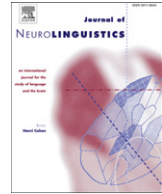




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Foreign accent syndrome due to conversion disorder: Phonetic analyses and clinical course

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ABSTRACT

We describe the case of a 36-year-old native speaker of English who began using a foreign accent after abrupt onset of problems involving multiple sensory and motor functions. Neurological and neuroradiological examinations were within normal limits and no organic explanation for the difficulties could be identified. After eight months, the patient made a full recovery, allowing a detailed comparison between the speech patterns displayed during the period of foreign accent and those of her typical speech. Perceptual and acoustic analyses showed specific changes in vowel and consonant production and in intonation and stress patterns during the accented speech. These changes were similar to those described in the literature for patients with foreign accent syndrome following left cerebral hemisphere lesions. A review of presenting symptoms and clinical course of our case, however, indicated strong evidence for a conversion disorder. This case demonstrates that a psychogenic etiology for foreign accent syndrome should sometimes be considered and that the prognosis for recovery, including spontaneous remission, may be good in such cases. Differential diagnosis between psychogenic and neurogenic etiologies is discussed relative to the case presentation, history, course, and recovery.

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1. Introduction

1.1. Speech characteristics

The foreign accent syndrome (FAS) is an acquired speech disorder in which a native language user suddenly starts using prosodic and articulatory patterns that cause listeners to perceive a foreign-sounding accent. To date, approximately 50 cases of FAS have been published in scientific journals, with a dramatic increase in the proportion of cases published in recent years (9% prior to 1980; 11% 1980–1989; 25% 1991–2000; 55% 2001–2008). A review of these cases shows that the etiology and presenting symptoms of FAS are far from uniform. Despite good agreement among listeners that the accent sounds “foreign,” there is typically poor agreement as to the language associated with the accent (e.g., Blumstein, Alexander, Ryalls, Katz, & Dworetzky, 1987; Christoph et al., 2004; Gurd, Bessel, Bladon, & Bamford, 1988; Ingram, McCormack, & Kennedy, 1992). Detailed phonetic analyses of speech patterns have revealed allophonic variations that are atypical of the speakers’ native language, particularly in prosody and vowel quality, but the unavailability of similar measures from the individuals’ normal speech limits conclusions regarding the precise nature of the changes. In selected cases, preexisting audio recordings were available (e.g., Dankovičová et al., 2001; Gurd, Coleman, Costello, & Marshall, 2001), but the content could not be customized to the analysis needs. In other cases, a normal control speaker or published group data were used as a reference for unaccented speech (Blumstein et al., 1987; Coelho & Robb, 2001; Laures-Gore, Henson, Weismer, & Rambow, 2006; Miller, Lowit, & O’Sullivan, 2006; Varley, Whiteside, Hammill, & Cooper, 2006), an approach that does not account for individual dialectal or idiosyncratic articulatory and prosodic variations.

1.2. Etiology and clinical course

Recently, we (Albert, Haley, & Helm-Estabrooks, *in preparation*) reviewed 30 published cases of FAS for which neuroanatomical information on lesion localization was available. We found the most likely clinicoanatomical correlate to be a small lesion deep in left frontal white matter pathways, anterior and superior to the head of the caudate nucleus. In the majority of these cases, the etiology was stroke or traumatic brain injury and the foreign accent emerged as a transient stage of recovery following initial stages of muteness, nonfluent aphasia, apraxia of speech (AOS), and/or dysarthria. It is difficult to derive further clinical relationships from the FAS literature because information about the course, medical history, coexisting signs or symptoms, and intervention approaches has been incomplete.

A few published reports indicate that FAS can occur without organic etiology. Some of these cases differ markedly with regard to onset from those with confirmed brain lesions. For example, a foreign accent emerged in two individuals during exacerbation of a psychosis and in the context of delusions, hallucinations, and disordered thought processes (Reeves, Burke, & Parker, 2007; Reeves & Norton, 2001). In other cases with no evidence of neuropathology, psychogenic etiology of the FAS was more difficult to establish but seemed likely (Gurd et al., 2001; Poulin, Macoir, Paquet, Fossard, & Gagnon, 2007; Van Borsel, Janssens, & Santens, 2005). The speech characteristics in these cases were similar to those for individuals with documented brain lesions in that they included changes in prosody, vowel quality and duration, and allophonic consonant variations. Because very few detailed reports of FAS with possible psychogenic etiology have been published, and because the phonetic analysis has been limited in scope, it is critical to explore the phonetic speech characteristics in such cases in more detail.

In this report, we describe a case of FAS in which the etiology and evolution of the foreign accent were well documented and where detailed phonetic analyses were derived from identical speech samples with and without the accent. There was strong indication that the etiology was a conversion disorder. Six months after the onset of her symptoms, the patient made a complete recovery, allowing detailed phonetic comparison of identical speech samples produced with and without the foreign accent.

2. Case report

2.1. Presenting clinical history

DW, a 36-year-old woman, experienced sudden onset of symptoms one morning while showering. She described feeling a vibrating sensation in her head, ringing in her left ear, and an electric shock feeling in the left side of her body. She got out of the shower, looked in the mirror and noticed that the right side of her face was “scrunched up” while the left side was drooping. She tried to speak and thought her speech was slurred and difficult to understand. She was taken to a physician’s office, where she developed unsteady gait and a feeling of left-sided weakness. She also had intermittent visual blurring, altered hearing in the left ear, and paresthesias on the left side of her body. The only other recent event had occurred the prior night when she experienced fairly severe back pain with left foot weakness, but this had resolved by the time she awoke and before the new symptoms occurred.

When her physician initially saw DW, he described her speech as slurred and with abnormal cadence. He also noted weakness of the left side of her face and possible subtle weakness of the left arm and leg. She was admitted to a local hospital for evaluation of stroke. The workup included a brain MRI, echocardiogram, and blood screening for a hypercoagulable state. The MRI showed only rare punctuate T2 hyperintensities and no acute changes. The echocardiogram showed moderate mitral regurgitation that had also been present on a study two years previously. The hypercoagulability workup was negative. A neurology consultation yielded the impression that the patient might have a Bell’s palsy with additional conversion disorder symptoms. Notably, despite apparent facial asymmetry, she did not have any problems with left eye closure. At a later point, her facial asymmetry was described as a pulling up of the right side of the face, rather than drooping of the left side. For treatment she was started on a Prednisone taper and aspirin. A follow-up MRI ten days later showed no development of structural abnormalities and an MR angiogram showed no abnormality of brain vasculature.

Ten days after symptom onset, DW’s main concern was that her speech seemed unusual. Issues of less concern to her included mild trouble swallowing and continued abnormal sensations in the left face, arm, and leg. She did not have problems with left eye closure or dryness, and the flattening of the left nasolabial fold seemed less pronounced on exam. Due to the complex nature of her symptoms and initial negative stroke workup she was sent to a university medical center for further evaluation.

When seen by a stroke specialist five days later, DW reported that her symptoms had become suddenly worse the day prior to the visit. She said that her speech had worsened and walking was more difficult. On exam she was noted to be fluent in naming and spontaneous conversation, but the character of her speech was described as “quite odd” and foreign sounding. To the examiner, it sounded somewhat French or Spanish. DW’s exam was otherwise unremarkable with symmetrical reflexes and flexor plantar responses and with no weakness, cerebellar dysfunction, or apparent gait problems. An EEG was unremarkable, and an evaluation for multiple sclerosis (MS) was unrevealing with no inflammatory changes in the CSF.

Over subsequent months, DW reported intermittent but fairly frequent relapses of her neurological symptoms, generally lasting 2–4 days. These were sometimes preceded by back pain like her initial episode. Between relapses her speech problems were described as “lightly accented.” In contrast, during exacerbations requiring emergency room evaluations her speech was described as “heavily accented”. Her brother commented that during relapses she “sounded like a Jamaican from New York” and also mentioned that “it was like talking to a six year old.” Between episodes, her gait generally returned to normal, and dysesthesias and weakness symptoms subsided. Her reported reduced hearing in the left ear and blurring of vision, which were intermittently present during exacerbations, would also improve. At various times she had symptoms that were unlike those associated with her initial episode. On one occasion, she developed a week-long progressively worsening problem with bilateral “crushing” pain of the hands that would awaken her from sleep. On another occasion, after having a spinal tap, she developed right upper extremity weakness and uncontrollable shaking. She also reported problems with yelling and temper outbursts during episodes. Another brain MRI and a cervical MRI done during a subsequent relapse again showed no acute structural findings consistent with stroke or inflammatory lesions. Thus, her neurologist’s impression was that DW’s symptoms were primarily functional in nature and not consistent with stroke or MS.

2.2. Past neurological, medical, and social history

DW's prior neurological history was notable for another series of neurological symptoms that had occurred two years before the onset of those associated with speech problems and also did not have a clear etiology. This series of symptoms started with pain in both arms on a nightly basis for 20 min and was followed by several weeks of numbness and paresthesias mostly affecting the right hand. After about a month the paresthesias extended into the left hand and the right shoulder and face, and she reported difficulty performing manual tasks. With the symptoms she also had recurrent 5–60 min episodes of loss of vision affecting either eye and 15–20 min episodes of loss of hearing affecting one ear or the other. In addition she had two discrete episodes of weakness. One involved left-sided weakness lasting 2 h, and the other rendered her unable to move or speak for 2 h upon awakening. All symptoms disappeared for about three months, around the time she changed jobs. Then she had another recurrence of symptoms about a week after a gall bladder operation. These symptoms included right sided weakness, difficulty with coordination of her legs with a delay in movement affecting her left side more than her right, and involuntary jerking of the limbs. These latter symptoms gradually improved over 6–8 weeks. Neurological evaluation in the past had included an MRI, EEG, and blood laboratory testing for Lyme disease, lupus, and HIV. She was also treated for carpal tunnel with wrist braces without resolution of her hand numbness. A neurological consultation at a tertiary care center concluded that she did not meet criteria for MS.

DW's prior medical history also included chest pain and shortness of breath that led to cardiac catheterization, which was unrevealing of atherosclerosis but did show mitral valve regurgitation requiring yearly follow-up. DW was married and had no children. For the prior 14 years she worked as an ancillary medical professional. She was a native English speaker, born and raised in a Midwestern state to American parents. Around age 9, she started learning Spanish from friends and extended family members, and she described her current fluency in this language as moderate.

2.3. First visit: speech-language evaluation

We first saw DW about four months after the onset of the foreign-sounding accent. She reported that she was constantly being asked what country she was from and that many thought she was from the Caribbean. Her speech was characterized by an obvious foreign accent quality with normal fluency and no apparent language difficulties. Test results available from a neuropsychological evaluation approximately three months earlier described visual confrontation naming in the low average range on the Boston Naming Test (Kaplan, Goodglass, & Weintraub, 2001); normal auditory word and sentence comprehension, flawless sentence repetition, and normal oral and written spelling on subtests of the Multilingual Aphasia Examination (Benton, Hamsher, & Sivan, 2001). Oral sentence reading on the Boston Diagnostic Aphasia Examination (Goodglass, Kaplan, & Barresi, 2001) was normal, but on tasks of reading comprehension from the same test, she was reportedly slow to respond and her scores were inconsistently in the normal or impaired range. When evaluated in our clinic, she scored well within normal limits on all subtests of the Cognitive Linguistic Quick Test (Helm-Estabrooks, 2001).

Our exam showed normal motor function of the speech mechanism. She performed nonverbal movements on command accurately and with normal strength, range, and steadiness, although sometimes the response initiation was slightly delayed. There was no evidence of nonverbal oral or limb apraxia; no obvious asymmetry to her face, tongue, or velum; and no sign of dysfunction in respiratory, phonatory, or velopharyngeal subsystems for speech production. Alternating Motion Rates (AMRs) and Sequential Motion Rates (SMRs) for speech were produced with vowel and consonant changes consistent with the perceived accent, but with no hesitation, sound or pause prolongation, or articulatory imprecision. The rate was regular and in the lower normal range (4.0–4.6 syllables per second for AMRs; 4.8 syllables per second for SMRs).

DW repeated multi-syllabic words and sentences with the same vowel and consonant changes as in her conversational speech and without apparent effort or loss of fluency. She did not repeat or revise her productions. Conversational speech intelligibility was judged to be mildly reduced, in that it was sometimes necessary for the listener to request clarification or repetition. On these occasions, DW repeated the word and sometimes offered a synonym or description, but did not otherwise attempt to

revise the utterance. Her speech patterns were the same for spontaneous conversation, automatic speech, and repetition. The speaking rate and pitch appeared normal, and acoustic analysis of a 2-min speech sample supported these impressions (rate: 134 words per minute, including pauses; mean fundamental frequency: 200 Hz, SD = 27 Hz).

2.3.1. *Perceptual analyses*

To characterize the overall nature of DW's speech, a 2-min video sample from the initial interview was presented to 58 second-year graduate students in speech-language pathology. The students were asked to comment, independently and in writing, on the speech characteristics. Although the instructions did not mention the possibility of a foreign accent, all students responded that the speaker used a foreign accent, with a variety of accent types proposed, the most frequent being Caribbean, African, and French. No students detected a speech disorder of any kind.

A digital audio recording (22 kHz sampling rate, 10 kHz low-pass filter, 16 bit quantization) was obtained from oral reading of 50 monosyllabic single words and a standard text ("Grandfather Passage"). Single word intelligibility was estimated by presenting the 50 words, one at a time, to five naïve listeners and asking them to type the words they thought the speaker was trying to say. This procedure yielded an intelligibility score of 50%, indicating that the consonant and vowel changes had salient effects on listeners. Finally, the conversational speech, paragraph reading, and single word sample were reviewed independently by two of the authors (KLH and AT), using narrow phonetic transcription. Segmental phonologic and allophonic substitution patterns and prosodic characteristics identified by both transcribers are reviewed in the next section.

2.3.2. *Results of phonetic transcription analyses*

There were three prominent changes in DW's vowel system. First, the distinction between tense and lax vowels with adjacent place of articulation, such as /i/ vs. /ɪ/ and /u/ vs. /ʊ/ was severely compromised. For example, the word "bit" was perceived as "beet," "seek" was perceived as "sick," and "looking" was perceived as "lukeing." Similarly, the central vowel in words, such as "public," "grunt," and "bunch" was abnormally prolonged. Second, many vowel diphthongs were monophthongized. For example, the word "foundation" was pronounced as "fondation," the word "spoiled" as "spold," and the word "trail" as "drell." Third, there was often centralization or backing of tongue position, so the word "back" was produced as "buck," and the word "animal" was produced as "unimal."

For consonant production, DW displayed four distinct patterns. First, interdental fricatives /θ/ and /ð/ were stopped. For example, "this" was produced as "dis," and "worth" was produced as "wort." Second, she often produced incorrect or ambiguous stop consonant voicing, such as "drell" for "trail," and "guite" for "quite." Third, alveolar stops /t/ and /d/ were dentalized, with the impression of tongue tip contact with the upper incisors rather than the alveolar ridge. Fourth, liquids /r/ and /l/ were intermittently distorted. Vocalic /r/ lost the rhotic quality, leading to pronunciation such as "monin," for "morning" and "wok" for "work." Prevocalic /r/ sounded distorted and approached /w/, so that "street" sounded more like "stweet." Finally, postvocalic "dark" /l/ was either absent or produced as the clear /l/ typical of prevocalic positions.

DW's pitch contour was unusual and at times appeared to alternate rigidly between higher and lower pitch levels. Variations in word and sentence stress were atypical for American English. For example, "donate" was produced with stress on the second syllable and "baby sitter" with stress on the third syllable. She could, however, easily vary affective and linguistic prosody according to instructions to display specific emotions or in contrastive stress tasks.

2.4. *Speech and motor behaviors during "relapse"*

The week after her initial appointment in our clinic, DW experienced a relapse of multiple symptoms, which resolved within days in the typical fashion. With the help of her husband, she made a home video to demonstrate the speech and gait problems she was experiencing. Because the speech problems were variable and the gait problems had not been seen at clinic visits, the video provided valuable information to understand the presentation of her problems better.

On the video DW described the onset of the relapse and the visual, auditory, and motor problems she was experiencing. Pragmatically, her demeanor appeared somewhat disinhibited; several times she laughed out loud while explaining her difficulties. Her face was asymmetric with the right nasolabial fold more pronounced than the left and she commented that the right side of her face was “scrunched up.” The speech quality gave the impression of unusual, childish, or disordered speech. While present, the foreign accent quality was not as striking as before. Additionally, her pitch was abnormally high with clearly exaggerated intonation, and the voice quality was mildly strained with an intermittent mild hypernasal quality. Acoustic analyses verified that the mean fundamental frequency was approximately 40 Hz higher and more variable (240 Hz, SD = 60 Hz) than in the previous sample. In comparison, the speaking rate was also slightly lower (108 words per minute, including pauses). At times, her facial movements appeared stiff, but there was no indication of weakness or involuntary movement. There were occasional whole word repetitions in her speech, which she described as stuttering, but no part-word repetitions, no segmental or inter-segmental prolongations, and no self-corrections.

A striking feature in this speech sample was that she consistently produced /w/ instead of /r/ in prevocalic position (e.g., “bwen” for “brain”) and dropped rhotic vowel quality in postvocalic position (e.g., “hoot” for “hurt”). The intermittent liquid distortions that characterized the initial speech sample were no longer present. Like previously, there was a tendency to centralize vowels (e.g., “hud” for “head” and “cutching” for “catching”) and a tendency to back high front vowels was more evident than before (e.g., “spooch” for “speech,” “fooling” for “feeling”). As before, interdental fricatives were stopped throughout (e.g., “din” for “thin”). Stop consonant voicing ambiguity and dentalization changes were present but appeared less prominent than before.

DW's gait during the videotaped relapse was markedly abnormal, appearing almost spastic. Movements of the legs were jerky and deliberate, and the legs appeared to cross slightly while she was walking. In contrast, her arms were held out away from her body and bobbed back and forth in response to the jerky movement of her lower body. As her body jerked to stop with each step, her arms would jiggle and her head would bob and then she would take the next step. She would lean to one side, but would maintain the narrow base and had no apparent tendency to fall.

2.5. *Second visit: speech sample recording*

When we saw DW the following week, her gait was normal and her speech patterns were similar to those of her first visit. To examine our perceptual impressions acoustically, we recorded an additional speech sample, consisting of words with phonetic segments we had noted to be affected by her accent and following the methodology of previous cases described in the literature (Blumstein et al., 1987; Kurowski, Blumstein, & Alexander, 1996; Laures-Gore et al., 2006). The findings will be detailed in later sections describing acoustic analyses.

2.6. *Third visit: treatment and recovery*

We suspected a possible psychogenic etiology for the foreign accent and therefore administered a symptomatic treatment session to attempt to modify DW's speech pattern and explore possible factors that may have triggered the altered accent. The approach followed general principles for symptomatic intervention in psychogenic voice and speech disorders (Duffy, 2005). The clinician reviewed the normal structural-functional and motor speech examinations, raised the possibility of a psychological reason for the speech changes, and suggested that a series of exercises may help her remember her normal speech patterns. Treatment was then administered using exercises typical of accent reduction for nonnative speakers of English (see, for example, Sikorshi, 2005) but at a considerably higher pace, so that all main articulatory and prosodic features that contributed to the foreignness of her speech were targeted within the single session. A combination of repetition, auditory perceptual rating, and self-correction techniques were used, progressing from syllables to paragraphs. During the exercises, DW repeatedly produced targeted structures accurately; but she did not embrace her accomplishments either spontaneously or when they were pointed out to her and the normalized speech patterns did not generalize to her spontaneous speech. Although she accepted the

idea that her speech changes could possibly have been triggered by psychological factors, she did not identify any life changes or emotional stresses that might have been related to the onset of her symptoms.

Following this treatment session, DW continued a similar treatment program in a different outpatient clinic. After four sessions, she requested a break from therapy. She subsequently reported that she started feeling better gradually over a period of 2–3 weeks. She woke up one morning and noticed that her accented speech was gone and her speech had returned to normal. She described being elated with the sudden resolution of her problems and spent the morning calling all her friends so that they could listen to her normal speech. At this time all other motor and perceptual changes disappeared as well.

2.7. Fourth visit: return to normal speech

We saw DW for the fourth and final visit 6 weeks after the foreign accent had disappeared and approximately four months after the initial visit to our clinic. At this time, her speech had no trace of a foreign accent and she reported that she had not experienced any of the other perceptual or motor symptoms since her recovery. She explained that she was very pleased with how everything was just like before, even though she could not explain what might have triggered either the onset of the difficulties or their resolution.

The speech samples that had been recorded during her first two visits were repeated on this final visit, using identical audio recording equipment and procedures. This time, her single word intelligibility was normal at 94%. The speech AMRs were produced at a rate of 6.2 syllables per second and the SMRs were produced at a rate of 7.3 syllables per second, well within the normal range. Conversational speaking rate was slightly faster at 150 words per minute and the mean fundamental frequency was slightly lower at 185 Hz (SD = 59 Hz) than at the initial visit when she spoke with a foreign accent (134 words per minute, 200 Hz).

2.8. Acoustic analyses of speech with and without foreign accent

Acoustic analyses were conducted on the speech samples recorded while DW was using a foreign accent and after recovery. Segmental measures were completed independently by two observers and inter-rater reliability was estimated for 100% of the utterances. For formant measures, point-to-point agreement within 100 Hz was between 85% and 97% ($r = .98$) and for duration measures, point-to-point agreement within 10 ms was 97% ($r = .98$).

2.8.1. Vowel changes

Eight words were elicited five times each in pseudorandom order (heed, hid, head, had, who'd, hood, hawed, and hod). Linear Predictive Coding (LPC) analyses were performed at the vowel midpoint or steady-state (if one existed), and the center frequencies for the first (F1) and second (F2) formants were determined. Fig. 1 shows an F1–F2 plot based on measurements during the FAS period and after recovery. Consistent with the perceptual analysis, there was a spectral merging of tense and lax high vowels during the foreign accent, followed by a normal distinction after recovery. Interestingly, after recovery she also showed normal dialectal fronting of high back vowels (e.g., Hagiwara, 1997).

To examine the tense/lax vowel contrast further, vowel duration was measured from the beginning of periodicity in the vowel to the vocal tract closure for the following stop consonant. As illustrated in Fig. 2, duration values were significantly longer during FAS ($M = 301$ ms, $SD = 40$ ms) than after recovery ($M = 246$ ms, $SD = 43$ ms; $t = 7.24$; $p < .001$). More importantly, the distinction between tense and lax vowels was affected, so that duration values for the long vowels /i, u, æ/ were no different from the normally shorter vowels /ɪ, ʊ, ɛ/ during FAS, but significantly longer after recovery ($t = 5.49$; $p < .001$).

Finally, fundamental frequency was estimated for each vowel production by dividing the number of cycles visible in the vowel waveform by the duration of the vowel segment. These measures were higher during the FAS ($M = 261$ Hz, $SD = 11$ Hz) than after recovery ($M = 199$ Hz, $SD = 11$ Hz; $t = 35.47$; $p < .001$), possibly reflecting increased production effort or vocal tract tension.

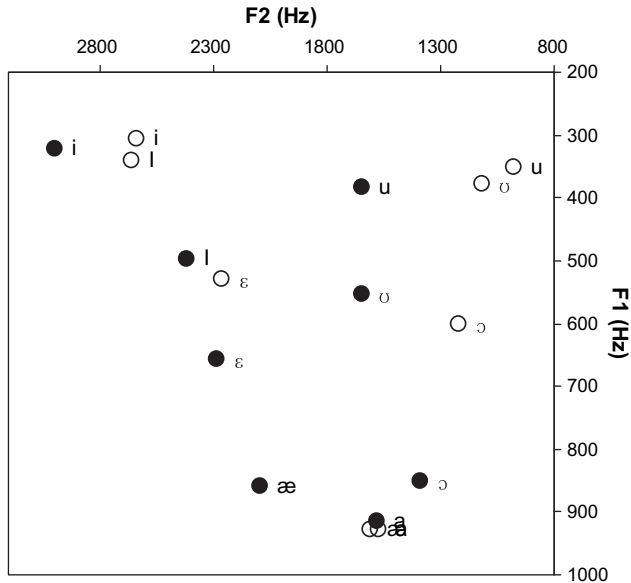


Fig. 1. F1–F2 plots for vowels produced during the period with FAS (open circles) and after recovery (closed circles).

2.8.2. Consonant changes

The perceptual analysis indicated that two major changes for stop consonants were ambiguous voicing and dentalization of alveolar stops. To examine these features acoustically we used a speech sample consisting of monosyllabic words starting with a voiced /b, d, g/ or voiceless /p, t, k/ stop and then the vowel /a/. The words were produced in the carrier phrase “this _____,” and repeated four times in random order. Voice onset time (VOT) was measured from the release of the burst to the onset of voicing. As shown in Table 1, there was no overlap between voiced and voiceless productions, either during FAS or after; and VOT increased progressively from bilabial to alveolar to velar place of articulation in the normal fashion. However, VOT for voiceless stops was significantly shorter during FAS than after

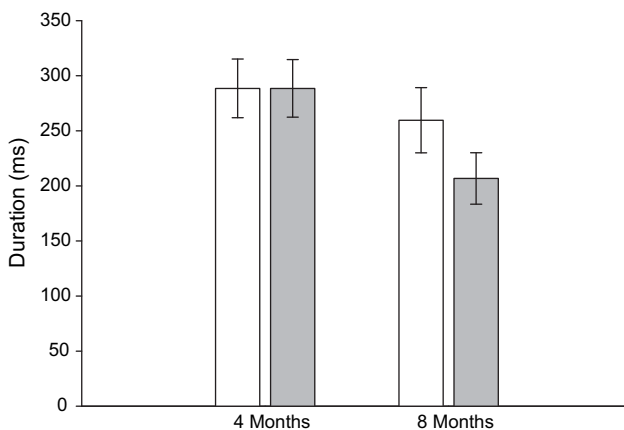


Fig. 2. Mean duration for intrinsically long vowels /i, ε, u/ (white bars) and intrinsically short vowels /I, ɛ, ʊ/ (grey bars) during the period with FAS (4 months) and after recovery (8 months). Error bars represent one standard deviation.

recovery ($t = -12.4$; $p < .001$), with values for many productions in or very near the expected range for voiced stops, thus supporting the voicing ambiguity detected in auditory perceptual analysis. Additionally, negative VOT (prevoicing) in voiced stops was more prevalent and of greater magnitude during FAS than after recovery, an articulatory feature that may be indicative of increased production effort.

We used spectral moment analysis to examine the effect of dentalization on alveolar stop production. The shapes of short time spectra derived at consonant release are known to correspond to different places of articulation (Stevens & Blumstein, 1978). Alveolar stops are characterized by a spectrum that rises gradually in amplitude from lower to higher frequencies. If produced with a more anterior constriction, such as dentalization, a wider spectral distribution would be expected (Lahiri, Gewirth, & Blumstein, 1984). Thus, we predicted that the second spectral moment, the variance, would be greater for dentalized productions than for normal alveolar productions. Using the 20 words from the stop consonant sample that started with /t/, a 25.6 ms half-Hamming window with pre-emphasis was placed at the onset of the burst, an LPC analysis was performed, and the second moment was derived from this analysis. As predicted, it was significantly greater during FAS ($M = 2900$ Hz; $SD = 590$ Hz) than after recovery ($M = 1230$ Hz; $SD = 220$ Hz; $t = 5.37$; $p < .001$), reflecting a greater spread of energy for the dentalized stops than for the normally produced alveolar stops.

A comprehensive speech sample was not elicited for quantification of changes to the liquid consonants /r/ and /l/. Instead, 13 words from the speech intelligibility test that contained prevocalic /r/ and/or postvocalic /l/ were examined. A distinguishing characteristic of /r/ is a lowering of the third formant (F3), very nearly approaching F2. Eleven words with prevocalic /r/ were examined (brain, brave, breed, grand, grip, group, proud, rude, scrap, trail, and write). The F3 center frequency was measured at the beginning of the syllable or following the burst release and aspiration of a preceding stop consonant. During FAS, the mean F3 onset for these words was higher than expected ($M = 2653$ Hz; $SD = 139$ Hz) and after recovery it approximated F2 ($M = 2118$ Hz; $SD = 142$ Hz), indicating that the perceptually ambiguous or absent /r/ quality was matched by equally ambiguous or absent acoustic /r/ characteristics during the accented speech. To examine acoustic correlates of a dark /l/ quality, we utilized the fact that the second format, F2, signals relative advancement of the tongue in the oral cavity, with more posterior constrictions associated with a lowering of this formant. Three words from the intelligibility sample had a postvocalic /l/ (false, spill, trail) and in these words we measured the F2 center frequency at the last point in the spectrogram where the formant was visible. As expected from the perceptual analysis, F2 lowering was seen after recovery (1260 Hz; 1020 Hz; 1230 Hz), but not during the period with FAS (1745 Hz; 1530 Hz; 1533 Hz).

Finally, qualitative inspection of target words with interdental fricatives, taken from the “Grandfather Passage” and the 50-word intelligibility sample, consistently showed a pattern of gradually increasing frication amplitude after recovery and a pattern of abrupt onset frication during the period with FAS. The abrupt frication onset during accented speech was associated with a visible burst release and/or preceding attenuation reflective of vocal tract closure, as would be expected with a stop manner of production.

2.8.3. Prosody changes

An oral reading of the “Grandfather Passage” allowed direct comparison of prosodic variations during FAS and after recovery. To some extent, the observed differences were secondary to a lower than normal reading rate (70 words per minute) and elevated fundamental frequency ($M = 240$ Hz, $SD = 44$

Table 1
Mean voice onset time (VOT) in milliseconds during the period of FAS (4 month visit) and after recovery (8 month visit). The range is reported in parenthesis.

	4 Months	8 Months
p	29 (19–43)	53 (39–70)
t	33 (16–56)	68 (48–84)
k	54 (41–76)	70 (56–92)
b	–101 (–145 to 16)	–6 (–70 to 28)
d	–73 (–117 to 14)	20 (13–31)
g	–52 (–122 to 25)	31 (23–38)

Hz) during FAS. Note that these values were different from the conversational speech sample recorded at the first visit to our clinic (201 Hz fundamental frequency, 134 words per minute), most likely reflecting increased effort during the oral reading task. Following recovery, DW's oral reading rate of 150 words per minute was the same as her conversational speaking rate, as was her fundamental frequency ($M = 185$ Hz, $SD = 59$ Hz).

Fig. 3 shows fundamental frequency contours for two phrases from the paragraph reading task. In addition to differences in duration and average fundamental frequency, there were marked qualitative differences in the shape of the fundamental frequency contours. Note the regularly spaced alternation between higher and lower frequencies during FAS and the less rigid intonation contour after recovery. This acoustic pattern was associated with the perception of a “sing-song” manner of intonation and, in combination with equalization of syllable duration, the perception of stress on most words in the utterances. It should be noted that DW's speech did not always have this alternating pitch quality. For example, her conversational speech was sometimes characterized by stretches with relatively flat intonation followed by fundamental frequency elevation at what seemed to be unusual locations. Because it was not possible to reproduce this conversational speech sample verbatim, acoustic comparisons on an utterance-by-utterance basis could not be completed for these prosodic variations.

3. Discussion

3.1. Nature and characteristics of speech changes

3.1.1. Differential speech diagnosis

The differentiation of FAS from more common neurogenic disorders that affect speech articulation and prosody is important for both clinical and theoretical reasons. In the majority of FAS cases, the accented

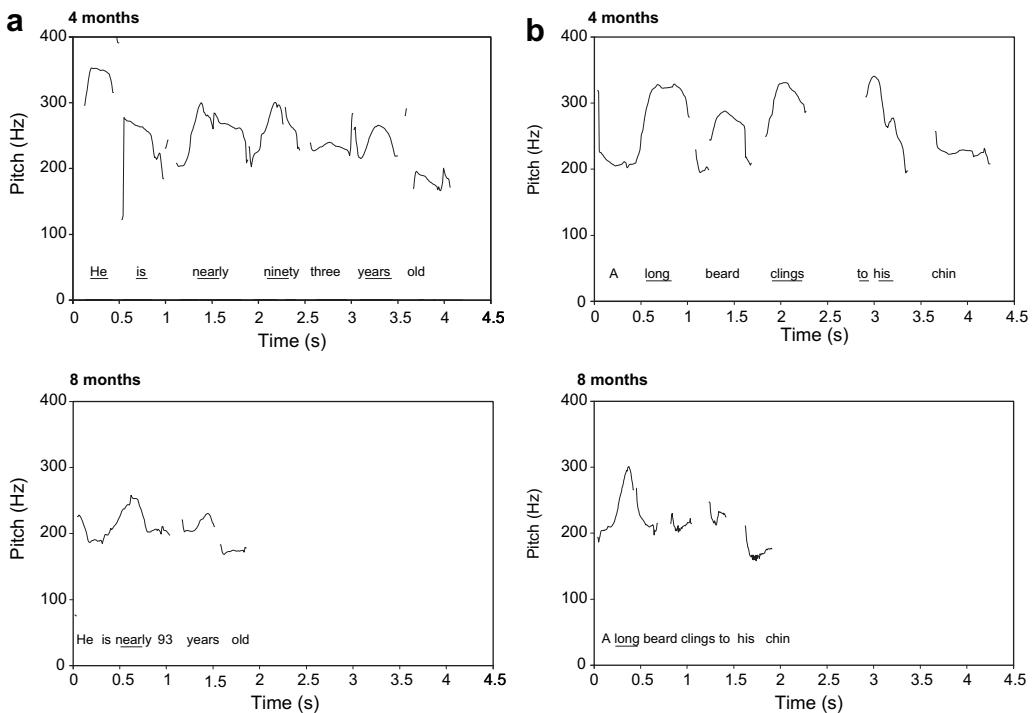


Fig. 3. Fundamental frequency contours for two phrases from the “Grandfather Passage,” recorded during the period with FAS (top; 4 months) and after recovery (bottom; 8 months). Syllables perceived to be stressed are underlined.

speech evolved from, or coexisted with, nonfluent aphasia, apraxia of speech (AOS) and/or dysarthria. The close relationship with these neurogenic communication disorders has prompted some authors to question the validity of the diagnosis as a separate entity. For example, noting the presence of a “motor aphasia” in many published cases, [Ardila, Rosselli, and Ardila \(1988\)](#) suggested that FAS may be an “aphasic epiphenomenon” that listeners detect when residual speech difficulties in an aphasic individual align with characteristics of authentic foreign accents. For similar reasons, others have suggested that some cases of FAS may be mild forms or a subtype of AOS ([Duffy, 2005](#); [Whiteside & Varley, 1998](#)).

Numerous cases in the FAS literature are described as having evolved from nonfluent aphasia and/or AOS, or as including residual symptoms of these disorders ([Ardila et al., 1988](#); [Christoph et al., 2004](#); [Graff-Radford, Cooper, Colsher, & Damasio, 1986](#); [Ingram et al., 1992](#); [Varley et al., 2006](#)). In one case the foreign accent evolved to AOS during recovery from a left hemisphere stroke ([Mariën et al., 2006](#)). Unlike these cases, DW had no history of nonfluent aphasia, reduced phrase length, difficulty with grammatical formulation, or speech that in any way sounded effortful or hesitant. Although DW reported occasional difficulties coming up with words, there was no mention of anomia in her medical records. During the interviews in our clinic, her sentence formulation was consistently fluent, and there were no verbal paraphasias, circumlocutions, excessive use of imprecise word choices, or unusual word retrieval pauses. On formal language testing, her performance was normal with the possible exception of reading comprehension, where her scores inconsistently were in the impaired range on some subtests.

Some features of DW’s speech were similar to AOS. For example, at the segmental level, her speech was characterized by numerous consonant and vowel changes. Some of these led to perceived sound substitutions (e.g., i/l, r/w, t/θ, g/k) and others were perceptually and acoustically ambiguous (e.g., distorted /r/, prolonged vowels, unclear voicing). Prosodically, errors in stress assignment and a tendency in the oral reading task to equalize stress were also at least superficially similar to prosodic characteristics of AOS. However, many other features of DW’s speech were uncharacteristic of AOS. Individuals with AOS typically have marked changes in speaking rate and fluency and they characteristically self-correct, but DW’s conversational speaking rate and fluency were normal. She had no initiation difficulties, no restarts or revisions, and no attempts to self-correct her utterances. Some of her vowels were prolonged, but there were no perceptible prolongations of consonants or inter-syllabic pauses. DW’s prosodic changes were not limited to problems with stress assignment and rate, but prominently included unusual intonation patterns that at times gave the impression of a “sing-song” quality and at other times simply appeared unusual, features that are not associated with AOS. Finally, whereas individuals with AOS experience particular difficulty with multi-syllabic utterances and alternation among different places of articulation, DW did not have more difficulty with these tasks than with monosyllabic words and her production of speech SMRs was comparable to her production of AMRs.

In some published cases of FAS, there were indications that the accented speech evolved from a dysarthric or slurred quality ([Berthier, Ruiz, Massone, Starkstein, & Leiguarda, 1991](#); [Blumstein et al., 1987](#); [Coughlan, Lawson, & O’Neill, 2004](#); [Dankovičová et al., 2001](#); [Fridriksson et al., 2005](#); [Graff-Radford et al., 1986](#)). In other cases, the foreign accent continued to be accompanied by dysarthric sounding speech ([Bakker, Apeldoorn, & Metz, 2004](#); [Kurowski et al., 1996](#)). Based on DW’s history, it appears that her initial speech changes may have included mild dysarthria. When she first sought medical attention, the physician observed that her speech had a slurred quality with abnormal cadence and noted that the left side of her face was weak. Another physician mentioned the possibility of Bell’s palsy. Four months later, when we saw her, the speech and structural functional examinations were normal. At this time, unlike in most dysarthrias, there were no abnormalities in respiratory, phonatory, or velopharyngeal subsystems. Furthermore, the consonant and vowel changes DW produced never gave the impression of articulatory imprecision or incoordination, but rather of unusual sound substitution patterns or allophonic variations.

3.1.2. *Phonetic characteristics*

Because DW made a full recovery from her acquired foreign accent, we were able to repeat our initial examination and thereby examine in detail the phonetic qualities of her speech with and without the accent. The validity of the phonetic profile was supported by excellent agreement between

perceptual and acoustic levels of analysis. The speech pattern that emerged was similar to those described for other cases of FAS, including those with documented left hemisphere focal pathology. Thus, there was a combination of unusual patterns in prosody and allophonic or phonemic changes in the production of both consonants and vowels. Virtually all phonetic changes during DW's period of foreign accent have been described in the FAS literature. Among the most consistently reported are changes in vowel quality and duration (Ardila et al., 1988; Blumstein et al., 1987; Ingram et al., 1992; Kurowski et al., 1996; Laures-Gore et al., 2006; Lippert-Gruener, Weinert, Greisbach, & Wedekind, 2005; Scott, Clegg, Rudge, & Burgess, 2006). Unusual VOT patterns or errors in prevocalic stop voicing are also common (Blumstein et al., 1987; Gurd et al., 1988; Miller et al., 2006; Scott et al., 2006), and there are many other mentions of monophthongization of vowel diphthongs, liquid /r/ and /l/ distortions, and stopping of interdental fricatives (Gurd et al., 1988; Scott et al., 2006; Varley et al., 2006). We conclude, therefore, that the speech patterns associated with an acquired foreign accent can be similar for cases with varying etiologies, including those with and without organic brain pathology.

A recurring question in the FAS literature is what qualities of the speech give listeners the impression of being "foreign," rather than disordered. Our phonetic analyses of DW's speech provide some insight. In addition to having DW's normal speech pattern for comparison, we were able to observe two distinct speech changes during the course of her disorder, one that sounded like an uncomplicated foreign accent and one that also had a childish or disordered quality. Blumstein and colleagues have suggested that the speech changes that occur in FAS do not sound disordered because they are consistent with potential attributes of natural language, whereas neurogenic speech disorders, such as AOS, nonfluent aphasia, and dysarthria are associated with changes that violate natural language variations (Blumstein et al., 1987; Blumstein & Kurowski, 2006). Consistent with this hypothesis, most of the changes that occurred in DW's speech during the period of foreign accent, such as the merging of tense and lax vowels, the shortening of stop consonant VOT, and the absence of alveolar stop consonants, interdental fricatives, and dark /l/, are all found in natural languages and would be expected in native speakers of such languages who learned English later in life. Unusual intonation contours and stress patterns are, similarly, consistent with the application of prosodic rules from a different language system. In contrast, the elevated pitch, strained phonation, and stiff articulation that were observed during DW's relapse are not in the realm of natural language variations and would therefore have contributed to the perception of abnormal or unusual, rather than accented speech. The other prominent feature during the relapse was the consistent w/r substitutions. Because this substitution pattern is not typical of speakers who are unfamiliar with the English /r/, but a very common developmental phenomenon in children who are native speakers of English, it is likely to have contributed to the impression of a childish, rather than foreign, speech pronunciation.

3.2. *Neurological differential diagnosis*

3.2.1. *More common organic conditions*

Some of the most common organic disorders associated with episodic and variable neurological symptoms are: TIA or stroke, multiple sclerosis (MS), partial complex seizures, and complicated migraine. Stroke symptoms are typically sudden in onset like those DW described when in the shower, and stroke commonly causes sensory symptoms and weakness on one side of the body as well as speech changes. Right hemisphere strokes, in particular, can cause left-sided weakness and speech disorders such as dysarthria and aprosodia. Initial clinical tests did not support the diagnosis of stroke, however, and several episodes of subsequent acute changes in her neurological status were not associated with any structural brain changes. The possibility that she was having TIAs without structural sequelae also seemed untenable because the duration of her symptoms was longer than in TIA and should have resulted in structural correlates.

Sudden onset of symptoms and episodic recurrence of symptoms can occur with MS and episodes can be triggered in the presence of heat such as in a shower (Uhthoff's phenomenon). The possibility of MS was ruled out because despite repeated episodes DW never had the expected inflammatory brain lesions. Furthermore, there was no evidence of an inflammatory process in analysis of the CSF.

Sudden onset changes in sensation, vision, hearing, and/or motor function can also occur during partial complex seizures but EEG obtained at a time when DW was symptomatic did not show

epileptiform activity. DW's more extended history also included events affecting different sides of her body and lacked the stereotypy common in seizure disorders.

Complex migraine can be associated with alteration in auditory perception, vision, sensation, speech and motor function. During DW's acute exacerbations, care providers consistently asked about headache, presumably considering this possibility, but this was repeatedly denied as a prominent symptom. Note too that some of her symptoms, e.g., shaking of the extremities, and "crushing pain" in her hands waking her out of sleep, are not symptoms associated with migraine. In many cases of complex migraine, especially when neurological problems last more than 24 h, there are also structural changes seen on MRI.

3.2.2. *Less common organic conditions*

Less common entities that can cause recurrent and variable neurological symptoms with "stroke-like" episodes include mitochondrial diseases such as MELAS, and the rare genetic disorder, CADASIL (cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy). Both of these conditions result in structural changes on brain imaging. Rheumatological disorders including Behcet's disease and lupus can produce episodic symptoms. The most common symptoms in neuro-Behcet's disease include visual disturbances, ataxia, and encephalopathy. DW's visual disturbance was more intermittent than that seen in Behcet's and she did not have ataxia or encephalopathy. Lupus flares are typically longer lasting than DW's episodes and if central neurological signs are present they are often associated with inflammation in the CNS.

Episodic sensory symptoms and weakness can occur with porphyria, but attacks are often associated with abdominal pain, and sensory or motor findings are more likely to be symmetrical. They also typically do not recur as frequently as DW experienced. Lyme disease, sarcoidosis and syphilis can affect many parts of the nervous system and have an extended course. None of these would be expected to suddenly remit completely, and they are less likely to present with simultaneous onset of symptoms in multiple neurological domains. We can rule out an episodic recurrent movement disorder with some confidence as these are not typically associated with sensory symptoms which were prominent in DW's case. Recurrent metabolic disorders are another possibility, but are usually associated with encephalopathy. Guillain Barre can present with pain, paresthesias, cranial nerve findings, and gait difficulties, but these patients have diminished reflexes. Arnold Chiari malformations can cause intermittent brain stem impairment and speech disorders associated with dizziness and gait difficulty, as well as pain and paresthesias, but DW's brain imaging showed no evidence of this anatomical abnormality.

3.3. *Medical and neurological history in relation to conversion disorder*

It is important *not* to overlook the possibility that DW's condition might represent a recurrent episodic disorder of organic etiology. Based on a thorough consideration of alternative diagnoses, including conditions associated with more complex neurological symptomatology, however, an organic etiology for her FAS seems unlikely. Rather, there are a number of features of DW's history suggestive of conversion disorder. First, she had some notable reports of experiences in her history that could not be explained by organic pathophysiology. One example was her report that she had woken up on one occasion and not been able to move or speak for 2 h. Narcolepsy patients can have the experience of being awakened and not being able to move or speak, but these episodes typical last only a few minutes and occur in the context of symptoms suggestive of narcolepsy which she did not have. Perhaps the strongest evidence for conversion disorder in her case is the fact that she had a wide spectrum of different types of symptoms. Symptoms affected one side of her body and then the other, and problems ranged from shaking, walking difficulty, paresthesias, numbness, weakness, vision problems, hearing problems, and speaking problems. All of these problems occurred for months in a waxing and waning course which then completely remitted leaving no neurological sequelae or structural imprint.

Other features of her case that support a diagnosis of a conversion disorder include an affect consistent with *la belle indifférence*. She reported that it was people around her who became more concerned about her problems, and it was they, rather than she, who frequently suggested that she

seek evaluation. DW worked in an ancillary medical profession, and individuals with this type of experience have been reported to have a greater likelihood of developing a conversion disorder than those with less exposure to medical disease, although this fact alone would not be cause for suspicion for conversion disorder.

It is also of interest that her speech during symptom worsening, in addition to the foreign accent, had a childish quality, both in terms of prosody, voice quality, and segmental substitution patterns. Childlike speech patterns, or infantile speech, can be seen in various psychogenic disorders, and is not characteristic of neurogenic pathology.

3.4. Establishing the diagnosis of conversion disorder

Establishing the psychogenic nature of any neurological disorder must be approached with great care, as a significant proportion of patients (from 4 to 30%) initially diagnosed with a psychogenic disorder are later found to have an organic illness to account for their symptoms (Couprie, Wijdicks, Rooijmans, & van Gijn, 1995; Stone et al., 2005).

Fahn and Williams (1988) developed a classification scheme for assigning patients with neurological movement disorders to one of four levels of certainty with regard to their likelihood of having a psychogenic etiology. The criteria from this classification scheme can be used to help identify the features that are particularly likely to establish that someone has a psychogenic disorder. Based on these criteria our case can be classified as a “documented” psychogenic disorder because of her resolution of speech symptoms during short therapy sessions (which did not persist after the sessions), and her later development of abrupt and complete resolution of symptoms. She also meets criteria for having a clinically established psychogenic disorder because she had: (1) symptoms that were incongruent with organic disease as discussed in her case above, (2) symptoms that were inconsistent over time, and (3) suffered from multiple somatizations.

Whenever considering the possible diagnosis of psychogenicity, it is important to remember that as many as 28% of patients with psychogenic neurological disorders also have organic neurological illness of some type (even if this does not account for the psychogenic symptoms). A good example of this occurs in patients with non-epileptic psychogenic pseudoseizures, 10–37% of whom can have true seizures and epilepsy (Krumholz & Niedermeyer, 1983). The phenomenon of conversion symptoms occurring in association with the same organ affected by an organic process has been referred to as “somatic compliance” (Sapir & Aronson, 1990). Conversion disorders affecting movement have been reported to occur with increased frequency following minor trauma or insult. Thus, even when a diagnosis of conversion disorders is quite certain, it is still necessary to consider other organic processes or injuries that might be affecting the patient. In DW's case, it is possible that she had a true organic facial palsy and that this condition triggered her subsequent symptoms. Although DW did not display the facial asymmetry signs that would be definitively indicative of an organic facial palsy (i.e., she did not have the inability to close the eye on the affected side and Bell's phenomenon), she may have had a partial Bell's palsy. Bell's palsy can appear suddenly after a single night's sleep, can be incomplete, and may be associated with mild dysarthria. We have also observed cases of Bell's palsy that have been associated with subsequent non-organic hemibody weakness, hemibody numbness, and sometimes unusual prosodic speech changes.

3.5. Conclusions

The details of our case and the availability of strong data for analysis allowed more definitive diagnosis of a psychogenic origin for FAS than has been possible in previous reports.

Of published cases with acquired foreign accent, only a few had indicators of psychogenic etiology or no evidence of brain lesions (Gurd et al., 2001; Poulin et al., 2007; Reeves et al., 2007; Reeves & Norton, 2001; Van Borsel et al., 2005). To our knowledge, only one previous case of acquired foreign accent was definitively diagnosed as a conversion disorder. Verhoeven, Mariën, Engelborghs, D'Haenen, and De Deyn (2005) described a 51-year-old Dutch woman who started using a French accent after a “near” traffic accident that resulted in no physical damage. Like in our patient, the neurological and neuroradiological exams were normal except for a “bizarre” gait. Unlike our patient, the woman had

extensive prior experience with the specific accent she acquired, having worked for several years as a teacher of Dutch at a French company. Furthermore, her foreign accent was accompanied by code-switching, with occasional French words and grammatical errors typical of native French speakers. Similar features have been reported in a handful of cases (e.g., Gurd et al., 2001; Reeves & Norton, 2001; Roth, Fink, Cherney, & Hall, 1997; Ryalls & Whiteside, 2006; Seliger, Abrams, & Horton, 1992). In contrast, our patient's speech profile, like the majority of FAS cases, was generically foreign in nature, unrelated to previous accent exposure, and restricted to pronunciation changes. As detailed in this report, phonetic analyses showed a general pattern of segmental and prosodic changes similar to previous FAS cases.

Given the similarity of the speech profile to previous FAS cases, we suggest that a psychogenic etiology should be considered as a possibility in other cases where a patient suddenly begins to speak with a foreign accent. Despite superficial similarities among patients, there are likely clinically important differences in need of exploration. To treat such individuals successfully, it is essential that clinicians accurately diagnose the nature of the disorder and consider medical, psychological and speech modification interventions. Hopefully, future case studies of FAS will include medical and social history, neurological presentation, clinical course, and treatment response that delineate variations critical to differential diagnosis and clinical management.

References

- Albert, M. A., Haley, K. L., & Helm-Estabrooks, N. Foreign accent syndrome: clinical and neuroanatomical analysis, in preparation.
- Ardila, A., Rosselli, M., & Ardila, A. (1988). Foreign accent: an aphasic epiphenomenon? *Aphasiology*, 2, 493–499.
- Bakker, J. I., Apeldoorn, S., & Metz, L. M. (2004). Foreign accent syndrome in a patient with multiple sclerosis. *The Canadian Journal of Neurological Sciences*, 31, 271–272.
- Benton, A. L., Hamsher, K., & Sivan, A. B. (2001). *Multilingual aphasia examination* (4th ed.). Lutz, FL: Psychological Assessment Resources.
- Berthier, M., Ruiz, A., Massone, M. I., Starkstein, S. E., & Leiguarda, R. C. (1991). Foreign accent syndrome: behavioral and anatomical findings in recovered and non-recovered patients. *Aphasiology*, 5, 129–147.
- Blumstein, S. E., Alexander, M. P., Ryalls, J. H., Katz, W., & Dworetzky, B. (1987). The nature of the foreign accent syndrome: a case study. *Brain and Language*, 31, 215–244.
- Blumstein, S. E., & Kurovski, K. (2006). The foreign accent syndrome: a perspective. *Journal of Neurolinguistics*, 19, 346–355.
- Christoph, D. H., de Freitas, G. R., dos Santos, D. P., Lima, M. A. S. D., Araujo, A. Q. C., & Carota, A. (2004). Different perceived foreign accents in one patient after prerolandic hematoma. *European Neurology*, 52, 198–201.
- Coelho, C. A., & Robb, M. P. (2001). Acoustic analysis of foreign accent syndrome: an examination of three explanatory models. *Journal of Medical Speech – Language Pathology*, 9(4), 227–242.
- Coughlan, T., Lawson, S., & O'Neill, D. (2004). French without tears? Foreign accent syndrome. *Journal of the Royal Society of Medicine*, 97, 242–243.
- Couprie, W., Wijdicks, E. F., Rooijmans, H. G., & van Gijn, J. (1995). Outcome in conversion disorder: a follow up study. *Journal of Neurology, Neurosurgery, and Psychiatry*, 58, 750–752.
- Dankovićová, J., Gurd, J. M., Marshall, J. C., MacMahon, M. K. C., Stuart-Smith, J., Coleman, J. S., et al. (2001). Aspects of non-native pronunciation in a case of altered accent following stroke (foreign accent syndrome). *Clinical Linguistics and Phonetics*, 15(3), 195–218.
- Duffy, J. R. (2005). *Motor speech disorders: Substrates, differential diagnosis, and management*. St. Louis, MO: Elsevier-Mosby.
- Fahn, S., & Williams, D. T. (1988). Psychogenic dystonia. *Advances in Neurology*, 50, 431–455.
- Fridriksson, J., Ryalls, J., Borden, C., Morgan, P. S., George, M. S., & Baylis, G. C. (2005). Brain damage and cortical compensation in foreign accent syndrome. *Neurocase*, 11, 319–324.
- Goodglass, H., Kaplan, E., & Barresi, B. (2001). *Boston diagnostic aphasia examination* (3rd ed.). Philadelphia: Lippincott, Williams & Wilkins.
- Graff-Radford, N. R., Cooper, W. E., Colsher, P. L., & Damasio, A. R. (1986). An unlearned foreign “accent” in a patient with aphasia. *Brain and Language*, 28, 86–94.
- Gurd, J. M., Bessel, N. J., Bladon, R. A. W., & Bamford, J. M. (1988). A case of foreign accent syndrome. *Neuropsychologia*, 26, 237–251.
- Gurd, J. M., Coleman, J. S., Costello, A., & Marshall, J. C. (2001). Organic or functional? A new case of foreign accent syndrome. *Cortex*, 37, 715–718.
- Hagiwara, R. (1997). Dialect variation and formant frequency: the American English vowels revisited. *Journal of the Acoustical Society of America*, 102(1), 655–658.
- Helm-Estabrooks, N. (2001). *Cognitive linguistic quick test*. San Antonio, TX: Psychological Corporation.
- Ingram, J. C., McCormack, P. F., & Kennedy, M. (1992). Phonetic analysis of a case of foreign accent syndrome. *Journal of Phonetics*, 20, 457–474.
- Kaplan, E., Goodglass, H., & Weintraub, S. (2001). *Boston naming test* (2nd ed.). Philadelphia: Lippincott, Williams & Wilkins.
- Krumholz, A., & Niedermeyer, E. (1983). Psychogenic seizures: a clinical study with follow-up data. *Neurology*, 33, 498–502.
- Kurovski, K. M., Blumstein, S. E., & Alexander, M. (1996). The foreign accent syndrome: a reconsideration. *Brain and Language*, 54, 1–25.

- Lahiri, A., Gwirth, L., & Blumstein, S. E. (1984). A reconsideration of acoustic invariance for place of articulation in diffuse stop consonants: evidence from a cross-language study. *Journal of the Acoustical Society of America*, 76, 391–404.
- Laures-Gore, J., Henson, J. C., Weismer, G., & Rambow, M. (2006). Two cases of foreign accent syndrome: an acoustic-phonetic description. *Clinical Linguistics and Phonetics*, 20, 781–790.
- Lippert-Gruener, M., Weinert, U., Greisbach, T., & Wedekind, C. (2005). Foreign accent syndrome following traumatic brain injury. *Brain Injury*, 19(11), 955–968.
- Mariën, P., Verhoeven, J., Engelborghs, S., Rooker, S., Pickut, B. A., & DeDeyn, P. P. (2006). A role for the cerebellum in motor speech planning: evidence from foreign accent syndrome. *Clinical Neurology and Neurosurgery*, 108, 518–522.
- Miller, N., Lowit, A., & O'Sullivan, H. (2006). What makes acquired foreign accent syndrome foreign? *Journal of Neurolinguistics*, 19(5), 385–409.
- Poulin, S., Macoir, J., Paquet, N., Fossard, M., & Gagnon, L. (2007). Psychogenic or neurogenic origin of agrammatism and foreign accent syndrome in a bipolar patient: a case report. *Annals of General Psychiatry*, 6, 1–7.
- Reeves, R. R., Burke, R. S., & Parker, J. D. (2007). Characteristics of psychotic patients with foreign accent syndrome. *Journal of Neuropsychiatry and Clinical Neuroscience*, 19, 70–76.
- Reeves, R. R., & Norton, J. W. (2001). Foreign accent-like syndrome during psychotic exacerbations. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, 14(2), 135–138.
- Roth, E. J., Fink, K., Cherney, L. R., & Hall, K. D. (1997). Reversion to a previously learned foreign accent after stroke. *Archives of Physical Medicine and Rehabilitation*, 78, 550–552.
- Ryalls, J., & Whiteside, J. (2006). An atypical case of foreign accent syndrome. *Clinical Linguistics and Phonetics*, 20, 157–162.
- Sapir, S., & Aronson, A. E. (1990). The relationships between psychopathology and speech and language disorders in neurologic patients. *Journal of Speech and Hearing Disorders*, 55, 503–509.
- Scott, S. K., Clegg, F., Rudge, P., & Burgess, P. (2006). Foreign accent syndrome, speech rhythm and the functional neuroanatomy of speech production. *Journal of Neurolinguistics*, 19(5), 370–384.
- Seliger, G. M., Abrams, G. M., & Horton, A. (1992). Irish brogue after stroke. *Stroke*, 23, 1655–1656.
- Sikorshi, L. D. (2005). Foreign accents: suggested competencies for improving communication pronunciation. *Seminars in Speech and Language*, 26(2), 126–130.
- Stevens, K. N., & Blumstein, S. E. (1978). Invariant cues for place of articulation in stop consonants. *Journal of the Acoustical Society of America*, 64, 1358–1368.
- Stone, J., Smyth, R., Carson, A., Lewis, S., Prescott, R., Warlow, C., et al. (2005). Systematic review of misdiagnosis of conversion symptoms and hysteria. *British Medical Journal*, 331, 989–991.
- Van Borsel, J., Janssens, L., & Santens, P. (2005). Foreign accent syndrome: an organic disorder? *Journal of Communication Disorders*, 38, 421–429.
- Varley, R., Whiteside, S., Hammill, C., & Cooper, K. (2006). Phases in speech encoding and foreign accent syndrome. *Journal of Neurolinguistics*, 19(5), 356–369.
- Verhoeven, J., Mariën, P., Engelborghs, S., D'Haenen, H., & De Deyn, P. (2005). A foreign speech accent in a case of conversion disorder. *Behavioral Neurology*, 16, 225–232.
- Whiteside, S. P., & Varley, R. A. (1998). A reconceptualisation of apraxia of speech: a synthesis of evidence. *Cortex*, 34, 221–231.