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The nature of speech production impairments in anterior aphasics: An acoustic analysis of voicing in fricative consonants

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Abstract

This study investigated the acoustic characteristics of voicing in English fricative consonants produced by anterior aphasics and the effects of phonetic context on these characteristics. Three patients produced voiced and voiceless fricative–vowel syllables in isolation, following a voiced velar stop, and following a voiceless velar stop. Acoustic analyses were conducted of the amplitude and patterning of glottal excitation, as well as fricative noise duration. Results showed that, although the patients are able to coordinate the articulatory gestures for voicing in fricative consonants, they demonstrated abnormal patterns of glottal excitation in the amplitude measures, owing to weaker amplitudes of glottal excitation in voiced fricatives. Context effects failed to emerge because of dysfluent speech. These results suggest that the locus of the speech production deficit of anterior aphasics is not at the higher stages of phoneme selection or planning but rather in articulatory implementation, one related to laryngeal control.

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1. Introduction

One of the clinical manifestations of aphasia is the presence of deficits in speech production. These deficits are particularly evident in anterior aphasics. These patients are diagnosed either in terms of their primary lesion site (i.e., anterior brain structures), their speech output characteristics (i.e., nonfluent, slow, labored, and phonetically distorted), or their aphasic classification (i.e., Broca's aphasia). Recent research in aphasia has sought to determine the nature of these deficits, often

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focusing on the stages of processing that underlie speech production. More specifically, the deficits displayed by these patients could be the result of either higher order impairments in the selection and planning of speech output or a low level impairment in the articulatory implementation of selected and planned speech segments.

While a number of studies have indicated that nonfluent aphasics including Broca's aphasics have impairments in the selection and planning of speech segments (Blumstein, 1973; Dunlop & Marquardt, 1977; Hatfield & Walton, 1975; Klich, Ireland, & Weidner, 1979; Trost & Canter, 1974), the more prevalent characteristic of nonfluent aphasic speech is a severe phonetic disorder relating to the articulatory implementation of the phonetic parameters of speech (see Blumstein, 2001, for review). Studies investigating the acoustic properties or articulatory parameters underlying phonetic dimensions such as voicing and nasality have shown that the anterior aphasics demonstrate significant deficits (Blumstein, Cooper, Goodglass, Statlender, & Gottlieb, 1980; Blumstein, Cooper, Zurif, & Caramazza, 1977; Freeman, Sands, & Harris, 1978; Gandour & Dardarananda, 1984a; Itoh, Sasanuma, Hirose, Yoshioka, & Ushijima, 1980; Itoh et al., 1982; Itoh, Sasanuma, & Ushijima, 1979; Shewan, Leeper, & Booth, 1984). These deficits have been characterized in terms of an impairment in the timing and coordination of two independent articulators. In the case of voicing in stop consonants, it is the timing relation between the release of the stop closure and the onset of glottal excitation. In the case of nasal consonants, it is the timing relation between the release of the closure in the oral cavity and the opening of the velum.

Nonetheless, the anterior aphasics' disorder in temporal coordination does not appear to be symptomatic of a pervasive timing impairment. Acoustic analyses of the speech productions of these patients show that they produce normal fricative durations (Harmes et al., 1984), maintain intrinsic fricative duration differences that vary with place of articulation (Baum, Blumstein, Naeser, & Palumbo, 1990), and appropriately modify fricative/syllable durations at slow and fast rates of speech (Baum, 1996). Similarly, studies of vowel production have indicated spared timing patterns in these patients (Baum et al., 1990; Gandour & Dardarananda, 1984b; Ryalls, 1987). They maintain intrinsic (tense/lax) and contrastive (context-dependent) vowel duration in English (Baum et al., 1990), as well as durational phonemic contrasts in Thai (Gandour & Dardarananda, 1984b).

Additional analyses suggest that anterior patients have deficits in laryngeal control apart from laryngeal timing or coordination with the supralaryngeal vocal tract. For example, changes in the spectral characteristics of stop consonants (Shinn & Blumstein, 1983) and fricative consonants (Harmes et al., 1984) in utterances produced by anterior aphasic patients have implicated potential pathological maneuvers of the larynx. Additionally, such patients have shown prosodic impairments, impairments that also suggest poor laryngeal control. Their productions show restrictions in fundamental frequency range (Cooper, Soares, Nicol, Michelow, & Goloskie, 1984; Harmes et al., 1984; Kent & Rosenbek, 1983; Ryalls, 1982), abnormally long syllabic nuclei involving unstressed vowels (Kent & Rosenbek, 1983), and a lowered proficiency in correctly implementing lexical tone (Gandour et al., 1992).

There are other phonetic attributes of speech that require a complex interaction of timing parameters and laryngeal control, and hence provide a good test case of the hypotheses proposed for the basis of the speech output impairments of anterior aphasics. Voicing in fricative consonants is a good case in point. While the distinction between voiced and voiceless fricatives has classically been defined as the presence or absence of glottal excitation throughout the duration of the fricative noise, more recent studies have shown a number of other acoustic differences

between voiced and voiceless fricatives, including the duration of fricative noise (Crystal & House, 1988) and the pattern and extent of fundamental frequency transitions into the following vowel (Stevens, Blumstein, Glicksman, Burton, & Kurowski, 1992). Moreover, the distinction between the presence and absence of glottal vibration coincident with fricative noise is more complicated than the traditional picture allows. Even in phonetically voiced fricatives, glottal excitation does not necessarily occur throughout the entire duration of frication noise (Haggard, 1978; Stevens et al., 1992). Rather, voiced fricatives contain considerable periods of little or no glottal excitation. From these findings, it is clear that the simple, binary phonological distinction between voiced and voiceless fricatives depends upon several distinct acoustic properties as well as the patterns within and interactions between these properties. Acoustically, therefore, voicing in fricatives is a “continuous” variable, rather than a clearly defined binary one.

Despite the complex acoustical differences between voiced and voiceless fricatives, several studies have shown that the presence of at least 30 ms of glottal excitation at the beginning or end of the fricative noise duration can consistently and correctly categorize voicing in fricatives across a number of phonetic contexts (Pirello, Blumstein, & Kurowski, 1997; Stevens et al., 1992). Moreover, the patterns of voicing, i.e., whether glottal excitation is more likely to occur at the beginning or end of the fricative noise, are further influenced by the phonetic context in which the fricative appears. Specifically, voiced fricatives following voiced velar stops are more likely to show glottal excitation at the onset of frication (e.g., “big [za]”) (Pirello et al., 1997). In contrast, voiced fricatives preceded by voiceless velar stops or by silence are more likely to show a delay in glottal excitation (e.g. “speak [za]”). In light of these findings, the present study attempts to explore the acoustic parameter for voicing in fricatives in the speech production of anterior aphasic patients by addressing several questions. First, do these patients implement voicing in fricative consonants, as do normal subjects? That is, do they have sufficient articulatory control to implement glottal excitation in the 30 ms at the onset or offset of the fricative noise and is their patterning of glottal excitation similar to normals? Second, will these patients also vary the duration of the fricative noise, as do normal subjects in the production of voiced and voiceless fricatives? That is, do they have sufficient articulatory control to both time the onset of the glottal excitation relative to the fricative noise interval and to maintain the correct durational parameters of the fricative noise segment? Third, will the aphasic patients show influences of phonetic context on the production of voicing, as do normal subjects?

A review of the literature investigating aphasic speech deficits shows that most research has been conducted on utterances produced in citation form. Few studies have examined the production of speech in context and explored the extent to which aphasic patients show normal coarticulatory effects. Those that have suggest that anticipatory coarticulation effects are present in aphasic speech, but that these effects are delayed on the order of 20–30 ms in subjects with nonfluent aphasia or apraxia of speech (Kent & Rosenbek, 1983; Tuller & Story, 1988; Ziegler & von Cramon, 1985). However, a subsequent series of studies performed by Katz and others strongly argued that the time course of anticipatory coarticulation is relatively normal in nonfluent aphasics, though coarticulatory effects are impaired in other ways (Katz, 1988; Katz, Machetanz, Orth, & Schonle, 1990a; Katz, Machetanz, Orth, & Schonle, 1990b; Sussman, Marquardt, MacNeilage, & Hutchison, 1988).

There are suggestions in the literature that anterior aphasics will display impairments in the production of voicing in fricative consonants. Frequent devoicing of initial and medial voiced fricatives has been reported in anterior aphasics’ speech

production (Code & Ball, 1982; Harmes et al., 1984; Kent & Rosenbek, 1983), as has premature onset of voicing in voiceless fricatives before the frication segment has ended (Kent & Rosenbek, 1983). These results suggest an inability to combine vocal cord vibration and supralaryngeal articulatory gestures. Additionally, Harmes et al. (1984) ascribed the presence of numerous 10–30 ms silent gaps during alveolar fricative production ([s z]) in one aphasic speaker to “rapid glottal closure and/or pauses in respiratory drive” (p. 382).

The present study is designed to examine the production of voicing in fricative consonants in both citation form and in phonetic context by anterior aphasic patients. As described below, a number of acoustic measures will be made including the presence of glottal excitation, the patterns of glottal excitation in the fricative noise interval, and the duration of the fricative noise. Although previous studies have explored a number of acoustic parameters associated with the implementation of voicing, these studies have typically not explored the implementation of a number of different acoustic attributes in the same patients. With respect to the role of phonetic context, two broad questions will be addressed. First, will anterior aphasics show increased impairments in the implementation of the phonetic parameters of speech associated with fricative voicing if their utterances are produced in the more complex but more natural condition of surrounding phonetic context? Second, do difficulties in the coordination of independent articulators that have been shown for anterior aphasics lead to deficits in the coarticulatory maneuvers required for making smooth transitions from one speech segment to the next?

2. Method

2.1. Subjects

The aphasic subjects included two adult males and one female. All three subjects were classified as nonfluent aphasics and diagnosed as Broca’s aphasics on the basis of clinical examination (Boston Diagnostic Aphasia Examination, Goodglass & Kaplan, 1972). Table 1 provides an abbreviated clinical profile for each subject, including fluency and articulation ratings as well as lesion localization. All of the subjects were native speakers of English.

2.2. Stimuli

Two sets of stimuli were used, one for each experimental condition. Both sets consisted of CV syllables beginning with the fricatives [s z] followed by one of the vowels [i e a o u]. In the first condition, the target CV syllables were to be produced in isolation, i.e., in citation form. In the second condition, the target CV syllables were to be produced in two contexts: (1) following a voiced velar stop; and (2) following a voiceless velar stop. The isolation condition provided a baseline for comparing potential differences in patterns of voicing as a function of context.

Each fricative–vowel combination occurred eight times in each condition: that is, once in each of eight separately randomized blocks of tokens. The stimuli were printed in orthographic form on 3 × 5 cards. The subjects were instructed to read each token, speaking clearly but naturally. If a subject was unable to produce the actual token by reading the card, the experimenter read the token and asked the subject to repeat it. If the subject could neither read nor repeat a token after several attempts, that token was set aside and attempted once more at the end of the block.

Table 1
Clinical profile of subjects

Subject	A	B	C
Gender	Male	Male	Female
Age at recording	65	55	54
Time post onset	18 years	9 years	9 years
Fluency ^a	.54	.57	.57
Articulation ^a	.40	.50	.50
Z (ACS) ^b	.83	.87	.95
Etiology	Aneurysm	Stroke	Stroke
Lesion	Left frontal lesion involving the posterior half of Broca's area and most of the middle frontal gyrus extending into the white matter deep to these areas, and also includes the head of the caudate and anterior limb of the internal capsule. It extends superiorly into the pre-motor, motor, and sensory cortex areas and the white matter deep to these areas including the periventricular white matter and undercutting fibers of the supplementary motor area. There is a clip in the anterior communicating artery	Lesion involving the entire lenticulostriate artery distribution of the middle cerebral territory including the left caudate nucleus, globus pallidus, and intervening anterior internal capsule. It also involves the medial temporal cortex, insula, and the periventricular white matter on the left anteriorly, as well as the anterior temporal lobe. There is a separate lesion in the area of the sub-thalamic nucleus and thalamic tract	Large left insular lesion extending to the anterior temporal lobe. It spares the anterior region of Broca's area as well as Wernicke's area

^a Language assessment was based on the Boston Diagnostic Aphasia Exam.

^b Z-score for the four auditory comprehension subtests of the BDAE.

Short breaks were provided at the end of each block or as needed. Each subject produced a total of 80 CV syllables in the isolation condition.

In the context condition, each of the fricative–vowel syllables was read in two different sentence contexts which provided the targeted phonetic environments: (1) following a voiceless velar stop consonant (“Please speak _ again”) and (2) following a voiced velar stop consonant (“Say a big _ again”). Each carrier phrase and fricative–vowel combination appeared once in each of eight randomly ordered blocks of sentences for a total of 160 tokens. Pilot work indicated that one of the aphasic patients (Subject A) was unable to produce the full carrier phrase. As a consequence, the context condition was reduced to “Speak_” and “Big_” for this subject. The other two subjects were allowed to use this reduced carrier phrase when multiple attempts were required to correctly produce some of the target tokens. As in the isolation condition, the subjects were instructed to read as clearly and naturally as possible; if the subject could not produce a carrier phrase with its target, the experimenter would utter the sentence and ask the subject to repeat it. Tokens that the subject could not produce after several attempts were presented again at the end of the stimulus block.

Because the goal of this study is to examine the acoustic properties of voicing in fricatives and the effects of phonetic context on voicing patterns, the acoustic analyses were limited to those utterances that were perceived by the examiners as the correct phonemic target. Thus, voicing errors or place of articulation errors were not included in the analyses. However, those tokens that were perceived by the experimenters as distorted versions of the correct target (e.g. “Big sa” with a drawn out “s”) were included.

2.3. Procedure

Stimuli were recorded onto magnetic tape using a Sony Walkman Professional tape recorder and a Sony stereo microphone. The recorded stimuli were then digitized onto a VAX Station II at a sampling rate of 20 kHz with a 9.0-kHz lowpass filter (Butterworth 24 dB/octave) and a 12 bit quantization.

For each fricative–vowel token, cursors were placed at the onset and offset of the fricative noise using a software waveform editor. The beginning of frication noise was located on the waveform display by the presence of high frequency noise. The onset cursor was placed at the earliest position on the waveform at which DFT and LPC analyses showed an increase of at least 10 dB within the high frequency range (i.e., between 5 and 10 kHz) as compared to the background noise level, using a 15 ms full-Hamming window. The offset of frication noise was determined by the absence of high frequency noise in the waveform, the emergence of the harmonic spectrum corresponding to the onset of the following vowel, and by an increase of at least 10 dB in spectral energy at or above 2 kHz. To this end, both LPC and DFT analyses were conducted using a 15 ms full-Hamming window. A concurrent drop of at least 10 dB in the 5–10 kHz frequency range was used as a secondary measure to confirm the termination of frication noise.

If an epenthetic vowel or any other form of vocal cord activity preceded the onset of the fricative noise, it was not included as part of the fricative unless there was frication noise, as determined by the criteria described above, coincident with the vocalic activity. In short, the boundaries of the fricative were defined by the presence of high frequency frication noise.

In the context condition stimuli, a third cursor was placed immediately after the release of the preceding velar stop consonant, e.g., ‘speak CV’ ‘big CV.’ The release of the stop was identified on the waveform as a short burst of noise in the low to middle frequency range (about 2–4 kHz) following a flattening of the harmonic pattern of the preceding vowel. The cursor marked the position at which the spectral energy in this low to middle frequency range returned to background levels, as determined by DFT and LPC analyses with a 15 ms full-Hamming window. The placement of this cursor allowed for measuring the time interval between the release of the final stop consonant and the onset of the fricative noise corresponding to the beginning of the fricative–vowel syllable.

Once the cursor settings were in place, demarcating the boundaries of the fricative noise and the release of the stop consonant, two measures of voicing were taken. The first measure of voicing was based on a metric initially developed by Stevens et al. (1992) to analyze the amount and extent of glottal excitation in fricatives produced in the same vowel and context conditions as those used in the current study. Specifically, the amplitude of the first harmonic during the fricative noise interval was used as a measure of the amplitude of glottal excitation (cf. Stevens et al., 1992). The amount and extent of glottal excitation was charted throughout the duration of the fricative noise as follows. A 30-ms full-Hamming window was placed at the cursor marking the onset of frication noise and the amplitude of the first harmonic (H1) was measured using a DFT analysis. The window was then advanced in 10-ms steps and the first harmonic was recorded at each interval until the entire window had passed the cursor marking the end of frication noise, such that the window included only the vowel. This last window measured the amplitude of H1 for the vowel, which served as the reference amplitude against which the amplitude of the glottal excitation was compared. If the amplitude difference between the vowel and the glottal excitation was less than or equal to 10 dB, the frame was considered voiced; if the amplitude difference was greater than 10 dB, the frame was considered voiceless. Following

Stevens et al. (1992) and Pirello et al. (1997), a fricative was classified as “Voiced” if there were *at least* 30 ms of contiguous voicing (i.e., three consecutive 30-ms windows advanced in 10 ms intervals) during the frication noise interval. If this criterion was not met, the fricative was classified as “Voiceless.” Using this measure, Stevens et al. (1992) accurately classified 83% of voiced and voiceless fricatives produced by normal subjects and Pirello et al. (1997) correctly classified 99.9% of voiceless and 86% of voiced fricatives.

The second measure of voicing investigated was the duration of the fricative noise. The duration was determined by the time interval (in ms) between the cursor settings described above to establish the onset and offset of the fricative noise interval.

3. Results

3.1. Glottal excitation

3.1.1. Measure of glottal excitation

Table 2 summarizes the percent correct classification results of the glottal excitation measure (using the 10 dB criterion) for the three aphasic subjects across the three experimental conditions. As the table shows, the utterances of only one aphasic patient (B) were consistently categorized at 10 dB. Both patients A and C showed classification values for voiced fricatives that were below chance. These results suggest that the anterior aphasics do not have sufficient laryngeal control to distinguish voiced from voiceless fricatives.

To investigate in more detail the potential reasons for the failure of the voiced utterances to meet the voicing criterion, an examination of the distribution of the individual tokens was undertaken. These utterances were compared to the results obtained in a similar study conducted with normal subjects (Pirello et al., 1997). The data from one of those subjects (a 35-year-old male) are presented in the current study (see Fig. 1) to provide a normative baseline from which to compare the performance of the aphasic patients. Figs. 2–4 show this distribution for the three aphasic subjects (A, B, and C). Each data point represents the amplitude of the vowel referent relative to the amplitude of the glottal excitation of the fricative. The fricative amplitude value was determined by selecting the weakest amplitude of the three consecutive windows (advanced in 10 ms intervals) comprising the 30-ms window of analysis. The dotted lines in Figs. 1–4 represent the 10 dB criterion used to categorize voiced and voiceless tokens. As Fig. 1 shows, there is a large separation between the voiced and voiceless tokens of the normal speaker at 10 dB, as well as tight clustering of the fricatives by voicing in all three conditions. In contrast, as

Table 2
Percentages of fricatives correctly categorized using a measure of glottal excitation

Criterion	Fricatives	Subjects			Mean
		A	B	C	
At 10 dB	Voiced	21%	73%	57%	50%
	Voiceless	99%	100%	100%	99%
	Overall	59%	86%	79%	75%
At 20 dB	Voiced	79%	98%	83%	87%
	Voiceless	90%	89%	87%	89%
	Overall	84%	94%	85%	88%

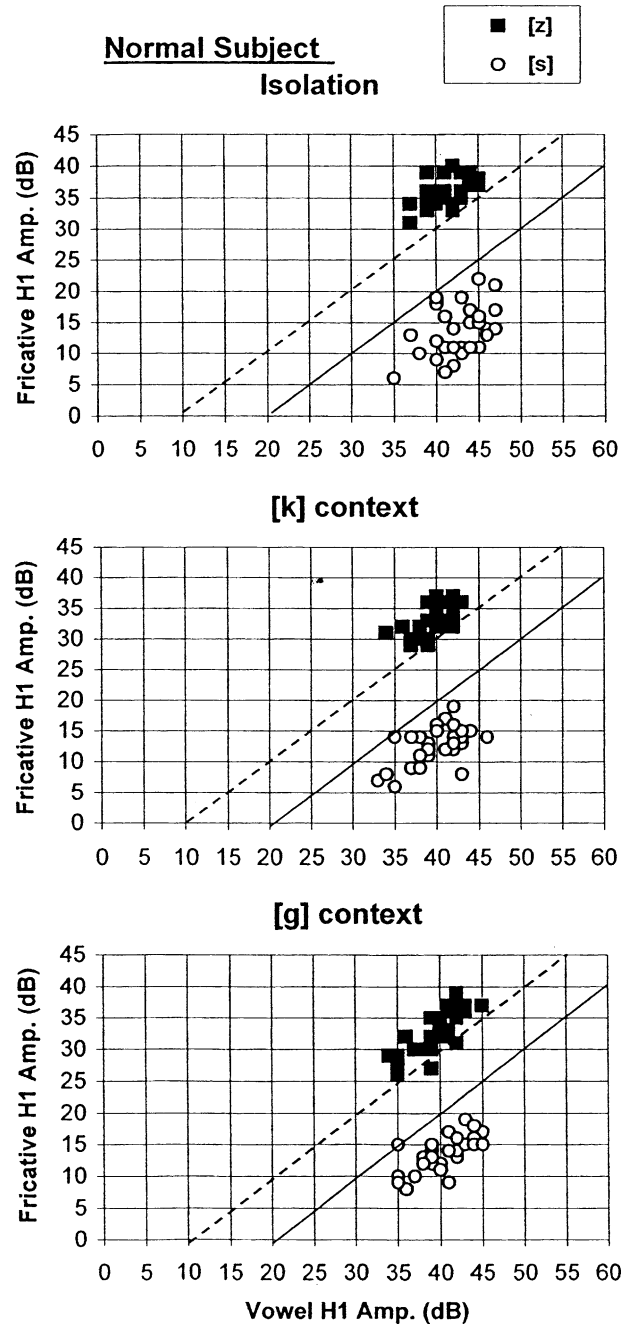


Fig. 1. Scatter plots showing the H1 amplitude of the fricative versus the H1 amplitude of the vowel for each token produced by a normal control subject in isolation, voiceless context, and voiced context. Dark squares represent voiced token targets and open circles represent voiceless token targets. The dotted line and solid line represent the voiced/voiceless boundary using a 10 and a 20 dB criterion, respectively.

shown in Figs. 2–4, there is considerable overlap in the distribution of voiced and voiceless tokens for the aphasic patients, particularly for patients A and C.

Further examination of the distribution of these tokens as shown in Figs. 2–4 suggests that the amplitude values of the voiced fricatives are weaker compared to those of the normal control subject. If the criterion value for defining the presence of

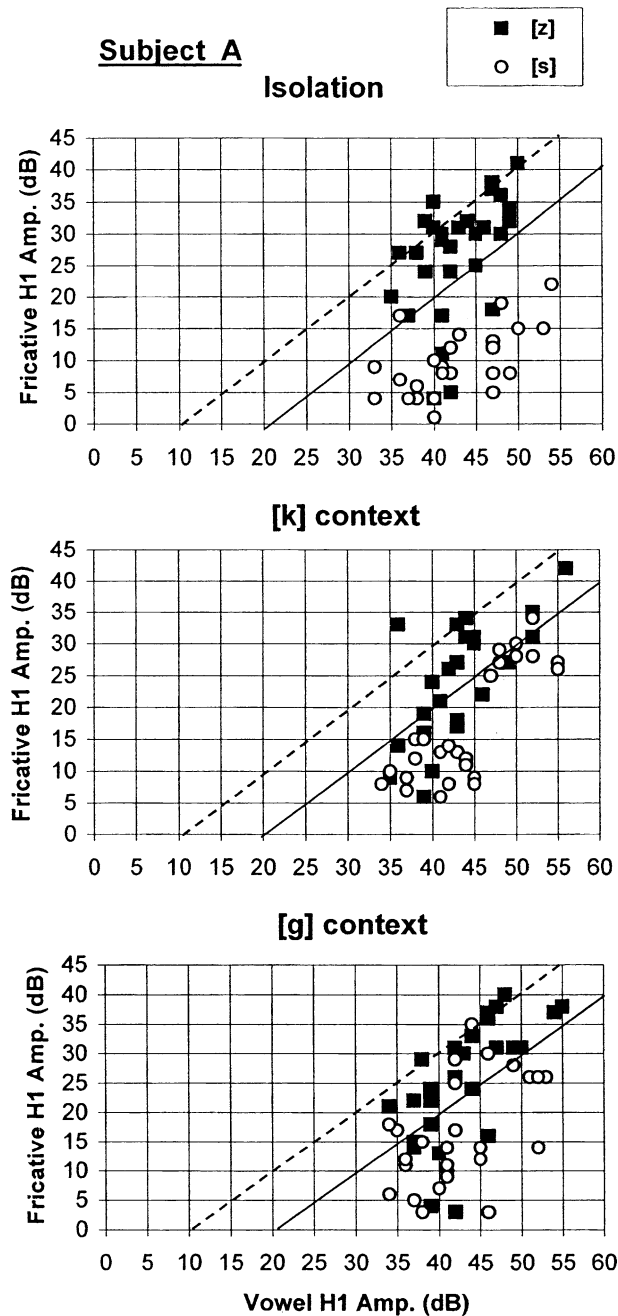


Fig. 2. Scatter plots showing the H1 amplitude of the fricative versus the H1 amplitude of the vowel for each token produced by Subject A in isolation, voiceless context, and voiced context. Dark squares represent voiced token targets and open circles represent voiceless token targets. The dotted line and solid line represent the voiced/voiceless boundary using a 10 and a 20 dB criterion, respectively.

voicing were changed from 10 to 20 dB, as represented by the solid lines in Figs. 1–4, then the patients appear to be able to use glottal excitation to distinguish voiced from voiceless fricative consonants. These findings emerge for each speaker in each condition. To investigate more systematically whether the patients do indeed distinguish voiced from voiceless fricative consonants using glottal excitation, the

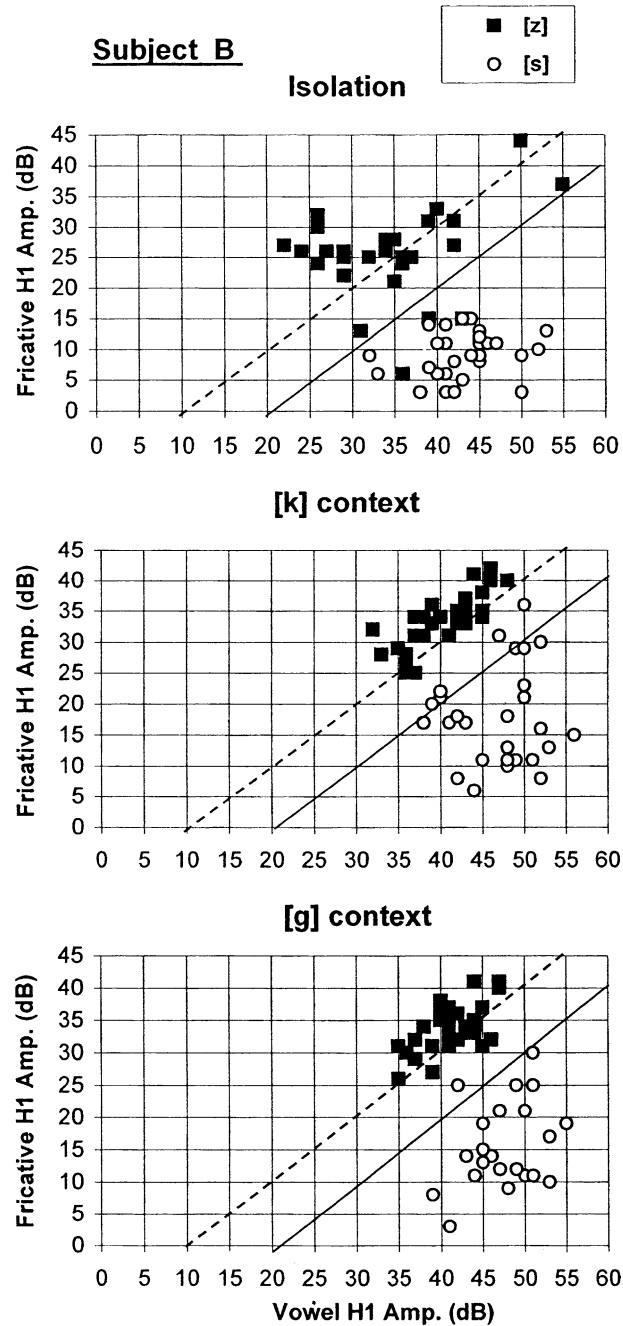


Fig. 3. Scatter plots showing the H1 amplitude of the fricative versus the H1 amplitude of the vowel for each token produced by Subject B in isolation, voiceless context, and voiced context. Dark squares represent voiced token targets and open circles represent voiceless token targets. The dotted line and solid line represent the voiced/voiceless boundary using a 10 and a 20 dB criterion, respectively.

original fricative data were reanalyzed using a 20 dB rather than the original 10 dB criterion value for voicing. Table 2 shows the results. For all patients, the majority of both voiced and voiceless productions are correctly classified, indicating that the patients are able to use glottal excitation to distinguish voiced from voiceless fricatives. Nonetheless, all three aphasic subjects show less distinct voiced/voiceless

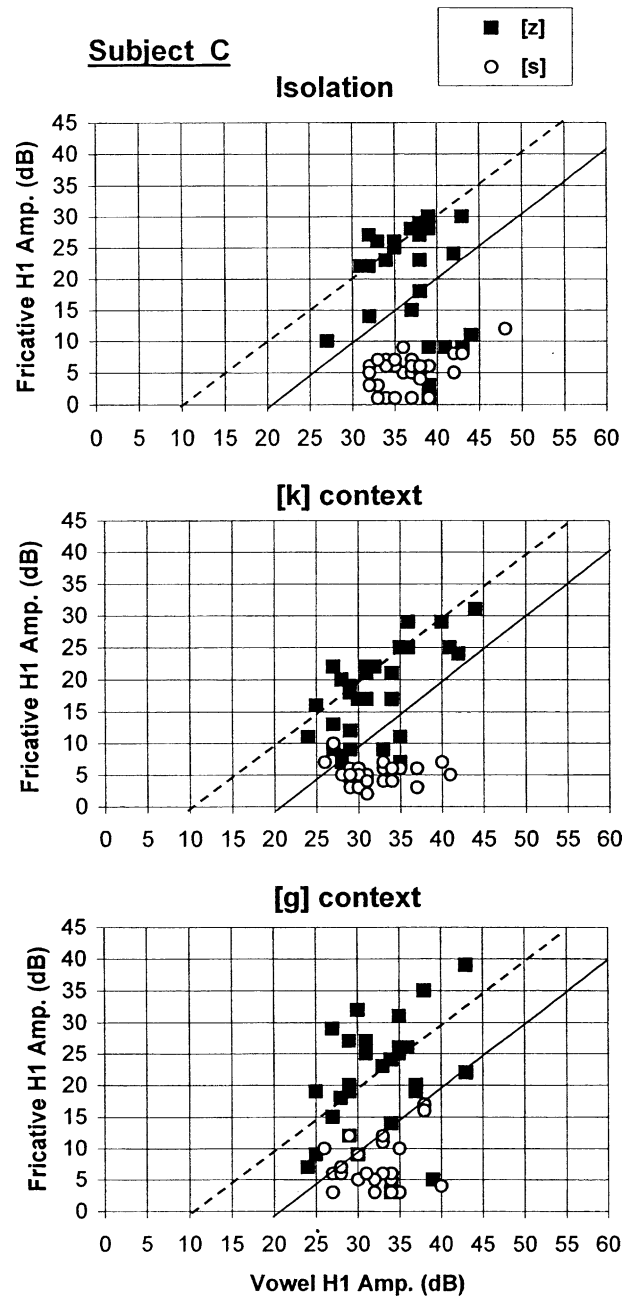


Fig. 4. Scatter plots showing the H1 amplitude of the fricative versus the H1 amplitude of the vowel for each token produced by Subject C in isolation, voiceless context, and voiced context. Dark squares represent voiced token targets and open circles represent voiceless token targets. The dotted line and solid line represent the voiced/voiceless boundary using a 10 and a 20 dB criterion, respectively.

separation and weaker amplitude of glottal excitation for the voiced fricative tokens compared to the normative baseline.

3.1.2. Effects of phonetic context on patterns of glottal excitation

Results of analyses of the production of voiced fricatives in normal subjects show that voiced fricative consonants typically do not contain glottal excitation

throughout the duration of the frication noise (Haggard, 1978; Pirello et al., 1997; Stevens et al., 1992). Rather, there are a number of patterns of glottal excitation that emerge and these patterns appear to be influenced by phonetic context. In particular, when a voiced fricative is preceded by a voiced stop consonant, voicing tends to occur at the onset of frication. Conversely, voiced fricatives that occur after a voiceless stop consonant, where the vocal cords are configured to inhibit glottal vibration, tend to show voicing delay.

To determine whether aphasic patients show a similar influence of phonetic context on the implementation of voicing in fricative consonants, each voiced fricative token was categorized in terms of its voicing pattern including the location and extent of glottal excitation during the fricative noise interval. Based on results of analyses in the preceding section, voicing was defined in terms of the 20 dB criterion. The categories of voicing patterns are summarized in Table 3. These patterns are both exhaustive and mutually exclusive, so that each fricative token falls into one and only one category.

The results of categorizing the aphasic productions into these voicing patterns are summarized in Table 4 for each condition. The isolation condition serves as a baseline from which to compare the two phonetic context conditions. For all three subjects, there was no effect of context on the pattern of voicing in voiced fricative consonants. Subjects B and C showed a preponderance of the ‘Voicing Throughout’ pattern, irrespective of the voicing of the preceding velar consonant, and, as a result, context effects did not emerge.

To further explore the effects of phonetic context on patterns of voicing, the categories of Table 4 were collapsed into two broad categories: voicing at onset and voicing delay. Voicing at onset consisted of tokens in which the first three or more windows of frication noise were voiced according to the 20 dB criterion and included Voicing Throughout and Leading Edge. Voicing delay included tokens which showed a lag of at least one window between the onset of frication noise and the

Table 3
Patterns of voicing

Leading Edge (LE)	At least 30 ms of voicing at the onset of the fricative noise
Trailing Edge (TE)	At least 30 ms of voicing at the end of the fricative noise
Voicing Throughout (VT)	Continuous voicing throughout the duration of the fricative noise interval
Other Voicing (OV)	At least 30 ms of voicing but occurring within the fricative noise interval (i.e., neither at the immediate onset nor offset of the noise)

Table 4
Distribution of voicing patterns in voiced fricatives using a 20 dB criterion

Subject	Condition	Voicing Throughout	Leading Edge	Trailing Edge	Other Voicing
A	After [g]	36%	46%	4%	14%
	After [k]	27%	27%	26%	20%
	Isolation	40%	22%	30%	8%
B	After [g]	97%	—	3%	—
	After [k]	100%	—	—	—
	Isolation	61%	3%	28%	8%
C	After [g]	72%	20%	8%	—
	After [k]	69%	19%	12%	—
	Isolation	42%	—	58%	—

Table 5
Distribution of voicing at onset and voicing delay using a 20 dB criterion for aphasic subjects and overall normative patterns (Pirello et al., 1997)

Subject	Condition	Voicing at onset	Voicing Delay
A	After [g]	91%	9%
	After [k]	67%	33%
	Isolation	64%	36%
B	After [g]	97%	3%
	After [k]	100%	0%
	Isolation	68%	32%
C	After [g]	92%	8%
	After [k]	88%	12%
	Isolation	43%	57%
Normals	After [g]	89%	3%
	After [k]	46%	47%
	Isolation	62%	26%

onset of voicing; this category included Trailing Edge and Other Voicing. Table 5 shows the results of this analysis. Also included in Table 5 are the mean percentages for voicing at onset and voicing delay from the Pirello et al. (1997) data for normal subjects. Again, the isolation condition serves as a baseline from which to compare the two phonetic context conditions. The voiced fricatives of normal subjects show an increase in voicing at onset after voiced velar stops and an increase in voicing delay after voiceless velar stops. The aphasic patients do not show these normative patterns.

The influence of the voicing of a preceding stop consonant on the voicing of the fricative consonant is believed to reflect mechano-inertial forces in the process of the implementation of articulatory gestures (Bell-Berti, Krakow, Gelfer, & Boyce, 1995). Such carryover effects, however, are short-lived, influencing the first 10s of ms of the following segment. It is possible then that the failure to show context effects in the aphasic patients is due less to a failure of the mechano-inertial system than to dysfluent speech output. That is, if the patients were sufficiently dysfluent such that the context word and target word were not immediately temporally juxtaposed, then contextual influences would not have emerged. Examination of duration measures from the offset of the final voiced or voiceless velar stop consonant to the onset of the frication noise for [s] or [z] showed that, indeed, this was the case. There was often a considerable duration of silence between the offset of the final consonant of the context utterance, e.g., ‘speak’ or ‘big’, and the onset of the target fricative–vowel syllable. In fact, the majority of the utterances for each subject (34/38 for Subj. A; 38/58 for Subj. B; and 29/47 for Subj. C) had silent intervals greater than 50 ms, often extending to intervals as long as 2.5 s.

3.1.3. Misclassifications

The previous analyses examined the voiced fricative productions that were appropriately classified using the measure of glottal excitation (i.e., a 20 dB amplitude difference between fricative and vowel sustained for at least 30 ms). Consideration of those productions that failed to meet the 20 dB criterion and hence were classified by the measure of glottal excitation as voiceless provides another potential source of information about the speech production impairments of aphasic patients. There were three sources of misclassification errors: (1) the duration of voicing was less than 30 ms; (2) the H1 amplitude of the fricative was too weak to meet the 20 dB

Table 6
Voiced fricative targets (in%) misclassified as voiceless fricatives using the 20 dB glottal excitation criterion

Subject	Condition	<30 ms Voicing duration	>20 dB Amplitude difference	No voicing	<i>n</i>
A	After [k]	30	20	50	10
	After [g]	83	—	17	6
	Isolation	100	—	—	6
B	After [k]	—	—	—	0
	After [g]	—	—	—	0
	Isolation	100	—	—	2
C	After [k]	83	—	17	6
	After [g]	60	40	—	5
	Isolation	67	—	33	6
Total Misclassified (in%)		68	10	22	—

Table 7
Number of voiceless fricatives misclassified as voiced fricatives using the 20 dB glottal excitation criterion

Subject	Isolation	After [k]	After [g]
A	0	2	6
B	0	7	2
C	0	9	7
Total	0	18	15

criterion; and (3) there was no visible or measurable glottal excitation in the waveform.

Table 6 summarizes the data analysis for aphasic voiced tokens that failed by the glottal excitation measure to be classified as voiced using the 20 dB amplitude criterion. The preponderance (68%) of misclassification errors across subjects was the result of a failure to sustain glottal excitation for at least 30 ms. The second most common misclassification was due to a failure of any measurable glottal excitation to emerge during the fricative noise (22%).

A second approach is to consider the target voiceless fricatives that had glottal excitation during the fricative noise and met the 20 dB criterion. Table 7 summarizes this data. The results show that misclassification of voiceless fricatives occurred only in the context conditions, suggesting that context exacerbates the aphasics' articulatory deficits. Interestingly, this is not the case for the voiced fricative consonants where misclassifications occur across all three speaking conditions (see Table 6).

3.2. Fricative duration

3.2.1. Measure of fricative duration

The measurements of duration of the fricative noise interval are summarized in Table 8, including normative values from Pirello et al. (1997). In keeping with findings reported in earlier studies of both normal (Baum & Blumstein, 1987) and aphasic (Baum et al., 1990) fricative durations, all three aphasic subjects maintained duration differences between the voiced and voiceless fricatives. Namely, voiced fricatives tended to be shorter than voiceless fricatives.

To further determine whether the duration of the fricative noise varied as a function of context, a Consonant Voicing \times Context Condition analysis of variance (ANOVA) was conducted on the duration measures for each subject. Neuman–Keuls tests were

Table 8
Mean duration of fricative noise (in ms) for the aphasic subjects compared to normals (Pirello et al., 1997)

Subject	Isolation		[k]-Context		[g]-Context	
	[s]	[z]	[s]	[z]	[s]	[z]
A	233	196	324	218	298	207
B	195	145	225	139	202	132
C	215	133	207	125	215	134
Mean	214	158	252	161	238	158
Normals	236	167	173	140	174	130

used for all post hoc analyses. For Subject A, a significant main effect was found for Voicing ($F(1, 58) = 29.24$, $p < .0001$). The mean duration of Subject A's fricatives across contexts was 213 ms for voiced tokens and 285 ms for voiceless tokens. There was also a significant main effect for Context ($F(2, 116) = 7.69$, $p < .0001$) due to the fact that the mean duration of fricatives in isolation was significantly shorter than the mean duration of fricatives in the voiced and voiceless contexts. The Voicing \times Context interaction approached significance ($F(2, 166) = 2.99$, $p < .0540$). Although voiceless fricatives were significantly longer than voiced fricatives in all three conditions, the duration differences were greater in the two context conditions than they were in isolation.

Although Subject B's fricative durations tended to be shorter than those of Subject A, the relationships of fricative duration to voicing and context were very similar in the two subjects. A Consonant Voicing \times Context Condition ANOVA showed significant main effects for Voicing ($F(1, 58) = 193.46$, $p < .0001$), Context ($F(2, 116) = 4.04$, $p < .0196$), and a significant interaction for Context \times Voicing ($F(2, 116) = 4.42$, $p < .0141$). The main effect for Voicing shows that, in Subject B's productions, voiced fricatives are significantly shorter than voiceless fricatives (138 ms for voiced fricatives versus 207 ms for voiceless fricatives). Similar to Subject A, the smallest duration difference (51 ms) between voiced and voiceless fricatives was found in the isolation condition compared to the voiceless (86 ms) and the voiced (70 ms) context conditions.

As shown in Table 8, Subject C's voiced fricatives had the shortest absolute durations of all three subjects. For Subject C, mean fricative durations were 131 ms for the voiced fricatives and 212 ms for the voiceless fricatives. A Consonant Voicing \times Context Condition ANOVA showed a significant main effect for Voicing ($F(1, 47) = 203.99$, $p < .0001$), indicating that Subject C's voiced fricatives are significantly shorter than the voiceless fricatives. Unlike the results for Subjects A and B, Subject C's data showed no main effect for Context nor was there a significant interaction of Voicing \times Context. For Subject C, the mean duration difference for the voiced fricatives differed by less than 2.2 ms across the three conditions: 80.17 ms ([g] context); 81.57 ms (isolation); and 82.36 ms ([k] context).

4. Discussion

It was the goal of this study to explore the production of voicing in fricative consonants in nonfluent aphasics. To that end, acoustic analyses were conducted of a number of acoustic parameters contributing to the voiced/voiceless phonetic distinction. These parameters included amplitude of glottal excitation, the patterns of glottal excitation in the fricative noise interval, and the duration of the fricative noise.

The results of these analyses showed that nonfluent patients are able to implement the voicing distinction. That is, these patients appear to be able to coordinate the

articulatory gestures required to implement the multiple acoustic parameters contributing to voicing in fricative consonants. Nonetheless, although they have sufficient articulatory control to implement glottal excitation in the 30 ms at the onset or offset of the fricative noise interval, their patterns of glottal excitation were not normal. Specifically, their production of voiced and voiceless fricatives showed more overlap in the amplitudes of glottal excitation, as well as lower than normal amplitudes of glottal excitation. This was evident both in the analysis of the amplitude distribution of the individual tokens (Figs. 2–4) and in the low percentages of aphasic productions of fricatives that were correctly classified by the 10 dB criterion. Nonetheless, when the amplitude criterion was changed to 20 dB, taking into account the lower amplitude of glottal excitation, over 88% of the fricative tokens were correctly classified, indicating that the aphasics were able to maintain the voiced/voiceless distinction using glottal excitation. These results suggest that the locus of the speech production deficit of the anterior aphasic patients is not at the higher stages of speech production involving phoneme selection or planning but rather in articulatory implementation, one related to laryngeal control. The presence of such an impairment in laryngeal control is consistent with previous findings (Baum et al., 1990; Blumstein et al., 1980; Code & Ball, 1982; Harmes et al., 1984; Kent & Rosenbek, 1983).

The interpretation of these findings needs to be tempered by the fact that the stimuli analyzed in this study were limited to only those utterances that were perceptually correct (i.e., a voiceless fricative target was perceived by the examiners as voiceless). In point of fact, anterior aphasic patients including Broca's aphasics also produce phonemic paraphasias involving the voicing distinction (Blumstein, 1973). Since such utterances were not analyzed in the current study, it is not clear at this time whether the underlying source for these phonemic paraphasias is in the stages of phonological selection and planning or in articulatory implementation.

The acoustic patterns of voicing in the aphasics' fricative productions suggest that this impairment is not a global one affecting all aspects of the implementation of fricative voicing. Unlike the deficits in the production of voicing in stop consonants which appear to be the result of a timing disorder (Baum et al., 1990; Blumstein et al., 1980; Hoit-Dalgaard, Murry, & Kopp, 1983; Kent & Rosenbek, 1983; Shewan et al., 1984), impairments in the implementation of voicing in fricative consonants cannot be attributed to timing problems alone. Consistent with this view is the finding that the patients are able to appropriately control the timing of the onset of voicing relative to the fricative noise. That is, most voiced fricatives contained at least 30 ms of glottal excitation during the fricative noise interval. Moreover, they are also able to control appropriately the duration of the fricative noise. The speech production impairment of these patients seems to stem from an inability to produce and sustain sufficiently high levels of glottal excitation during the fricative noise. These results were evident not only from the analysis of the amplitude of glottal excitation but also from the patterns of misclassification of voiced fricatives. In the latter case, the predominant reason for misclassification was an inability to sustain 30 ms of glottal excitation in the fricative noise interval.

The articulatory state that appears to give rise to these acoustic patterns relates to gestures of the larynx. Several articulatory gestures are required to sustain glottal excitation at the amplitude values seen in normal subjects. This includes sufficient adduction of the vocal folds and expansion of the volume of the vocal tract by either lowering the larynx or expanding the pharyngeal volume (Stevens, 1998). Failure to appropriately adduct the vocal folds or to expand the volume of the vocal tract will not only result in a reduction in the amplitude of glottal excitation, but also an inability to sustain glottal excitation.

We have considered the patterns of performance of the aphasic patients as a manifestation of the speech output of anterior (including Broca's) aphasics. Nonetheless, it is the case that there are demonstrated physiological changes to the speech apparatus with aging, and the patients we have studied are older than those studied by Pirello et al. (mean 58 years old for aphasics and 34 years old for normals). Thus, it is worthwhile considering whether the patterns of performance in the production of voicing in fricative consonants could have reflected changes due to the aging process rather than due to the underlying neural pathology. In point of fact, there is little, if any, evidence, showing that the physiological changes accompanying aging have significant effects on the implementation of phonetic/phonological contrasts (see Linville, 2001, for review). Although there are demonstrated changes in speaking rate affecting sentence, syllable, and CV durations, in oral motor precision, and in fundamental frequency, acoustic investigations of the implementation of voicing in stop consonants (measured by voice-onset time) in elderly normal subjects have not shown consistent changes with aging. Similarly, acoustic analyses of vowel formant frequencies have also shown considerable variability across subjects, but no consistent changes in the pattern as a function of age. Of importance for the present investigation is that the measure of glottal excitation in voiced and voiceless fricative consonants used in this study is a relative measure comparing within each subject the amplitude of the first harmonic in the vowel with that of the fricative noise. Thus, even if the absolute amplitude of the first harmonic were affected in these patients, the relative measure should control for this possibility.

In contrast to normal subjects, the aphasic patients did not show any influence of carryover coarticulatory effects. In the case of the aphasic patients, there were long and highly variable pauses between the release of the preceding velar consonant and the onset of frication, in keeping with the nonfluent characteristics of the speech production of anterior aphasics (Kent & McNeil, 1987; McNeil, Liss, Tseng, & Kent, 1990; Ziegler & von Cramon, 1985). This time delay between the completion of one word and the onset of the second word was sufficiently long to neutralize the possibility for mechano-inertial effects to emerge.

A number of studies exploring the neural system underlying speech production and phonation have implicated anterior brain structures including Broca's area (areas 44 and 45) and the premotor and motor regions including the face and mouth areas, the white matter deep to these structures, as well as the basal ganglia and insula (Cummings, 1993; Damasio, 1998). It is of interest that Subject A had the largest and deepest frontal lobe lesion of the three patients, and he was the one who showed the most severe impairment in the implementation of voicing. His fricative utterances had the poorest classification of voicing using the measures of glottal excitation and longer, more variable fricative durations.

In sum, the nonfluent aphasic subjects in this study were able to maintain the phonetic distinction between voiced and voiceless fricatives. The presence of this distinction in nonfluent aphasic speech suggests that the deficits observed in these subjects are not due to the selection of inappropriate speech segments, but rather to lower level impairments in articulatory implementation. This deficit has its origins in laryngeal control. While many studies have found impairments in the timing of laryngeal gestures in nonfluent aphasics, the findings of the current study suggest that the speech output impairments do not always have their origin in the timing of articulatory gestures. Rather, these patients have a more general problem with laryngeal control per se resulting in difficulties in both attaining and sustaining normal amplitudes of glottal excitation.

Finally, the results of this study suggest that the complexity of speech production in phonetic context can reveal an array of impaired articulatory abilities in nonfluent

aphasics that do not appear in the production of isolated samples of speech. The difficulties in production of voiced and voiceless fricatives became most apparent when the target syllables were produced in a larger phonetic context. There have been relatively few quantitative studies reported in the literature that have examined the production of speech in larger phonetic/linguistic contexts. Rather, most studies have analyzed the production of syllables in isolation, i.e., in citation form. The failure of these studies to account for the complexities of context could explain the discrepancy between the severe speech deficits observed clinically in anterior aphasics and the relatively minor impairments described to date by quantitative analyses of anterior aphasic speech. To arrive at a better understanding of the speech production deficits in anterior aphasia, it will be necessary to explore the patterns of deficits that emerge in speech that is produced in the more natural condition of phonetic context.

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References

- Baum, S. (1996). Fricative production in aphasia: Effects of speaking rate. *Brain and Language*, *52*, 328–341, doi:10.1006/brln.1996.0015.
- Baum, S., & Blumstein, S. (1987). Preliminary observations on the use of duration as cue to syllable-initial fricative consonant voicing in English. *Journal of the Acoustical Society of America*, *82*, 1073–1077.
- Baum, S., Blumstein, S., Naeser, M., & 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.
- Bell-Berti, F., Krakow, R., Gelfer, C., & Boyce, S. E. (1995). Anticipatory and carryover implications for models of speech production. In F. Bell-Berti & L. J. Raphael (Eds.), *Producing speech: Contemporary issues* (pp. 77–97). New York: American Institute of Physics.
- Blumstein, S. (1973). *A phonological investigation of aphasic speech*. The Hague: Mouton.
- Blumstein, S. E. (2001). Deficits of speech production and speech perception in aphasia. In R. Berndt (Ed.), *Handbook of neuropsychology* (Vol. 3) (2nd ed.). The Netherlands: Elsevier.
- Blumstein, S., Cooper, W., Goodglass, H., Statlender, S., & Gottlieb, J. (1980). Production deficits in aphasia: A voice-onset time analysis. *Brain and Language*, *9*, 153–170.
- Blumstein, S., Cooper, W., Zurif, E. B., & Caramazza, A. (1977). The perception and production of voice-onset time in aphasia. *Neuropsychologia*, *15*, 371–383.
- Code, C., & Ball, M. (1982). Fricative production in Broca's aphasia: A spectrographic analysis. *Journal of Phonetics*, *10*, 325–331.
- Cooper, W. E., Soares, C., Nicol, J., Michelow, D., & Goloskie, S. (1984). Clausal intonation after unilateral brain damage. *Language and Speech*, *27*, 17–24.
- Crystal, T., & House, A. (1988). Segmental durations in connected-speech signals: Current results; Syllabic stress. *Journal of the Acoustical Society of America*, *83*, 1553–1585.
- Cummings, J. (1993). Frontal-subcortical circuits and human behavior. *Neurological Review*, *50*, 873–880.
- Damasio, H. (1998). Neuroanatomical correlates of the aphasias. In M. T. Sarno (Ed.), *Acquired aphasia* (pp. 43–70). New York: Academic Press.
- Dunlop, J., & Marquardt, T. (1977). Linguistic and articulatory aspects of single word production in apraxia of speech. *Cortex*, *13*, 17–39.
- Freeman, F. J., Sands, E. S., & Harris, K. S. (1978). Temporal coordination of phonation and articulation in a case of verbal apraxia: A voice-onset time analysis. *Brain and Language*, *6*, 106–111.
- Gandour, J., & Dardarananda, R. (1984a). Voice-onset time in aphasia: Thai. II: Production. *Brain and Language*, *18*, 389–410.
- Gandour, J., & Dardarananda, R. (1984b). Prosodic disturbances in aphasia: Vowel length in Thai. *Brain and Language*, *23*, 177–205.
- Gandour, J., Ponglorpisit, S., Khunadorn, F., Dechongkit, S., Boongird, P., Boonklam, R., & Potisuk, S. (1992). Lexical tones in Thai after unilateral brain damage. *Brain and Language*, *43*, 275–307.

- Goodglass, H., & Kaplan, E. (1972). *The assessment of aphasia and related disorders*. Philadelphia: Lea & Febiger.
- Haggard, M. (1978). The devoicing of voiced fricatives. *Journal of Phonetics*, 6, 95–102.
- Harmes, S. R., Daniloff, R., Hoffman, P., Lewis, J., Kramer, M., & Absher, R. (1984). Temporal and articulatory control of fricative articulation by speakers with Broca's aphasia. *Journal of Phonetics*, 12, 367–385.
- Hatfield, F. M., & Walton, K. (1975). Phonological patterns in a case of aphasia. *Language and Speech*, 18, 341–357.
- Hoit-Dalgaard, J., Murry, T., & Kopp, H. (1983). Voice-onset time production and perception in apraxic patients. *Brain and Language*, 20, 329–339.
- Itoh, M., Sasanuma, S., Hirose, H., Yoshioka, H., & Ushijima, T. (1980). Abnormal articulatory dynamics of a patient with apraxia of speech. *Brain and Language*, 11, 66–75.
- Itoh, M., Sasanuma, S., Tatsumi, I., Murakami, S., Fukusako, Y., & Suzuki, T. (1982). Voice-onset time characteristics in apraxia of speech. *Brain and Language*, 17, 193–210.
- Itoh, M., Sasanuma, S., & Ushijima, T. (1979). Velar movements during speech of a patient with apraxia of speech. *Brain and Language*, 7, 227–239.
- Katz, W. (1988). Anticipatory coarticulation in aphasia: Acoustic and perceptual data. *Brain and Language*, 35, 340–368.
- Katz, W., Machetanz, J., Orth, U., & Schonle, P. (1990a). Anticipatory labial coarticulation in the speech of German-speaking anterior aphasic subjects: Acoustic analyses. *Journal of Neurolinguistics*, 5, 295–320.
- Katz, W., Machetanz, J., Orth, U., & Schonle, P. (1990b). A kinematic analysis of anticipatory coarticulation in the speech of anterior aphasic subjects using electromagnetic articulography. *Brain and Language*, 38, 555–575.
- Kent, R., & McNeil, M. (1987). Relative timing of sentence repetition in apraxia of speech and Conduction aphasia. In J. Ryalls (Ed.), *Phonetic approaches to speech production in aphasia and related disorders* (pp. 181–220). San Diego: College-Hill Press.
- Kent, R., & Rosenbek, J. (1983). Acoustic patterns of apraxia of speech. *Journal of Speech and Hearing Research*, 26, 231–249.
- Klich, R. J., Ireland, J. V., & Weidner, W. E. (1979). Articulatory and phonological aspects of consonant substitutions in apraxia of speech. *Cortex*, 15, 451–470.
- Linville, S. E. (2001). *Vocal aging*. San Diego: Singular Thomson Learning.
- McNeil, M. R., Liss, J. M., Tseng, C., & Kent, R. (1990). Effects of speech rate on the absolute and relative timing of apraxic and Conduction aphasic sentence production. *Brain and Language*, 38, 135–158.
- Pirello, K., Blumstein, S., & Kurowski, K. (1997). The characteristics of voicing in syllable-initial fricatives in American English. *Journal of the Acoustical Society of America*, 101, 3754–3765.
- Ryalls, J. (1982). Intonation in Broca's aphasia. *Neuropsychologia*, 20, 355–360.
- Ryalls, J. (1987). Vowel production in aphasia: Towards an account of the consonant–vowel dissociation. In J. Ryalls (Ed.), *Phonetic approaches to speech production in aphasia and related disorders* (pp. 23–43). Boston: College-Hill Press.
- Shewan, C. M., Leeper, H., & Booth, J. (1984). An analysis of voice-onset time (VOT) in aphasia and normal subjects. In J. Rosenbek, M. McNeil, & A. Aronson (Eds.), *Apraxia of speech* (pp. 197–220). San Diego: College-Hill Press.
- Shinn, P., & Blumstein, S. (1983). Phonetic disintegration in aphasia: Acoustic analysis of spectral characteristics for place of articulation. *Brain and Language*, 20, 90–114.
- Stevens, K. N. (1998). *Acoustic phonetics*. Cambridge, MA: MIT Press.
- Stevens, K., Blumstein, S., Glicksman, L., Burton, M., & Kurowski, K. (1992). Acoustic and perceptual characteristics of voicing in fricatives and fricative clusters. *Journal of the Acoustical Society of America*, 91, 2979–3000.
- Sussman, H., Marquardt, T., MacNeilage, P., & Hutchison, J. (1988). Anticipatory coarticulation in aphasia: Methodological considerations. *Brain and Language*, 35, 369–379.
- Trost, J. E., & Canter, G. J. (1974). Apraxia of speech in patients with Broca's aphasia: A study of phoneme production, accuracy, and error patterns. *Brain and Language*, 1, 63–79.
- Tuller, B., & Story, R. (1988). Anticipatory and carryover coarticulation in aphasia: An acoustic study. *Cognitive Neuropsychology*, 5, 747–771.
- Ziegler, W., & von Cramon, D. (1985). Anticipatory coarticulation in a patient with apraxia of speech. *Brain and Language*, 26, 117–130.