kent and Rosenbek, 1983). Some investigations have found that speakers with AOS can and do decrease vowel length in contexts of increasing utterance length (e.g., zip, zipper, zippering). Collins et al. (1983) and Strand (1987) found that stem vowel durations (e.g., the /I/ in "zip"), produced by both normal speakers and speakers with AOS decreased as utterance length increased, but that speakers with AOS reliably produced longer vowels than the normal controls in all utterance length contexts. In general, the vowels produced by speakers with AOS in polysyllabic word and phrase length contexts have been found to be elongated relative to normal vowel durations. Furthermore, this prolongation persists despite the AOS speaker's ability to make relative decreases in vowel duration in contexts of increasing word length.

Baum et al. (1990) reported that the vowels produced by the anterior aphasic subjects in their study were significantly longer than either the posterior aphasic subjects or the aphasic subjects with both anterior and posterior lesions. Baum et al. (1990) suggested that the longer vowel durations produced by the anterior aphasic patients might be attributed to either a general slowness of articulation or to difficulty planning/implementing the post-vocalic segments. Relative to the former account, McNeil, Caliguiri, Weismer, and Rosenbek (1986) reported that although persons with AOS produced longer vowel durations, the peak velocities of their lip and jaw movements were within normal limits. The movement transduction data were interpreted to suggest that the longer vowel durations produced by speakers with AOS should not be attributed to a general articulatory slowness, but instead to a disturbance of spatiotemporal motor programming, or perhaps, an inability to maintain control over the steady state. Accordingly, articulatory prolongation could be accounted for by either deficiencies at the level of constructing or retrieving the temporal plan for movement or to deficiencies implementing the temporal plan, perhaps due to unreliable integration of information concerning the timing of movements as they are being executed.

Another alternative hypothesis to either the *slowness of articulatory movements* or the *spatiotemporal planning* accounts is that speakers with AOS and a concomitant aphasia elongate vowels in an attempt to utilize auditory information during the steady state portions of an utterance. Support for the hypothesis that prolonged vowel duration may be related to auditory monitoring is derived in part from Ladefoged (1967; as cited by Fucci, Crary, Warren, and Bond, 1977), who hypothesized that vowels were regulated primarily by auditory feedback, as they were most affected by white noise masking. This was in contrast to consonants which were viewed as being primarily regulated by oral sensory feedback, as they were most affected by oral anesthesia. Differences between vowel and consonant performance were also demonstrated by Fucci et al. (1977) who found that auditory masking had a lengthening effect on vowel duration while consonant duration consistently decreased among normal speakers. Fucci et al. (1977) suggested that vowel lengthening may be attributed to the speaker's attempt to search for a target position without adequate auditory feedback. This hypothesis is extended to question whether the vowel elongation phenomenon noted among persons with AOS and a concomitant aphasia can be attributed to the disordered speaker's attempt to monitor vowel accuracy. In other words, it is proposed that because vowel duration can be prolonged to permit on-line monitoring of target vowel accuracy (Fucci et al., 1977), this opportunity may be utilized by persons with AOS and a concomitant aphasia as a compensatory behavior, perhaps as an attempt to compensate for their diminished access to a normal motor program. By comparing vowel durations obtained under conditions of white noise masking to those obtained under normal listening conditions, this study tests the hypothesis that persons with AOS and a concomitant aphasia prolong vowels in order to monitor auditory information.

To date there have been only a few studies that have examined the effects of masking on the speech of persons with aphasia or AOS. Birch (1956) investigated 14 expressive aphasic subjects and found marked improvement in the areas of naming, reading aloud, and enunciation under conditions of masking. While historically interesting, this report lacks specificity concerning the nature of the subjects' speech production impairments as well as a detailed account of the improvements observed under masking conditions. Thus it is difficult to interpret Birch's findings in light of the current question. Deal and Darley (1972) examined phonemic accuracy under conditions of masking with 12 subjects with AOS and found no significant changes with respect to the number of phonemic errors produced during oral reading. Neither of these studies examined the effects of masking on temporal parameters of speech production.

This investigation examines whether one of the contributing factors causing vowel prolongation in individuals with AOS and a concomitant aphasia is the disordered speaker's reliance on auditory feedback, perhaps as an attempt to increase the accuracy of vowel production. If vowel durations are elongated because persons with AOS and aphasia are attempting to utilize auditory feedback during speech production, then vowel durations should decrease under conditions of masking. Accordingly, the following experimental question was posed: Do vowel durations decrease under conditions of masking for persons with AOS and a concomitant aphasia?

METHOD

Subjects

Three males with mild-to-moderate aphasia and moderate-to-severe AOS served as subjects. Subject A1 had a diagnosis of primary progressive aphasia and subjects A2 and A3 had aphasia secondary to cerebral vascular accidents in the distribution of the left middle cerebral artery. Three neurologically normal males were selected to serve as matched controls on the basis of age, years of education, and regional dialect. Table 1 displays information about the disordered subjects and an asterisk indicates those parameters by which normal controls were identified for participation. Additionally, the control subjects were selected on the basis of having no history of speech, language, hearing, or neurological impairments. Table 2 displays selected test results obtained within a two-week period of the experiment. In addition to assessment using the Dabul Apraxia Battery (Dabul, 1979), the disordered subjects were judged to have AOS by three experienced speech pathologists. The criteria employed by these speech pathologists were the same four characteristics Kent and Rosenbek used to make the determination that a subject would be called apraxic (1983, p. 232). The results reported for the Boston Naming Test (BNT; Kaplan, Goodglass, and Weintraub, 1983), reflect a secondary scoring method in which full credit was given for errors that were judged to be apraxic in nature (i.e., productions containing phonemic errors which nonetheless closely approximated the target). The disordered subjects were selected on the basis of having predominantly expressive language impairments with moderately severe AOS. The severity of AOS was determined by a combined use of the Dabul Apraxia Battery and clinical agreement by the three speech pathologists. All three disordered subjects were receiving speech–language treatment at the time of testing and for at least 6 months prior to serving as subjects.

Table 1. Subject Characteristics

Subject	*Age* *	*Education* *	*Dialect* *	*Etiology*	*MRI Results*	*Time Post Onset*
A 1	71	MA	NW	Primary Progressive Aphasia	Normal (marginal widening of left perisylvian region)	56 months post first symptoms
A 2	47	2 years in college	NW	Embolic CVA	Multiple infarcts in distribution of left MCA	12 months
A3	39	High school	NW	Thrombotic CVA	Multiple infarcts in parietal, insular, and operculum areas	63 months

Note: Parameters by which control subjects were matched; Age-years; MRI = magnetic resonance imaging.

Table 2. Aphasia/Apraxia Characteristics

Subject	*Western Aphasia Battery*	*RCBA: VI Sentence-Picture*	*Boston Naming Test*	*Dabul Apraxia Battery*	*Shortened Token Test*	*ACTS*
A1	Sequential Commands 76/80	100%	58/60	Moderate-Severe	21.5/29	19/21
A2	Aphasia Quotient 83.50	100%	40/60	Mild-Moderate	20.5/29	20/21
A3	Aphasia Quotient 67	90%	38/60	Moderate-Severe	10/29	18/21

Note: RCBA-Reading = Comprehension Battery for Aphasia; ACTS = Auditory Comprehension Test of Sentences.

Stimuli and Procedure

Target vowels were elicited in CVC contexts under normal listening conditions and under conditions of white noise masking in the carrier phrase "Say _____ again." The target stimuli, which consisted of 14 monosyllabic words beginning with /b/ and ending with either /t/ or /d/, contained the following vowels: /aI, ae, u, i, E, Λ, I/. Prior to the experiment, the subjects were asked to use the target items in a sentence completion task in order to familiarize them with the tokens in a semantically meaningful context.

White noise masking was delivered through Grason Stadler earphones (TDH 39-10Z) at 90 dBA measured at the earphones by a Quest sound level meter (ANSI S1.4 Type 2-IEC). Subjects were instructed to monitor a VU meter in order to maintain a consistent loudness level across masked and unmasked conditions. All subjects produced each target in the carrier phrase at least six times, three under normal listening conditions and three under masking conditions. The listening conditions were alternated, starting with the normal listening condition, until each token had been produced three times in each listening condition.

The subjects' productions were recorded using a Sony DAT recorder (TDC-D10 Pro II) and later digitized at 22 kHz into CSpeech (Milenkovic, 1994). Vowel durations were measured 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, served to demarcate the boundaries of the vowel segment in the spectrogram. Approximately 15% of the tokens were remeasured by a second person in order to assess the reliability of the measurement procedure. Tokens were randomly selected from both conditions (masked and unmasked) across all six subjects to be remeasured.

RESULTS

The experimenter judged the phonemic accuracy of every utterance immediately after the subject produced the token. When a phonemic error occurred, the experimenter gestured to the subject to repeat the utterance. Control subjects also had to repeat tokens when the experimenter judged that it had been produced in a manner that did not sound natural. Some subjects tended to over-articulate, and in these instances, they were instructed to speak as if they were in a conversation. Only the phonemically accurate and natural sounding productions were used in the analyses.

A portion of the vowel durations obtained in both listening conditions were independently measured by two of the authors. The results were analyzed using Pearson's correlation coefficients and are presented in Table 3.

Overall, the measurements were found to be highly reliable across all subjects and listening conditions ($r > .95$). The mean difference between measurements ranged from 1.8 msec to 7.5 msec.

The average vowel duration for each subject by condition is presented in Table 4. Figures 1, 2, and 3 show the mean vowel durations produced by each experimental subject relative to their individually matched control across listening conditions. Analysis of variance results comparing each experimental subject with their matched control are tabulated next to each graph.

Figure 4 shows the overall mean vowel durations for all subjects separated into experimental and control groups. The results of the analysis of variance show that the vowel durations produced by the experimental subjects were significantly longer than the controls' ($F_{1,832} = 22$; $p = .000$). The

Table 3. Reliability of Vowel Duration Measurements

Subject	*Percent Remeasured*	*Mean Difference Between Measurements*	*Pearson Correlation Coefficient*
A 1	12% (20/168)	4.2 ms	$r = 0.990$
A 2	17% (14/84)	1.8 ms	$r = 0.999$
A 3	14% (12/84)	2.4 ms	$r = 0.998$
C 1	12% (20/168)	7.5 ms	$r = 0.984$
C 2	17% (14/84)	7.2 ms	$r = 0.963$
C 3	17% (14/84)	1.9 ms	$r = 0.984$

Table 4. Mean and Standard Deviations of Vowel Durations (MSEC) in Unmasked and Masked Listening Conditions

	Subject	*Unmasked*		*Masked*
Apraxic				
	A 1	Mean	167	160
		SD	62	54
	A 2	Mean	210	231
		SD	66	69
	A 3	Mean	210	235
		SD	47	61
Controls				
	C 1	Mean	135	151
		SD	35	35
	C 2	Mean	184	211
		SD	52	66
	C 3	Mean	128	122
		SD	32	27

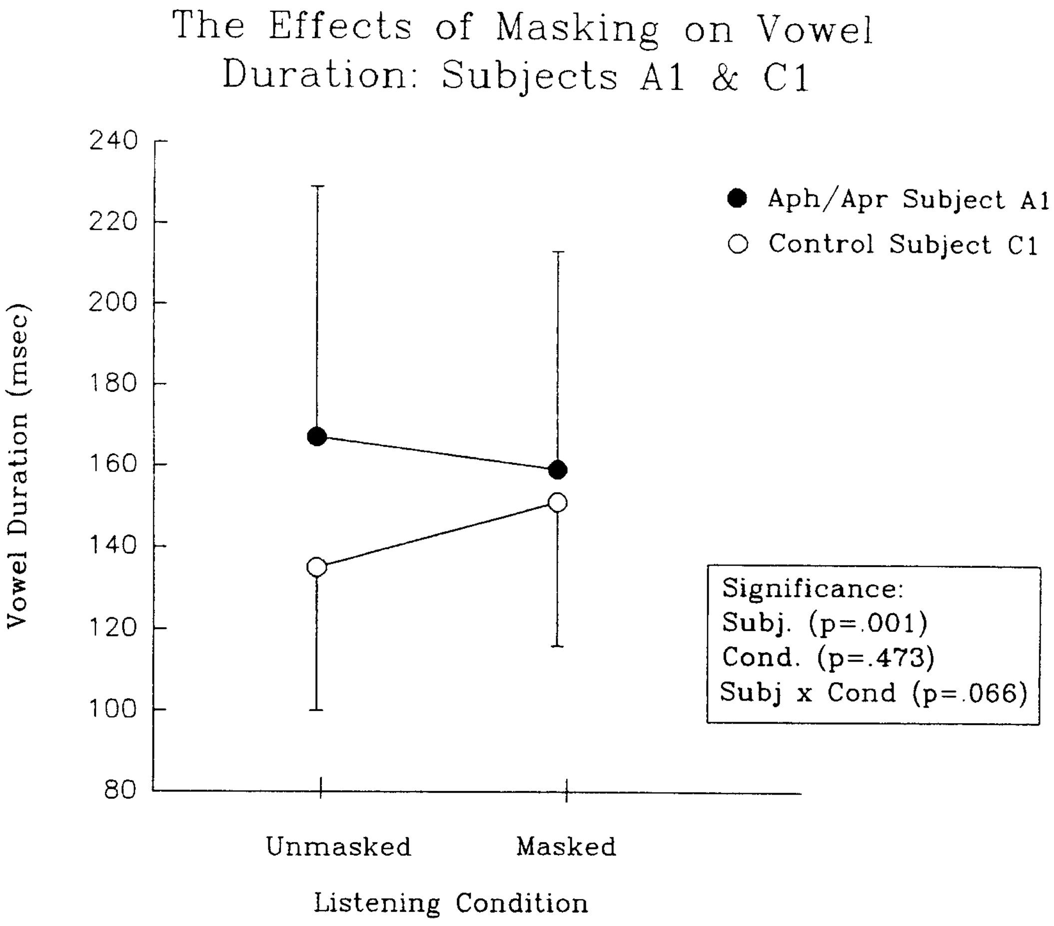

Figure 1. The Effects of Masking on Vowel Duration: Subjects A1 and C1.

masking condition yielded significantly longer durations for both groups ($F_{1,832}$ = 5.4; p = .020). The mean increase in vowel duration from the unmasked to the masked listening condition was 13 msec for the disordered subjects and 12 msec for the controls. The interaction between subject group and listening condition was not significant ($F_{1,30}$ = .008; p = .927).

DISCUSSION

The effects of masking on vowel duration did not differ for the disordered and control subjects. Both groups exhibited the typical behavior of increasing duration under conditions of masking. Subject A1 might be an exception to the overall pattern of results in that his vowel durations in the masked condition did exhibit a slight decrease (an average of 7 msec).

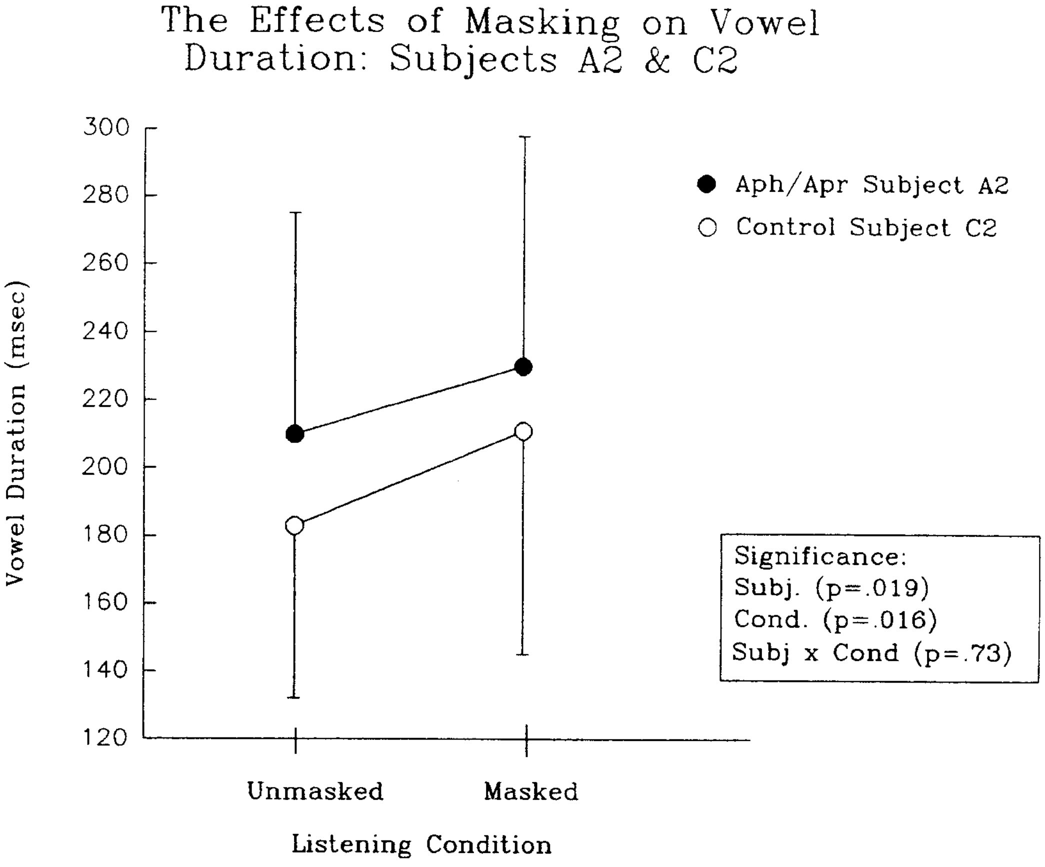

Figure 2. The Effects of Masking on Vowel Duration: Subjects A2 and C2.

This trend noted with subject A1 should be interpreted with caution because one control subject, C3, also exhibited slightly decreased vowel durations in the masking condition (an average of 6 msec). Subject C3 was the only control that had consistent difficulty speaking naturally as he tended to over-articulate. For both subjects, A1 and C3, the amount of decrease in vowel duration under conditions of masking was in the range of measurement error (i.e., approximately 7 msec). Because vowel durations increased approximately 20 msec under conditions of masking for all other subjects, the slight decreases exhibited by A1 and C3 are not given much weight. These disordered and matched control comparisons were conducted on a case-by-case basis in order to examine the individual differences among the disordered subjects. However, individual differences among the normal controls were as variable as the disordered group. The results of the analyses of individual subject pairs suggest that normal controls are equally as likely to exhibit the atypical effect of decreasing vowel length under conditions of masking as are the speakers with aphasia and AOS.

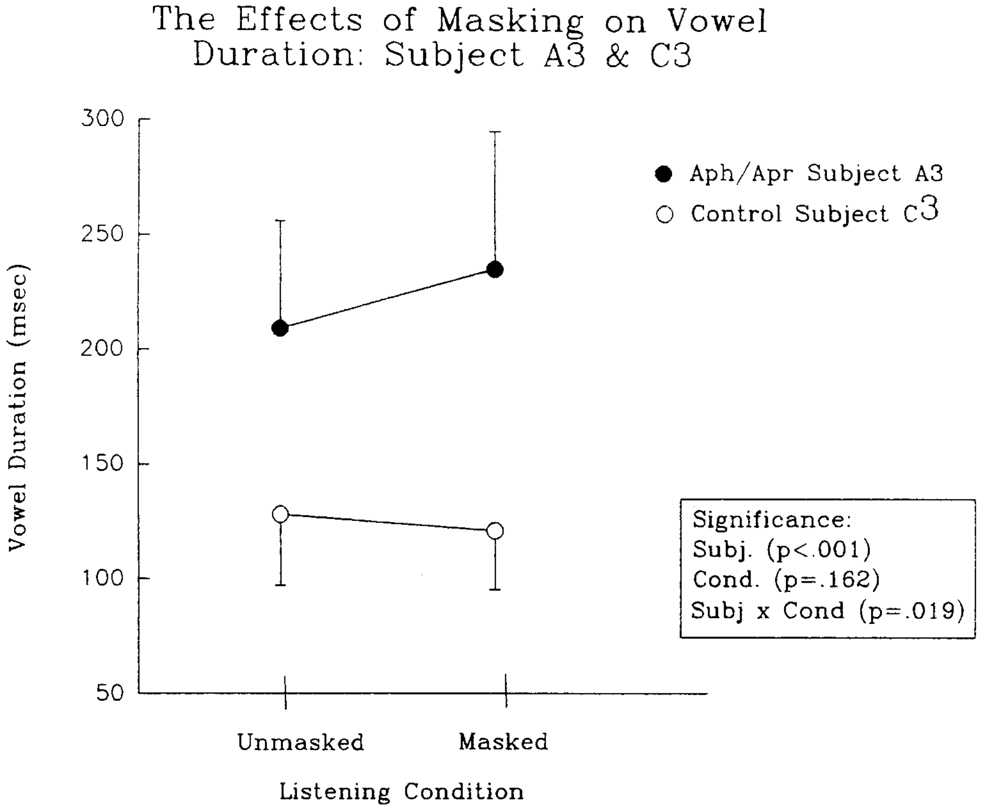

Figure 3. The Effects of Masking on Vowel Duration: Subjects A3 and C3.

Decreasing vowel length under conditions of masking is considered atypical because speakers tend to increase intensity and prolong vowels when they can not hear themselves—the Lombard or "hair dryer" effect (Lane and Tranel, 1971). The durational changes under conditions of masking are attributed to the increase in loudness and a tendency to simultaneously over-articulate. The subjects in this study were instructed to monitor a VU meter and maintain a consistent loudness level. Despite the best of intentions, it is possible that this goal may not have been entirely achieved. The acoustic analysis did not include measurement of root mean square (RMS) values, though the examiner perceptually monitored the subjects' loudness level during the course of the experiment. To the extent that changes in loudness did occur, it seems unlikely that it would have differentially affected the two groups of subjects.

The data was primarily examined on a case-by-case basis, comparing each disordered subject to his matched control because the Primary Progressive Aphasia subject did perform somewhat differently than the other two subjects with aphasia. Though the differences were slight, there is some indication that this subject (A1), might have performed differently due

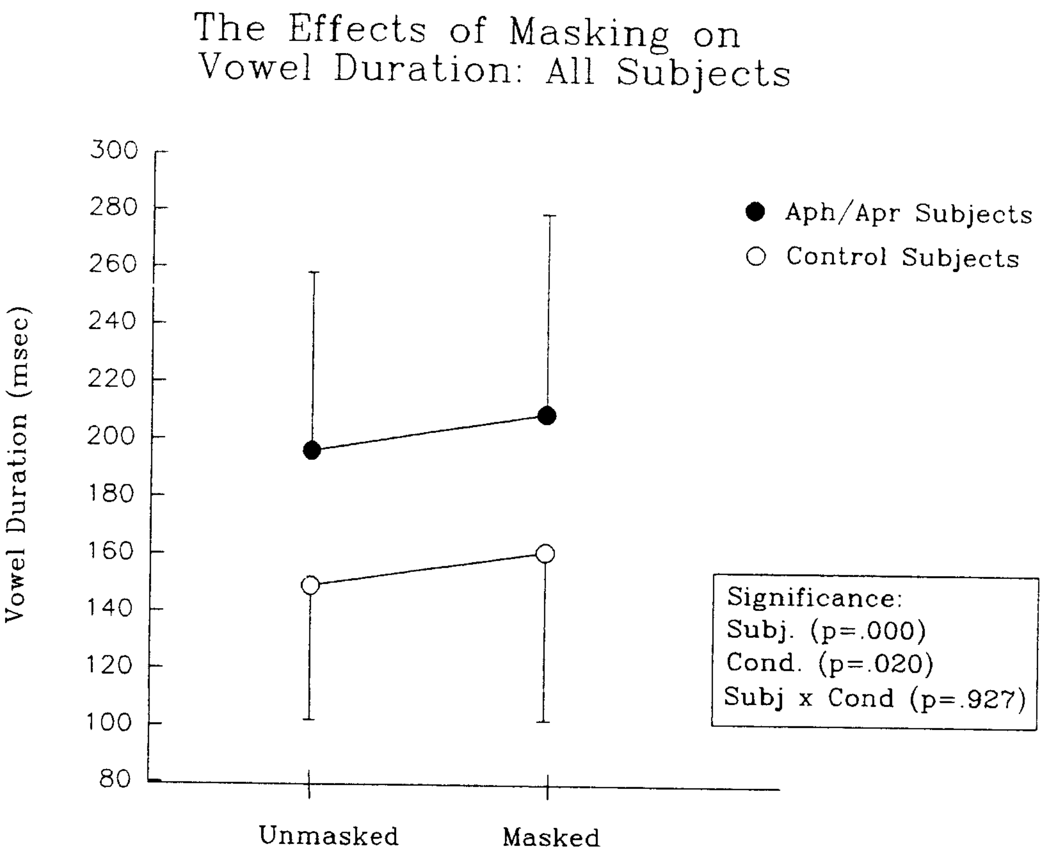

Figure 4. The effects of masking on vowel duration: all subjects.

to the slowly progressive nature of his disorder. It is possible that different compensatory strategies might be developed in patients for whom the onset of the motor speech and language disorder is degenerative as opposed to sudden. However, further research is required to assess this hypothesis.

Despite the small number of disordered subjects and the possibility that intensity variations would have differentially affected the vowel durations of the two groups, the results of this study tend to suggest that vowel prolongation is not primarily due to the disordered subject's attempt to monitor auditory information. To what then can we attribute the robust observation that speakers with AOS and aphasia tend to elongate vowels in connected speech? Kent and Rosenbek (1983) hypothesized that syllable segregation allows the speaker with AOS more time to plan the utterance. It is possible that articulatory prolongation also might serve the function of providing increased planning time for the programming, retrieval, and/or initiation of upcoming segments. The key to this puzzle may lie in better delineating those conditions that yield vowel prolongation and those that do not.

Three investigations have reported that vowel durations produced by persons with AOS are shorter or equivalent to the durations produced by normal speakers: Duffy and Gawle (1984), Caligiuri and Till (1983), and Ryalls (1986). In all three of these studies, vowel durations were measured in CVC monosyllabic speaking contexts. The production of monosyllables arguably entails fewer demands on planning for upcoming segments as the only segment following the vowel is the post-vocalic consonant. Differential performance by speakers with aphasia and AOS in monosyllabic versus polysyllabic contexts lends support to the hypothesis that articulatory prolongation can be ascribed to some aspect of planning, retrieval, and/or initiating the movements for upcoming segments.

Further investigations examining the effects of varying the length and complexity of post-vocalic targets are currently underway. The ultimate direction of this research is to better delineate the source(s) of articulatory prolongation in speakers with AOS and a concomitant aphasia. This information should promote a clearer understanding of how articulatory prolongation contributes to both the speech production successes and failures of speakers with AOS and a concomitant aphasia.

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