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# The Foreign Accent Syndrome: A Reconsideration

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This study compared the post-CVA speech of a patient presenting with the foreign accent syndrome (FAS) to both a premorbid baseline for that patient and to similarly analyzed data from an earlier reported case of FAS. The object of this research was to provide quantitative acoustic data to determine whether: (1) the constellation of phonetic features associated with FAS is the same across patients and (2) a common neural mechanism underlies FAS. Acoustic parameters investigated included features of consonant production (voicing, place and manner of articulation), vowel production (formant frequency and duration), and prosody. Results supported the characterization of FAS patients as having a ''generic'' foreign accent and the hypothesis that FAS deficits are qualitatively different from that of Broca's aphasia. However, comparison of this case with recent studies revealed the extent to which the constellation of phonetic features may vary among FAS patients, challenging the notion that a general prosodic disturbance is the sole underlying mechanism in FAS. 

## INTRODUCTION

The foreign accent syndrome (FAS) is a rare speech disorder characterized by the emergence of a ''perceived'' foreign accent in speech following left hemisphere brain damage. The syndrome has also been termed ''pseudo-

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accent'' (Lecours, Lhermitte, & Bryans, 1983) and ''unlearned foreign accent'' (Graff-Radford, Cooper, Colsher, & Damasio, 1986). To date, fewer than 15 cases have been reported in the literature, beginning with Pick's Czech patient first identified in 1919 (although cf. Aronson, 1980).

As only a few cases of FAS have undergone detailed lesion analyses, present knowledge of the neural basis of FAS is limited. Most cases of FAS have been due to stroke, but a small number have occurred after trauma (Monrad-Krohn, 1947; Nielson & McKeown, 1961; Aronson, 1980; Moonis, Swearer, Blumstein, Kurowski, Licho, Kramer, Mitchell, & Drachman, 1993). All cases with published lesion information have had left hemisphere damage. Lesion size has been small, typically involving a single gyrus or a greatest extent of less than 3 cm. Localization has not been uniform, but all cases have involved either prerolandic motor cortex (Brodmann's area 4), frontal motor association cortex (Brodmann's areas 6 or 44), or striatum. Mild facial and bulbar weakness has only been observed in some of the cases and voice volume has usually been normal. These findings suggest that there is no simple motor basis for FAS.

Neuropsychological and speech pathology evaluations in this small number of patients have also not identified any specific attributes or correlations with FAS. The patients have had normal melody in song or affect (when evaluated), dissociating FAS from the described right hemisphere aprosodias (Ross & Mesulam, 1979; Ross, 1981). They have had no facial apraxia. While some patients have had impairments in articulation, few patients had an articulation deficit as the presenting feature (Whitty, 1964; Schiff, Alexander, Naeser, & Galaburda, 1983). These observations clearly dissociate FAS from a straightforward impairment in left hemisphere speech processes. Most patients were transiently mute, but FAS became apparent soon after speech returned. Its prompt appearance suggests that FAS does not emerge as some unusual form of compensation for or adaptation to a voice or speech problem. Rather, it is a direct manifestation of damage to underlying brain mechanisms involved in speech production. At the time of the reported investigations, only about 50% of the patients were aphasic by standard clinical measures. The aphasia profiles have been clinically similar (hesitancies and delays in speech initiation and mild agrammatic qualities) but labeled differently, usually as transcortical motor aphasia or mild Broca's aphasia. FAS is, of course, not a typical feature of any aphasia type.

Precise identification of the perceived foreign accent in these cases has proven to be illusory. While some studies described the sudden acquisition of one specific foreign accent (Pick, 1919; Monrad-Krohn, 1947; Whitaker, 1982; Graff-Radford et al., 1986; Ardila, Rosselli, & Ardila, 1988), many researchers have noted a variety of responses to the question of what foreign accent was present in their patient's speech. For example, Blumstein, Alexander, Ryalls, Katz, and Dworetsky (1987) found that their American patient's accent was perceived as Eastern European, French, Dutch, and Scandinavian. Similarly, in a study by Ingram, McCormack, and Kennedy (1992), an Australian patient was variously judged as having an Asian, Swedish, or German accent.

It has been suggested that FAS does not reflect any particular foreign accent but rather is characterized by a ''generic foreign accent'' (Blumstein et al., 1987, p. 243; cf. also Gurd, Bessel, Bladon, & Bamford, 1988; Ingram et al., 1992). Detailed acoustic analyses of the speech production of a single FAS patient by Blumstein et al. (1987) revealed that the anomalous features reflected patterns of speech found in the regular sound structures of human language, although not necessarily of the patient's native language. None of these speech features were pathological in the sense that they violated the phonetic rules of language, as is the case for Broca's aphasics or patients with other speech production problems. For example, while the large Fo excursions at the end of phrases reported by Blumstein et al. in their patient's data do not occur in English, they occur in other languages such as French, and, while their patient made voicing errors, the production of VOT reflected normal English patterns. Likewise, a failure to observe the speech production rule in English which reduces medial [t, d] to flaps after a stressed vowel that had been reported by both Blumstein et al. (1987) and Ingram et al. (1992) must be viewed in light of the fact that other languages do retain full stops in such vowel contexts. Thus, although listeners perceive the speech patterns of FAS patients as nonnative, these speech patterns nonetheless reflect phonetic properties which occur in the inventory of sounds found in the languages of the world. As a consequence, listeners categorize these deviations as stereotypically ''foreign'' (cf. Blumstein et al., 1987; Gurd et al., 1988; and Ingram et al., 1992 for discussion).

Unfortunately, until recently, the investigation of FAS has been limited to largely descriptive approaches to the speech patterns of the patients, and, as a result, it is often difficult to compare across studies and to quantitatively assess the constellation of phonetic patterns that characterize the foreign accent syndrome. For example, every reported case of FAS included some mention of a dysprosody affecting either the word or the sentential level (or both). Even Gurd et al.'s report (1988, p. 243) of an exception to this potential common denominator of FAS listed at least one instance of ''bizarre intonation'' as well as the intermittent occurrence of a long pausing time in their patient's speech. However, in most of these studies, precise quantitative and acoustic analyses of the actual patterns of prosody were not conducted, and the impairments were described only as ''non-English'' (Van Lancker, Bogen, & Canter, 1983) or ''disrupted'' (Schiff et al., 1983). Likewise, on the segmental level, while deviant vowel productions were noted in many cases, only some studies provided quantitative analysis of these utterances.

To date, the available perceptual and acoustic data show that FAS speech is characterized by both prosodic and segmental deficits. With respect to prosody, FAS patients have demonstrated a number of anomalies involving stress, rhythm, and intonation. On the word and phrasal level, dysprosodies resulted from the failure to reduce unstressed syllables within words, lapses into syllable-timed instead of stress-timed speech (or vice versa in languages such as Japanese), resyllabification due to epenthetic vowel insertion, and poor transitions across word boundaries (Monrad-Krohn, 1947; Whitaker, 1982; Graff-Radford et al., 1986; Blumstein et al., 1987; Gurd et al., 1988; Ardila et al., 1988; Ingram et al., 1992; Takayama, Sugishita, Kido, Ogawa, & Akiguchi, 1993). There have been reports of FAS patients with sing-song phrasal intonation (Nielson & McKeown, 1961; Critchley, 1970) and with a staccato speech rhythm (Critchley, 1970). On the sentential level, a range of ‘‘foreign-sounding’’ intonation contours have been reported, including a sharply rising pitch at the end of sentences (Monrad-Krohn, 1947; Blumstein et al., 1987; Moonis et al., 1993), excessively steep terminal falls (Ingram et al., 1992) and even inverted pitch contours (Takayama et al., 1993). There are also reports of reduced fundamental frequency ranges (Graff-Radford et al., 1986) as well as a more general reduction of prosodic contrasts.

On the segmental level, there appear to be more vowel than consonantal anomalies (Gurd et al., 1988, being a notable exception). These vowel production anomalies have shown a variety of patterns, including vowel tensing (Van Lancker et al., 1983; Whitaker, 1982; Blumstein et al., 1987; Ingram et al., 1992), vowel lengthening (Van Lancker et al., 1983; Graff-Radford et al., 1986; Ardila et al., 1988), schwa coloring (Whitaker, 1982; Gurd et al., 1988), as well as vowel shortening (Pick, 1919). Nevertheless, the Blumstein et al. (1987) acoustic data showed normal vowel durations, although there was an overall greater variability in vowel formants. Other acoustic analyses (Moonis et al., 1993; Ingram et al., 1992) further showed an overall reduction in the acoustic vowel space, due to a restricted F1 range.

With respect to consonant production, the FAS data show a range of abnormalities, including manner changes (Nielson & McKeown, 1961; Ardila et al., 1988; Ingram et al., 1992), voicing errors (Blumstein et al., 1987; Ardila et al., 1988; Gurd et al., 1988), and place of articulation changes (Whitaker, 1982; Ardila et al., 1988). Only Gurd et al. (1988) reported undershoot of consonant targets, a deficit more commonly seen in the weakened consonantal gestures of dysarthrics. Ingram et al. (1992) suggest that many of these consonantal deficits point to a reduction in normal lenition processes.

On the basis of the range of prosodic and segmental anomalies described above, it is difficult to determine what constellations of phonetic features typically characterize the foreign accent syndrome. It is not clear, for example, whether certain pathological features of speech production *always* accompany FAS, such as increased Fo range and vowel tensing, or whether more general properties of speech such as prosody and vowel production are affected but potentially in different ways across patients. Moreover, because only a few acoustic analyses have been conducted, it is not known whether the speech production anomalies in FAS remain a part of the ‘‘universal’’ properties of speech as first proposed by Blumstein et al. (1987) rather than representing a mild form of dysarthria or speech production impairment. Until a clear-cut delineation of the speech patterns of FAS can be determined, it is difficult to ascertain the underlying mechanisms responsible for FAS. For example, Blumstein et al. (1987) proposed that the constellation of features in FAS reflect a common underlying deficit relating to speech prosody. The question remains whether the different patterns described clinically all fit within this common framework.

To address these questions, we have conducted a detailed acoustic analysis of the speech production of a patient diagnosed with the foreign accent syndrome. Similar analyses to that of Blumstein et al. (1987) were conducted so that direct comparisons could be made between the speech patterns of these two patients. Moreover, our patient provides a unique opportunity to study the acoustic–phonetic changes in the speech patterns of FAS since a premorbid speech sample was available for this patient. In this case, the FAS patient serves as his own control, and any changes in the acoustic patterns of his speech before and after the emergence of his ‘‘foreign accent’’ can be charted.

## CASE REPORT

The patient is a 45-year-old right-handed male who had a left middle cerebral artery stroke following 6 months of intermittent numbness in his right arm. His past medical history was unremarkable. At the time of the stroke, the patient developed right upper extremity plegia and right lower extremity paresis and was described as globally aphasic. Carotid arteriography showed no occlusive disease but possible granulomatous angiitis. A left frontal brain biopsy was nondiagnostic but the patient was treated with cytoxan and prednisone. Subsequent review of angiograms, MRIs and biopsy histology at another medical center concluded that there was no vasculitis. An unexplained infarct of unknown etiology was diagnosed. There was rapid recovery of motor function and significant improvement in speech and language. Four months later, a neurology evaluation described mild Broca’s aphasia. The patient was able to return to work as a research biochemist.

At 2 years postonset, the patient was evaluated for the first time at the Boston Veterans Administration Medical Center. He presented with fluent but generally slow speech that had a somewhat dysarthric quality. Most noticeable in his speech was the presence of a foreign accent which was variously described by listeners as British, Scottish, Irish, or (to at least one observer) Eastern European. A review of the patient’s personal history revealed that he was born in Chicago and grew up in Brooklyn in an English-speaking household. As a student, he had fulfilled various language require-

TABLE 1
BDAE Summary

| BDAE subtests | Scoring range | Patient's score |
|---|---|---|
| Fluency | | |
| Articulation rating | 1–7 | 4.5; also 6.0 (variable) |
| Phrase length | 1–7 | 7.0 |
| Melodic line | 1–7 | 3.5; also 7.0 (variable) |
| Grammatical form | 1–7 | 7.0 |
| Auditory discrimination | | |
| Word discrimination | 0–72 | 72 |
| Commands | 0–15 | 15 |
| Complex material | 0–12 | 11 |
| Repetition | | |
| High probability | 0–8 | 8 |
| Low probability | 0–8 | 8 |
| Writing | | |
| Written naming | 0–10 | 10 |
| Sentences to dictation | 0–12 | 11 |
| Naming | | |
| Confrontation | 0–114 | 112 |

ments by studying Spanish and French. After receiving his Ph.D., he did postdoctoral work in biophysics. His foreign travels were limited to a total of approximately 10 weeks in Europe. Hence, his foreign accent may be termed as essentially ''unlearned.'' In addition to his speech problems, the patient also complained of difficulties at work with writing, understanding complex material at seminars, and performing mathematical calculations.

Neurological findings at this time included a mild right central facial paresis and a very mild right upper extremity paresis. Sensory examination was remarkable for mild diminution of vibratory sensation in the lower extremities.

The neuropsychological assessment revealed some frontal lobe impairment, as characterized by difficulty with the Luria 3-Step, set difficulty, and mild impulsivity on sequence copying. His performance IQ was 115. Verbal and visual memory as well as drawing were generally very good. However, digit span was impaired, as well as mental tracking for spelling backwards. No left-right confusion or finger agnosia were noted. Calculations of moderate complexity were significantly slow but intact. His performance on interpretation of proverbs was variable, occasionally being rated as somewhat concrete. The patient demonstrated some anxiety and tearfulness concerning his present speech difficulties, as well as his general post-CVA situation.

Formal language assessment at 2 years postonset indicated that the patient had some residual problems, most notably with articulatory agility. A summary of the results of his Boston Diagnostic Aphasia Exam is provided in Table 1. Although repetition scores showed no deficit, two of the patient's

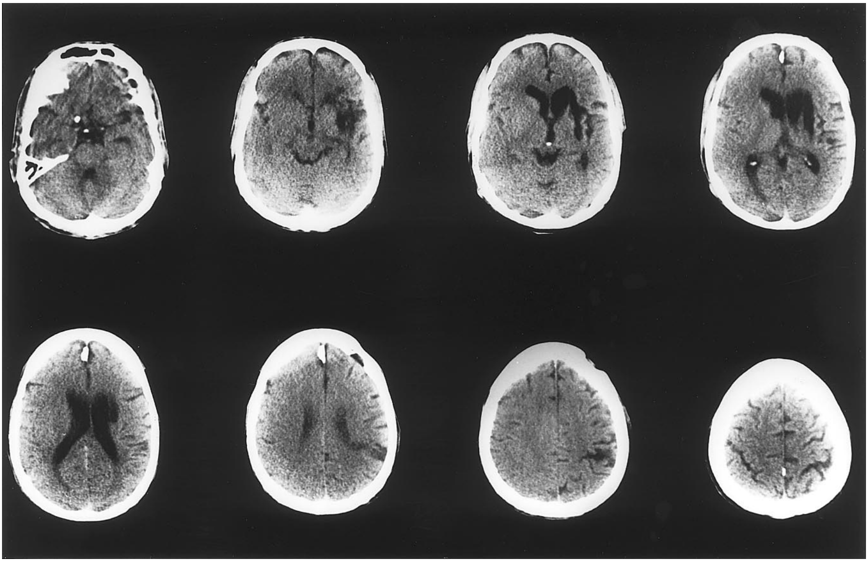


FIG. 1. CT scan from 2 years postonset reveals an area of infarction in the left lenticulostriate territory involving putamen, dorsolateral caudate, the lateral anterior limb of the internal capsule, and anterior periventricular white matter. There is a separate small lesion in the mid parietal lobe. These left-hemisphere lesions are on the right side of the CT scan.

fluency ratings (for spontaneous speech) were scored as variable, indicating some limitations in the area of articulatory agility and melodic line. Generally, his speech output was slow, often halting, but with normal volume. However, on the basis of his phrase length rating and the degree of syntactic complexity present in his sentences, he was described as generally fluent. He exhibited only minimal word-finding problems. There was no evidence from the BDAE testing of agrammatism, paraphasias, or reading problems, although infrequent literal paraphasias were noted in his spontaneous speech, along with some self-correction. Auditory comprehension was very good, with only one error on complex ideational material. While his ability to carry a familiar tune was rated as normal, his foreign accent was still evident in his singing. No bucco-facial or limb apraxia was demonstrated.

Regarding lesion localization, CT and MRI performed 2 years postonset showed very similar lesions. There was a small area of infarction in the left posterior supramarginal gyrus and a more extensive lesion in left subcortical structures (Fig. 1). The subcortical lesion involved the lateral putamen, lateral portions of the anterior limb of the internal capsule, and the frontal periventricular white matter, anterior and superior to the head of the caudate nucleus.

## DESCRIPTIVE CHARACTERISTICS OF SPEECH

As noted earlier, listeners variously described this patient's foreign accent as being British, Scottish, Irish, or Eastern European. Despite the lack of agreement as to the specific type of accent, these listeners generally cited the same characteristics in the patient's speech to support their overall perception of a foreign accent. One of the most frequently noted nonnative characteristics involved vowel quality. Tense vowel targets were often perceived as lax (i.e., [u] was heard as [U]). There was also sporadic substitution of a centralized vowel segment for every vowel type except the high vowels [i] and [u]. Anomalous consonant productions were also cited in some listeners' descriptions of the nonnative quality of this patient's speech. For instance, the liquids [l r] were perceived as non-English or, at the very least, as oddly articulated. Voiceless stop consonants were described as being too heavily aspirated at times. Voiced stop consonants, on the other hand, were often devoiced in word-final position. Prosody, however, appeared to be normal.

Taken together, these descriptive characteristics of the patient's spontaneous speech show some similarities to previous reports, particularly with respect to consonant and vowel production. However, they appear to provide a somewhat different constellation of speech deficits (and potentially a different underlying mechanism for FAS) than that described in Blumstein et al. (1987) and the majority of reported FAS cases. In nearly all of the earlier reported cases, prosodic impairments on the word and/or sentence level figured prominently as a characteristic of the speech pattern for FAS.

## ACOUSTIC ANALYSIS OF STOP CONSONANTS

Similar to Blumstein et al. (1987), the production of stop consonants was analyzed to explore the production of voicing, place of articulation, and manner of articulation. To this end, two acoustic analyses were conducted including voice-onset time, exploring the production of initial voicing in [p t k b d g], and spectral (LPC) analyses of initial [t d]. A third analysis investigated the production of alveolar stops produced in medial position after a stressed vowel (i.e. [t] in ''butter'').

### *Stimuli and Procedures*

The stimuli for the VOT and place of articulation analyses consisted of the 30 monosyllabic real words used in the Blumstein et al. (1987) study. Each word contained the initial stops [p t k b d g] followed by the vowel [a] and either one or two consonants. The stimuli are listed in Table 2. The words were printed on 3 × 5 cards. On each presentation, the patient was asked to read the word twice, using the carrier phrase ''this __'' each time. The carrier word ''this'' was used as in earlier production studies with Broca's aphasics (Blumstein, Cooper, Goodglass, Statlender, & Gottlieb, 1980) and in our earlier FAS study to provide an utterance other than the test word to begin the production in the event that the patient had difficulty initiating speech. These studies showed that the production of a preceding [s] did not affect either the VOT or aspiration typically shown for initial voiceless stop

TABLE 2
Stimuli Used in the Analysis of Voicing and Place of Articulation in Stop Consonants

| | | |
|---|---|---|
| Pond | Tart | Cop |
| Pot | Top | Car |
| Palm | Tot | Cod |
| Pop | Tar | Con |
| Part | Tom | Calm |
| Barn | Doll | God |
| Bog | Dark | Got |
| Bar | Dot | Gone |
| Bomb | Dock | Guard |
| Bob | Don | Gosh |

consonants. There were two presentations of the entire list of words, providing four tokens of each test stimulus.

The analysis of full versus flapped stop consonants in medial position after a stressed vowel was based on the following words: *bottle, butter, dotty, rider, writer,* and *relative.* Each word was read in a list three times.

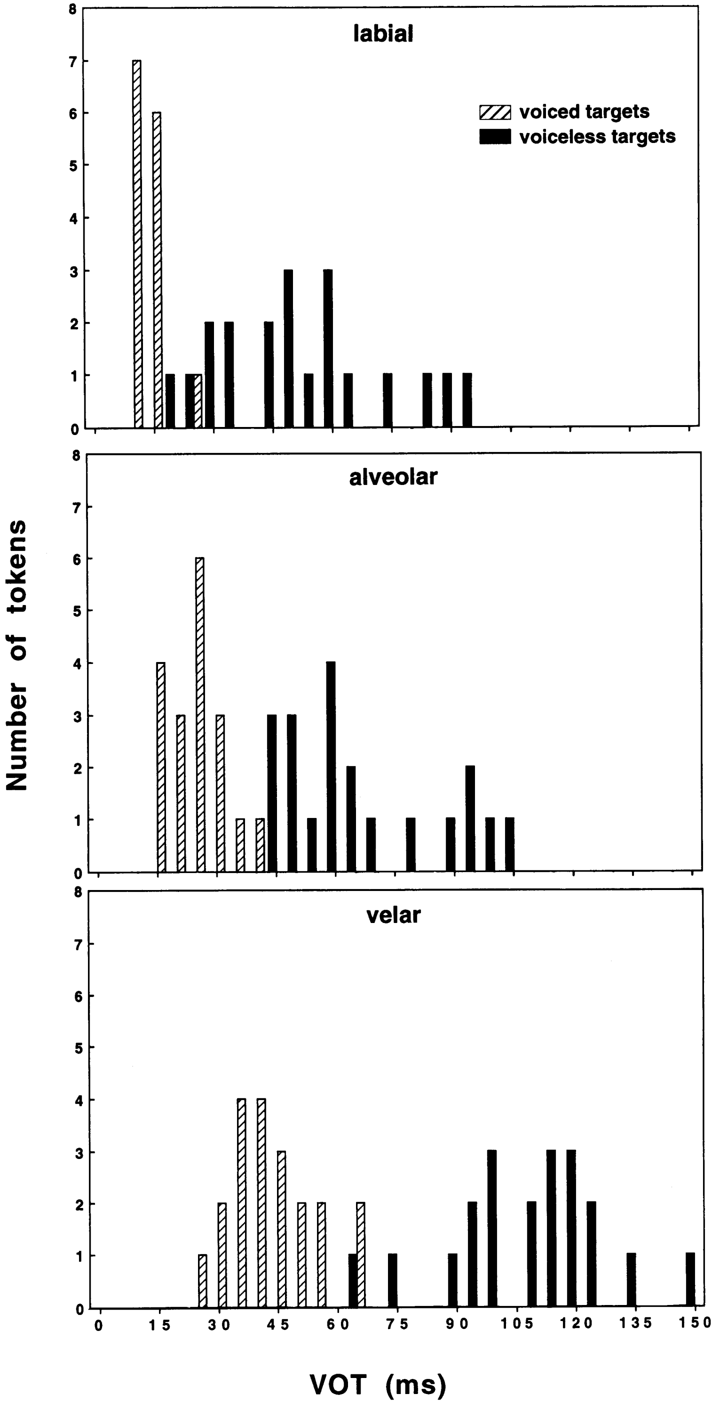
All of the stimuli were tape-recorded and then transferred to a Micro-Vax II computer at the Brown University Phonetics Laboratory, using a 10-kHz sampling rate with a 4.5-kHz low-pass filter setting and a 10-bit quantization.

## *Analysis of Results and Discussion*

*Voice onset time.* Blumstein et al. (1987) showed that the production of VOT in their FAS patient was similar to normals. In the current study, VOT was measured on the waveform display from the burst release of the stop closure to the onset of voicing. In the case of prevoiced stops, the duration measure was to be taken from the onset of voicing until the onset of the burst release, yielding a negative VOT value. No prevoiced stops were actually produced by our patient. The frequency distribution of the VOT responses was plotted for each place of articulation and is displayed in Fig. 2.

As Fig. 2 shows, our patient maintains the voiced/voiceless distinction for each place of articulation. Overlap in VOT occurred between voiced and voiceless consonants for only one labial token and two velar tokens. The distribution of VOT productions clearly patterns like that of an American English speaker in two ways. First, voiceless stop consonants are characterized by long-lag VOTs, and voiced stop consonants are characterized by short-lag VOTs. Second, the VOT values increase slightly as place of articulation changes from front to back.

The only aspect of our patient's VOT productions that was abnormal was the occurrence of an epenthetic vowel produced immediately preceding some

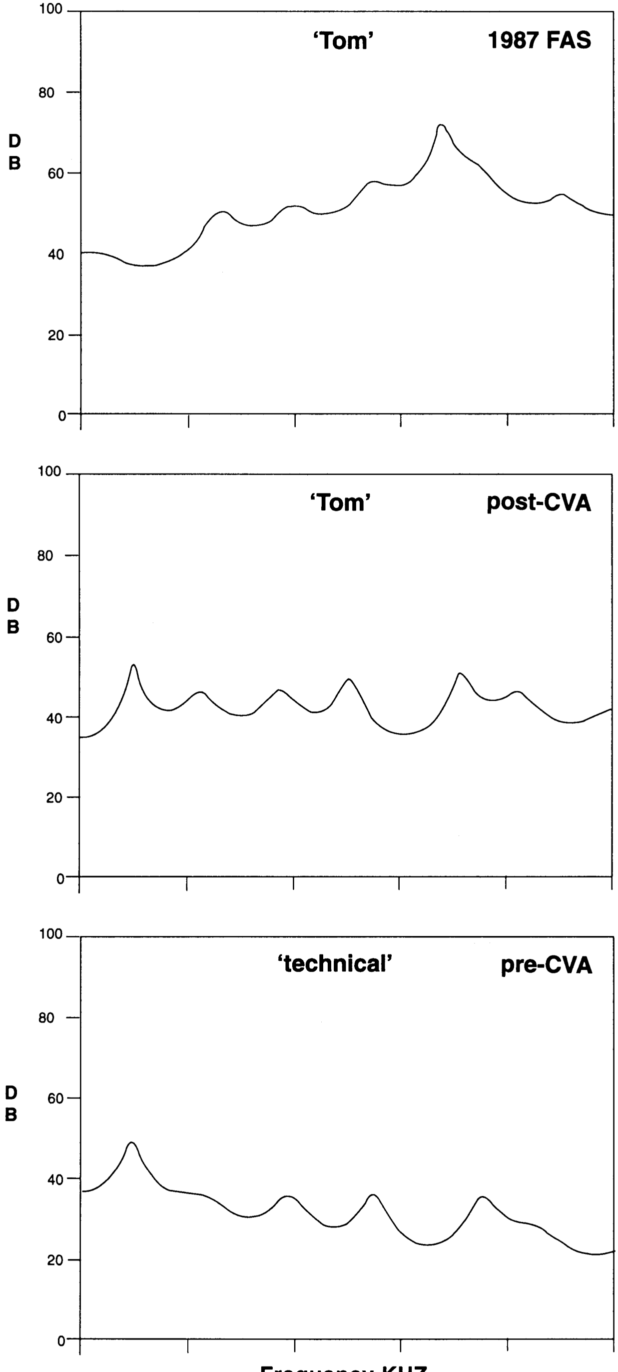

labial
voiced targets
voiceless targets
alveolar
velar
Number of tokens
0
1
2
3
4
5
6
7
8
0 15 30 45 60 75 90 105 120 135 150
VOT (ms)

voiced stops. In all, about 13% or 8 of 60 voiced stop tokens were preceded by an epenthetic vowel, as evidenced on LPC's by well-defined formant peaks throughout the spectrum. By contrast, the 1987 FAS patient had 33% of her voiced stops preceded by an epenthetic vowel. The significance of the occurrence of a vowel-like syllable prior to the release of the voiced stop consonants in one-syllable stimulus words is that it changes the syllable structure of the stimuli and consequently affects the ''rhythm'' or prosody of the entire test phrase.

*Place of articulation.* The same set of stop consonants used in the VOT study was further analyzed acoustically with respect to place of articulation. Earlier research with normals (Stevens & Blumstein, 1978; Blumstein & Stevens, 1979; Lahiri, Gewirth, & Blumstein, 1984) has shown that spectral properties at the moment of consonantal release distinguish labial, dental, alveolar, and velar places of articulation. Blumstein et al. (1987) showed that their FAS patient's stop consonants were produced as alveolars (diffuse-rising spectra) not dentals (fairly flat spectra), thus showing a pattern typical of an English speaker.

Employing the same analysis techniques, LPC spectra were obtained using a 25.6-msec half-Hamming window (with preemphasis) placed at the onset of the burst for each stop consonant token. Results showed that the spectral shapes of labial and velar productions were consistent with those found in normal English speakers. Moreover, the labial and velar spectral patterns were distinct from that of [t d]. The same labial and velar spectral patterns emerged in the pre-CVA (spontaneous speech) data as well. Results for the production of [t d], however, revealed a pattern distinct from the production of alveolar stop consonants in English. In particular, rather than a diffuse–rising spectral shape, the spectral shape for the production of [t d] was diffuse–flat. This latter pattern is more in keeping with the production of dental stops, suggesting that our patient's production of [t d] was ''foreign.'' Nevertheless, analysis of a number of [t d] tokens in initial position produced by the patient prior to his stroke suggests that even premorbidly, his production of [t d] was more dental than it was alveolar. Figure 3 compares the spectral shape for [t d] produced by the 1987 FAS patient with those of our patient both before and after his stroke. As the figure shows, the production of [t d] for the 1987 FAS patient is diffuse–rising (typical of English speakers), whereas the spectral pattern both premorbidly and poststroke for our patient is diffuse–flat. Taken together, these results suggest that the pre- and poststroke spectral patterns for place of articulation are similar, and, as a result, do not contribute to the emergence of the FAS in our patient.

FIG. 2. Frequency distribution of voice-onset time (VOT) responses for labial, alveolar, and velar stop consonants. The solid bars represent voiceless targets; the striped bars represent voiced targets.



'Tom'
1987 FAS
D
B
100
80
60
40
20
0
'Tom'
post-CVA
D
B
100
80
60
40
20
0
'technical'
pre-CVA
D
B
100
80
60
40
20
0
Frequency KHZ

TABLE 3
Percentage of Tokens Perceived as Full Stops

| Stimulus | Full stop responses (%) |
|---|---|
| Bo**tt**le | 67 |
| Bu**tt**er | 89 |
| Do**tt**y | 67 |
| Ri**d**er | 67 |
| Wri**t**er | 94 |
| Rela**t**ive | 100 |

*Manner of articulation.* When medial [t d] occur after a stressed vowel in English, these stop consonants are produced as flaps. This distinction may be quantified in terms of the duration of the closure and release interval, with full stops having longer average durations than flaps (Zue & Lafferiere, 1979). In the 1987 study, the FAS patient had failed to phonetically realize medial [t d] as flaps after stressed vowels, producing perceptually and acoustically full stops instead with durations of 107 msec ([t]) and 55 msec ([d]).

In the present study, we were unable to take similar durational measurements. Background noise on this portion of the tape made locating the endpoints of the closure–release interval impossible to ascertain. Instead, the patient's productions were presented to six phonetically trained listeners who were asked to label the 18 [t d] tokens (6 stimuli × 3 repetitions) as either flaps or full stops. Table 3 summarizes these results. In general, 77% of all stimuli with flaps as targets were perceived as full stops, with a range of 67 to 94%. The tokens for ''relative'' were judged 100% as containing full stops. This latter stimulus is the only stimulus item where the flapping rule should not apply. Thus, similar to the 1987 FAS patient, our patient failed to observe this context-sensitive flapping rule in English.

## ACOUSTIC ANALYSIS OF VOWELS

Vowel quality had been the most frequently cited parameter contributing to the perception of a foreign accent in our patient's speech. In particular, tense vowels were perceived as lax and several vowel types were sporadically replaced with what was perceived as a centralized mid–low vowel. These aspects of our FAS patient's speech differed from that of the 1987 patient, whose vowels had been described as more tense and less diphthong-

FIG. 3. LPC spectra for [t]-initial utterances, using a 25.6-msec half-Hamming window placed at the burst release. (Top) ''Tom'' is an exemplar (citation form) from the 1987 study. (Middle) ''Tom'' is from our patient's post-CVA speech (citation form); (Bottom) ''technical'' is from his pre-CVA (spontaneous) speech.

ized. Another question to be addressed in the present study concerned the degree of variability in this patient's vowel durations. Comparisons of the standard deviations for F1, F2, and duration measures of vowels produced by the 1987 patient with those of anterior and posterior aphasics (Ryalls, 1984) had shown a similar high degree of variability. To characterize acoustically and compare our patient's vowel productions to the 1987 data as well as to normal and aphasic baselines, acoustic analyses of vowel duration and formant frequencies were conducted.

*Stimuli and Procedures*

The stimuli for these analyses came from two sources: citation form and both pre- and post-CVA spontaneous speech. The words in citation form were from the following list: *heed, hid, hade, head, had, who'd, hood, hawed, hod, howed.* These words were printed on $3 \times 5$ index cards and presented to the patient for reading. The entire list was repeated five times. One vowel token that was not a good exemplar of the target phonemes was eliminated from the citation speech sample. The post-CVA spontaneous speech sample consisted of a conversation between the patient and a member of the research team. In that conversation, the patient was asked a number of questions concerning his family, job, and post-CVA activities. The pre-CVA spontaneous speech sample was extracted from a videotape of a speech given by the patient at a family celebration.

To try to match the stressed vowel targets obtained in the monosyllabic [hVd] citation sample, only vowel phonemes with primary stress were extracted from the spontaneous speech samples. The only exception was the inclusion of two [U] productions obtained from the words ''childhood'' and ''manhood'' in the pre-CVA sample. These stimuli were included because of the paucity of [U] segments produced by the patient in the sample. Both types of stimuli (i.e., [hVd], rapid speech) were subsequently digitized for computer analysis using a 20-kHz sampling rate with a 9-kHz low-pass filter setting and a 10-bit quantization.

*Duration Analysis*

As in the 1987 FAS study, vowel duration was measured from the beginning of periodicity in the waveform up to changes at the end of the vowel steady state associated with the following [d] closure. These same duration measures were performed on our patient's [hVd] series as well as on his rapid pre- and post-CVA speech tokens. Table 4 shows the averaged duration values for each of the vowel targets presented according to the stimulus source. In addition, duration values for the [hVd] series from the 1987 study are listed for comparison.

Comparing first the pre- and post-CVA data from our patient's rapid speech samples, the post-CVA vowel durations are similar to the pre-CVA

TABLE 4
Average Vowel Durations (msec) for Two FAS Patients
(Current Study and Blumstein et al., 1987)

| | [i] | [I] | [e] | [ɛ] | [æ] | [u] | [U] | [o] | [ɔ] | [a] | [ʌ] |
|---|---|---|---|---|---|---|---|---|---|---|---|
| Rapid speech: | | | | | | | | | | | |
| Current (pre-) | 130 | 72 | 152 | 93 | 122 | 98 | 82 | 128 | 99 | 130 | 82 |
| Current (post-) | 123 | 100 | 124 | 91 | 117 | 105 | 99 | 124 | 170 | 138 | 93 |
| [hVd] | | | | | | | | | | | |
| Current (post-) | 273 | 248 | 293 | 250 | 309 | 279 | 242 | — | 327 | 329 | — |
| 1987 (post-) | 178 | 139 | — | 127 | 239 | 257 | 141 | — | — | 236 | 179 |

data set. Moreover, each set (pre-, post-) maintains intrinsic vowel durations that distinguish between the tense and lax members of those vowel pairs in English (i.e., [i e u] are longer than [I ɛ U], respectively).

Turning to the patient's [hVd] series, although all of these vowel durations are at least twice as long as their rapid speech counterparts, intrinsic vowel durations are still maintained. It is not surprising to find vowel durations to be longer in citation form than in rapid speech. Nevertheless, it is worth noting that the overall durations of the vowels in the [hVd] condition are considerably longer in our FAS patient than in the Blumstein et al. patient. Still, it is significant that both patients maintain differences in intrinsic vowel duration.

What was not in evidence in our patient's speech that did emerge frequently in the 1987 patient was the introduction of a reduced centralized schwa following the word-final ([d]) in the [hVd] series. This additional vowel not only changed the syllable structure from CVC to CVCV but also that patient's rhythm of speech.

*Formant Frequency Analysis*

The formant frequencies of three different sets of stimuli were acoustically analyzed to determine the extent to which our FAS patient's vowel productions deviated from a normal baseline. The tokens for analysis were taken from both the pre- and the post-CVA rapid (or spontaneous) speech samples as well as from the patient's [hVd] series. Normal baselines were provided by the patient's pre-CVA speech and normative data for adult males from Peterson and Barney (1952).

LPC analyses were conducted to determine F1 and F2 values at the vowel midpoint, using 25.6-msec full-Hamming window. The data set consisted of all vowel targets which were perceptually correct as well as 10 additional tokens which were perceived as ''more lax than tense.'' To determine whether these latter tokens were acoustically distinct from their appropriate targets, the F1/F2 values were compared with the F1/F2 values for the per-

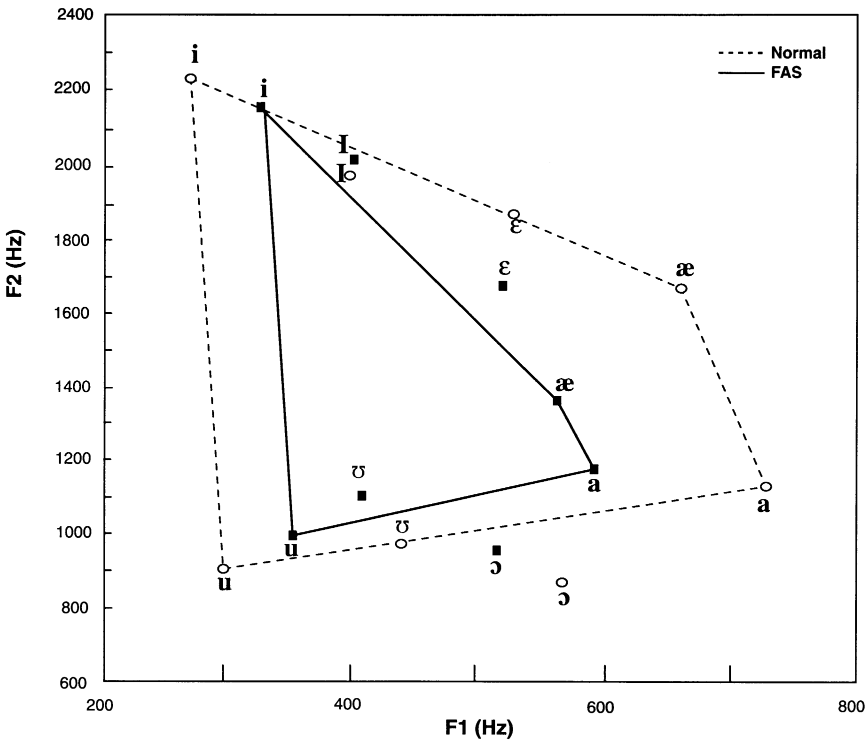



FIG. 4. Formant frequency plots (F1, F2) for vowel stimuli (citation form) from this FAS patient (solid lines) compared to average values for normal male speakers (dashed lines) taken from Peterson and Barney (1952).

ceptually correct targets. Interestingly, acoustic analyses showed that these tense vowel targets had one but not both of their formant values fall within the expected formant frequency range of the lax vowel. Moreover, the average difference between the perceptually ambiguous and correct targets was no more than 34 Hz for F1 and 87 Hz for F2. Because the formant frequency values were so similar, these productions were included in the data set. Any other vowel productions that differed from their target were labeled as phonemic errors and were excluded from the data analysis.

Figure 4 compares the vowel space of [hVd] tokens produced by our FAS patient with that of normative data from Peterson and Barney (1952). Although the shape of the vowel space is similar, there is a centralizing tendency that affects every vowel except [I]. Thus, the low F1 in the high vowels [i u] is raised in our patient, and is lowered in the remaining vowels. Similarly, F2 is raised in the back vowels and lowered in the front vowels. Taken together, our patient's vowel space is both centralized and constrained.

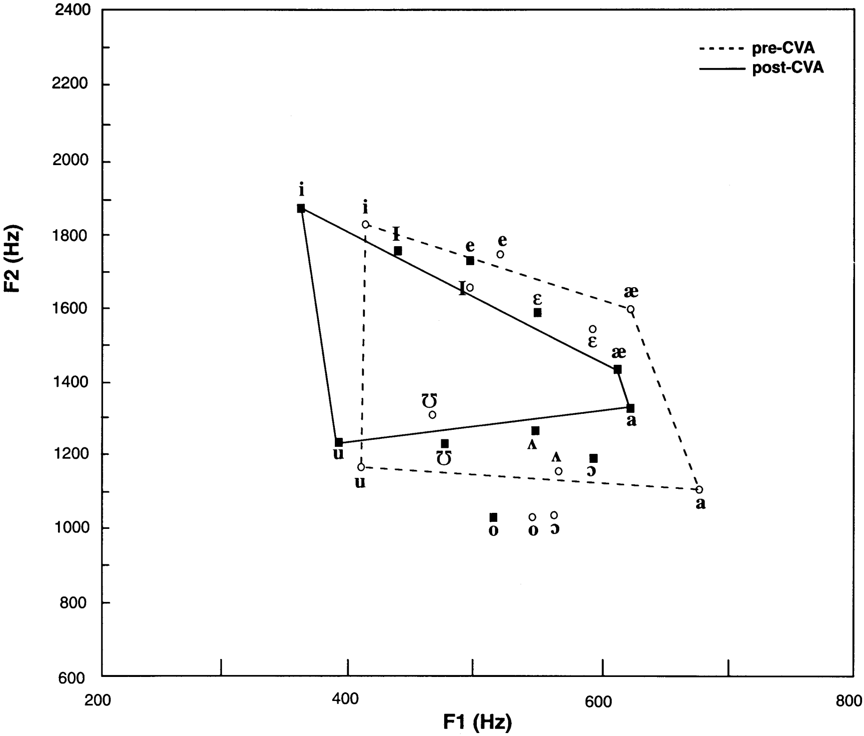



FIG. 5. Formant frequency plots (F1, F2) for vowel stimuli from this patient's rapid speech, comparing pre-CVA (dashed lines) and post-CVA (solid lines) speech production.

Figure 5 which compares the production of vowels produced in spontaneous speech by our patient before and after his stroke also shows this centralizing tendency. With the exception of the point vowels [i] and [u], vowels tend to be produced in a more centralized vowel space subsequent to the stroke. Overall, F1 values are lower and F2 values in the back vowels are higher. Interestingly, the vowel space of normals tends to be more constricted in rapid speech compared to citation form. A comparison of Figs. 4 and 5 shows this to be the case in our patient as well, and also indicates that for him there is an increased centralizing tendency from citation form to spontaneous speech over and above what is typically found in normal subjects.

Variability of formant frequency values and duration was also compared. In the 1987 Blumstein et al. study, the FAS patient's standard deviations for F1, F2, and overall vowel durations were comparable to or even higher than that of anterior and posterior aphasics (Ryalls, 1984). Table 5 compares these data with those from our patient's [hVd] series. As the table shows, our patient's standard deviations are in line with those of normals on all three measures.

TABLE 5
Comparison of Standard Deviations for F1, F2, and Vowel Durations in Normal, Aphasic, and FAS Data

| | FAS cases | | Normals | Aphasic groups | |
|---|---|---|---|---|---|
| Parameters | Current Pt. | 1987 Pt. | | Anterior | Posterior |
| F1 | 32.5 | 64.0 | 24.0 | 45.0 | 42.0 |
| F2 | 59.6 | 118.0 | 58.0 | 91.0 | 89.0 |
| V dur. | 20.9 | 43.3 | 20.8 | 48.4 | 44.7 |

To summarize results from the analysis of vowel production, our patient displayed normal intrinsic vowel duration patterns and differed little from his own (pre-CVA) normative baseline. His F1/F2 distribution plots, however, indicated an abnormal centralizing tendency which restricted his vowel space. Both our current patient and the 1987 FAS patient were noted to have lower F1 values than normative data, suggesting that at least some FAS patients may have a tense vocal tract setting. Last, a major difference to emerge between our FAS patient and the 1987 patient is the fact that our patient's vowel productions had normal standard deviations, while those of the other patient showed the high variability associated with aphasic speech.

## ANALYSIS OF SPEECH PROSODY

Prosodic disturbances on the word and/or the sentential level have been a hallmark of the foreign accent syndrome literature. In the 1987 study, where acoustic analysis was first used to determine the nature and extent of prosodic problems in an FAS patient, the patient's spontaneous speech was shown to have several prosodic aberrations: inappropriate terminal segments, unusually large and frequent pitch excursions, and changes in word-level contours. This was also true to a lesser extent in that patient's ''read'' speech, where prosodic disturbances were found in the terminal portion of the utterance and at points of local prominence within the sentence. On the basis of these considerations, prosodic analyses were conducted on the spontaneous and read speech of this patient.

### *Stimuli and Procedures*

The stimuli consisted of sentences and phrases extracted from the spontaneous speech sample and a read speech sample using the list of sentences from the 1987 study.

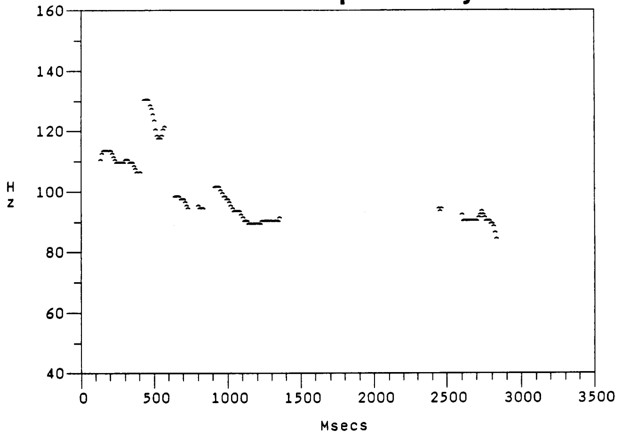
### *Analysis of Results and Discussion*

Fundamental frequency analyses (pitch plots) of individual sentences were derived by computer. Pitch plots were initially obtained for eight spontaneous speech sentences. Most of these productions had long pauses, due to his slower, halting speech. These pauses resulted in what appeared to be isolated or fragmentary clausal units on the pitch plots. This pattern is shown in Fig. 6 (top), where there is a long pause after the verb ''speak'' (''I feel I cannot speak . . . very well''). Despite these long pauses, the fundamental frequency patterns of the sentences appeared to be normal. There were appropriate continuation rises at the ends of phrases within the sentence as well as appropriate terminal falling contours. The overall global prosody was appropriate across sentence types. Even in the presence of a long delay, there was an overall continuity of the fundamental frequency contour, indicating that the speaker maintained the overall prosodic contour of the sentence and continued the pitch contour after the pause. Similar results were obtained with the read sentences. There was a normal pattern of pitch excursions with rising and falling intonation patterns as a function of the syntactic structures of the sentence (see Fig. 6, middle and bottom). These results confirm the perceptual judgements that this patient's overall speech prosody was normal.

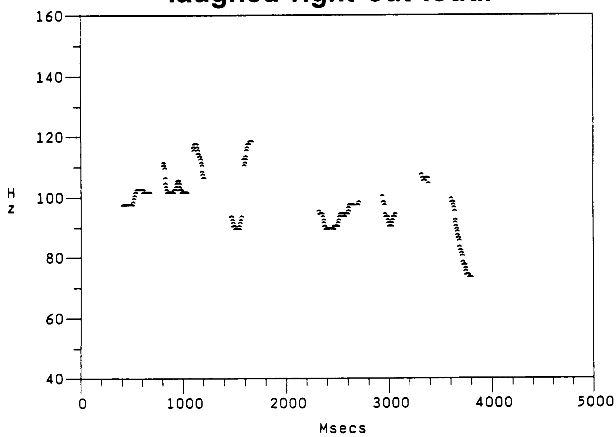
## GENERAL DISCUSSION

The results of this study provide some interesting comparative data to previous acoustic analyses of FAS and suggest some modifications to current interpretations of the nature and mechanisms underlying the foreign accent syndrome. Similar to earlier conclusions (Blumstein et al., 1987; Gurd et al., 1988; Ingram et al., 1992), FAS does not reflect the acquisition of a foreign accent. The anomalous phonetic features of our patient included vowel centralization, the production of flaps as full stops, and the relatively infrequent addition of an epenthetic schwa in a CVC environment. All other phonetic features analyzed (including voice-onset time, place of articulation in stop consonants, vowel duration, variability of vowel production in terms of F1 and F2 values and duration, and global prosody) were typical of normal native English speakers. Taken together, these speech characteristics are probably best described in the context of a ''generic'' foreign accent, generic because they all occur as phonetic characteristics in natural language but do not necessarily characterize the phonetic characteristics of any particular language.

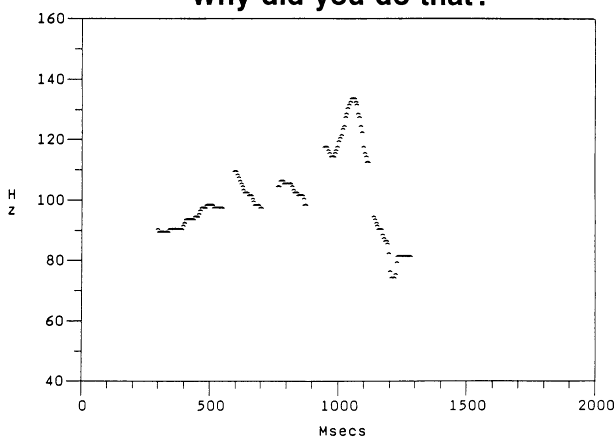
Nevertheless, the phonetic patterns of our patient also suggest that, while there may be a constellation of properties that accompany FAS, these properties may be affected in potentially different ways. Thus, our patient showed normal global prosody, in contrast to Blumstein et al. (1987) and a number of other cases who reported large pitch excursions and global changes in the prosodic contour of their patients' speech. Still, the limited local dysprosody that resulted from the addition of epenthetic schwas in our patient's speech must be viewed in the context of descriptive reports in the FAS literature of other cases of prosodic deficits only at the word and/or phrasal level (Pick, "I feel I cannot speak very well."




"When he heard this joke, he laughed right out loud."




"Why did you do that?"



1919; Whitty, 1964; Whitaker, 1982). As in our patient's case, these deficits often involved word stress and syllable structure. Thus, prosody in FAS may be affected globally, locally, or in some combination of these prosodic levels. Similarly, our patient showed a strong centralizing tendency in vowel production, and vowels tended to become more lax. In contrast, the Blumstein et al. FAS patient produced vowels that were more tense. Both characteristics, i.e., the production of vowels that are either more tense or more lax, have been reported in the FAS literature.

What these results suggest is that FAS patients have broadly similar phonetic characteristics affecting especially vowel production and prosody (in our patient at least to the extent that segmental characteristics contribute to word or phrasal stress patterns). It is these broad categories of impairment which give rise to the perceptual impression of a foreign accent. However, the particular pattern and direction of anomalous phonetic features may differ across patients. Thus, the foreign accent syndrome is just that, a syndrome, characterized by a constellation of features which may be more or less present and which may be affected in a limited number of different ways. In this sense, the foreign accent syndrome is similar to the aphasia syndromes where specific linguistic attributes defining the syndrome may be affected, and yet specific details may vary from patient to patient. For example, some agrammatic patients show greater preservation than others of grammatical words such as ''the'' and ''is.'' In addition, some agrammatic patients show syntactic comprehension impairments similar to their production impairments, and others do not. Finally, some have accompanying phonetic impairments, and others show minimal, if any, deficits of this type.

We are left then with the challenge of determining the nature of the underlying mechanism responsible for FAS. Lecours et al. (1983) proposed a paretic/hypertonic dichotomy in which undershoot errors indicated a paretic vocal tract setting, while overly tense productions indicated a tense vocal-tract setting. However, Gurd et al. (1988) found that their FAS patient produced both paretic and hypertonic errors. Graff-Radford et al. (1986) and Ingram et al. (1992) concluded that their patient's production errors were due to a tense vocal tract setting. In the case of Ingram et al., however, there were both lenition as well as fortition processes evident in their patient's speech, making a tense vocal tract setting an unlikely explanation for the deficit. In addition, the present study found evidence of lenition in vowels (i.e., tense vowels becoming more lax). Finally, Blumstein et al. (1987) attributed both segmental and prosodic deficits to a general prosodic disturbance. It is difficult to ascribe the speech pattern of our patient to a similar mechanism. First and foremost, global prosodic impairments were absent in

FIG. 6. Fundamental frequency plots of sentences from spontaneous speech (top) and from read speech (middle and bottom).

our patient. Moreover, certain accompanying features of segmental production affecting speech rhythm and thus speech prosody (i.e., the production of epenthetic vowels) were also quite infrequent. The salience of the vowel production changes overshadowed all other phonetic dimensions analyzed. Although changes in stress and rhythm could result in the production of more centralized vowels, the lack of more generalized prosodic impairments speaks against the basis of the vowel production patterns as being due to a prosodic disturbance. Rather, such changes probably more nearly reflect modifications to the shape of the vocal tract and in particular in the shape and position of the tongue. The nature of our patient's vowel productions suggest a failure to move the tongue body as much as it should to reach a phonetic target, and in this sense, the vocal tract is constricted.

While we are reluctant to dismiss the possibility, it is not clear whether a single mechanism could produce the constellation of deficits found in FAS. Our patient demonstrated vowel changes that relate to control of the supralaryngeal vocal tract. The Blumstein et al. 1987 patient had a deficit affecting not only vowel production but also prosody, the latter implicating laryngeal control problems. Their patient also showed vowel production anomalies with a lowering of F1 values corresponding to a more closed vocal tract, although the vowel space of the 1987 patient was more similar to normals than in the present case. Thus, the overriding prosodic disturbance may have ''masked'' the perceptual impact of the accompanying mild impairment in vowel production.

Be that as it may, while it is not clear whether one mechanism can be identified which characterizes FAS, it is clear that whatever the mechanism producing FAS, it is not the same as that producing the speech deficit in Broca's aphasia. Characteristic features of the speech pattern of Broca's aphasics include deficits in consonant production including the timing of independent articulators, phonetic distortions, and speech prosody characterized by a flattened ''monotonous'' intonation pattern both locally and globally. Thus, FAS patients do not manifest a qualitatively similar but milder form of the speech impairment of Broca's aphasics. Rather, their deficit is qualitatively distinct.

It is possible that the different patterns that have emerged in FAS reflect damage to different pathways and motor output systems (cf. for example, Alexander, DeLong, & Strick, 1986). In the seven cases with detailed publication of lesions (Schiff et al., 1983; Blumstein et al., 1987; Graff-Radford et al., 1986; Gurd et al., 1988; Takayama et al., 1993; Ardila et al., 1988; and the present case), there is no simple consistent finding. All seven have relatively small lesions: two involve the prerolandic cortex (Schiff et al., 1983) or the white matter immediately subjacent to prerolandic cortex (Blumstein et al., 1987) for much of the extent of representation of the contralateral facial and bulbar muscles; three involve the premotor association cortex, either area 6 (Graff-Radford et al., 1986; Takayama et al., 1993) or area

44 (Ardila et al., 1988), one extending to the anterior periventricular white matter (Graff-Radford et al., 1986); one involves only the posterior putamen (Gurd et al., 1988). In the present case, there is involvement of the putamen and the anterior periventricular white matter. These last five cases all involve nonpyramidal motor systems often considered to be involved in preparation and sequencing of complex motor performance (Alexander et al., 1986).

All cases had some prolongation of speech, in either vowel production, word duration, excessive pausing between words, or long latencies of speech onset. All cases may have had some abnormality of tone of the vocal tract—excessive tension or laxity. These two classes of deficits are likely related to mild deficits in either rolandic or nonpyramidal motor function.

FAS is diagnosed only when there are minor or no impairments in language, either content or syntax. The minor disturbances of verbal fluency and rare agrammatisms described in these patients may only reinforce the listener's impression of a ''foreign speaker.'' The diagnosis of FAS has also required that articulatory precision be adequate, that speech is not perceived as simply dysarthric. Having met these two criteria, apparently any of a number of anomalies of overall prosody, local prosody, and vowel production can generate the impression of ''foreignness.'' The lesions in these cases have all been within the region typically damaged in Broca's aphasia. Yet, as discussed above, the speech patterns of FAS and Broca's aphasics are not similar, nor do they seem to reflect differences in severity along a continuum. What these results suggest is that there may be a number of motor output systems that may function quasi-autonomously using some of the same or adjacent neural structures. To further attempt to identify both the functional and the neural basis of FAS, more detailed quantitative comparative analyses of the syndrome must be conducted in conjunction with more precise neuroanatomical investigations.

Based on the results of this study, it is appropriate to raise the possibility that in the final analysis the foreign accent syndrome may not be a syndrome at all. At this point in time, there does not seem to be an identifiable and consistent set of attributes to define the syndrome, there does not seem to be a selective lesion focus, and there has been no common mechanism identified to characterize the behavioral symptomatology—all essential elements in the definition of a syndrome. In fact, the only agreed-upon characteristic is that upon examination, patients clinically sound as though they are speaking with a foreign accent. There are accompanying negative characteristics as well—there is rarely an associated aphasia, patients are not dysarthric, and their speech patterns do not fit the patterns described under the syndrome of apraxia of speech. Such negative characteristics are probably not enough to define a syndrome.

Why then do we persist in seeking to characterize the phonetic characteristics of this disorder, its potential neuropathology, and its underlying mechanism, instead of concluding that the foreign accent syndrome is an epiphenomenon existing only in the ''ears'' of the beholder. While such a possibility exists, it is our view that such a conclusion is premature. First, there have simply been too few quantitative and systematic acoustic–phonetic studies to conclude that there is no consistency to the phonetic characteristics of these patients. Second, the failure to find a common neuropathology may be premature for the same reasons. If we consider the possibility that focal lesions impair networks rather than discrete isolated areas and associated functions (cf. Damasio, 1989), then it is possible that the different neural sites identified to date associated with the foreign accent syndrome affect a common neural system or neural systems that interact with each other. It is the case that patients described as having the foreign accent syndrome have all had left lateralized anterior–frontal lesions. No cases have been reported with unilateral right hemisphere disease or with left posterior lesions. Third, it is rarely the case that a single mechanism can explain the patterns of impairment and preservation which characterize a syndrome. In fact, it is not uncommon to find that not all characteristics of a syndrome emerge in a given patient or are present to the same degree among patients similarly classified. Neither the language problems associated with Broca's nor Wernicke's aphasia have been characterized to date by a single underlying mechanism (Goodglass, 1993). It is perhaps unfortunate that the foreign accent syndrome was named as such since it suggests in its name an explanation and characterization of the syndrome that we know now to be untrue. Patients with this speech output problem have not acquired a foreign accent. Presumably, agrammatic aphasia would still be considered a syndrome even if it turns out that the so-called productive agrammatism of these patients does not reflect an underlying syntactic deficit. The same may also be the case for the foreign accent syndrome. In effect, taxonomies in aphasia research and in research on associated language and speech disorders do not have to be theoretically motivated. Rather, syndromes (even loosely defined ones like the foreign accent syndrome) serve research to the extent that they support the collection of theoretically relevant data. The issue surrounding whether the foreign accent syndrome is a syndrome or not awaits the consideration of that data.

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