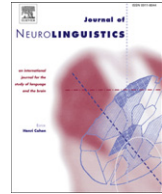




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A case of foreign accent syndrome: Acoustic analyses and an empirical test of accent perception

Raageen Kanjee^a, Scott Watter^a, Alexandre Sévigny^b, Karin R. Humphreys^{a,*}

^aDepartment of Psychology, Neuroscience & Behaviour, McMaster University, Canada

^bDepartment of Communication Studies & Multimedia McMaster University, Canada

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ABSTRACT

Foreign Accent Syndrome (FAS) is a rare acquired syndrome following stroke, manifesting as a perceived change in the speaker's accent. We present acoustic-phonetic analyses of the speech of a patient, RD, with FAS presenting as an apparent accent shift from Southern Ontarian to Atlantic Canadian who was first described in Naidoo, Warriner, Oczkowski, Sévigny, and Humphreys (2008). As well as more fully documenting this case, this paper also seeks to examine whether the accompanying articulatory deficits constitute a mild form of some other motor speech disorder such as apraxia of speech (AOS) or dysarthria. Acoustic-phonetic analyses showed increased vowel formant variability, overlap of voice onset times for phonemically contrastive stops, inconsistent consonant distortions, and global prosodic attenuation. These, combined with the patient's fluent speech, can account for the perception of a foreign accent as opposed to disordered speech. The observed inconsistency of consonant distortions coupled with other articulatory deficits suggest that at least in this case, FAS can be considered to be a mild form of AOS.

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* Corresponding author. Department of Psychology, Neuroscience & Behaviour, McMaster University, 1280 Main St West, Hamilton, Ontario, L8S 4K1, Canada. Tel.: +1 905 525 9140x23011; fax: +1 905 529 6225.

E-mail addresses: raageen.kanje@gmail.com (R. Kanjee), watter@mcmaster.ca (S. Watter), sevigny@mcmaster.ca (A. Sévigny), krh@mcmaster.ca (K.R. Humphreys).

1. Introduction

1.1. Background

Foreign Accent Syndrome (FAS) is an acquired condition, defined as an inability to produce speech under the phonological and phonetic parameters normal to one's native accent (Blumstein, Alexander, Ryalls, Katz, & Dworetzky, 1987; Dankovičová et al., 2001; Gurd, Bessell, Bladon, & Bamford, 1988; Katz, Garst, & Levitt, 2008; Laures-Gore, Henson, Weismer, & Rambow, 2006; Whitaker, 1982). The accompanying segmental, prosodic, and rhythmic abnormalities often lead listeners to perceive the individual's speech as foreign (Blumstein et al., 1987; Coelho & Robb, 2001). This impression may be reinforced by an accompanying agrammatism or anomia that has been reported in some cases (Blumstein et al., 1987). Etiologically, FAS occurs following brain injury such as stroke (Moen, 1990; Scott, Clegg, Rudge, & Burgess, 2006), multiple sclerosis (Bakker, Apeldoorn, & Metz, 2004), or traumatic brain injury (Lippert-Gruener, Weinert, Greisbach, & Wedekind, 2005).

FAS is a rare pathology, with the majority of reports being published in the last twenty years (Coleman & Gurd, 2006; Dankovičová et al., 2001). One of the first systematic studies was carried out by Monrad-Krohn (1947), involving a Norwegian woman who had acquired a German accent, which was clearly identifiable because of displacement of her vocalic triangle (/l/becomes/i/, /æ/becomes/a/), as well as changes to her pitch accent, indicative of the way in which Germans pronounce Norwegian (Dankovičová et al., 2001). The accent changes reported in the literature have been many and varied—for instance, an English patient sounding French (Gurd et al., 1988), a Norwegian with an English accent (Moen, 1990), and an American sounding Chinese, Dutch, or Canadian (Laures-Gore et al., 2006). This final example provides a key point regarding FAS: different listeners may perceive a particular speaker as possessing different accents. This pattern holds true for both naïve listeners and trained phoneticians (Blumstein & Kurowski, 2006). Blumstein et al. (1987) proposed to explain this by characterizing the accent acquired by patients as a 'generic' foreign accent. That is to say, their speech falls generally within the bounds of what is phonetically permissible (Varley, Whiteside, Hammill, & Cooper, 2006), but outside of the parameters of their local dialect (Kurowski, Blumstein, & Alexander, 1996). Although whether or not a 'generic' foreign accent is acquired has been briefly discussed in follow-up studies (e.g. Miller, Lowit, & O'Sullivan, 2006), the grounds for its justification remain speculative at present.

As previously noted, FAS is a pathology resulting from brain damage, most commonly stroke (Coelho & Robb, 2001). However, there has yet to emerge a consistent pattern of lesions; infarcts to numerous brain areas in both the left and right hemispheres, cortically and subcortically, have all led to the same symptomatology (Blumstein & Kurowski, 2006; Coelho & Robb, 2001; Laures-Gore et al., 2006). It has been suggested that this variation may be better understood by considering damage to functional neural systems rather than specific foci (Kurowski et al., 1996), particularly areas of motor cortex (and potentially its subcortical projections) involved in speech production (Blumstein & Kurowski, 2006). This hypothesis has found some initial support from a study by Fridriksson et al. (2005), who demonstrated cortical compensation following basal ganglia and speech system damage. Given that only a small subset of current cases have investigated their patients' lesion locations in detail (Blumstein & Kurowski, 2006), any neurological conclusions reached can be qualified as tentative at best.

1.2. Explaining foreignness

A question of primary interest to the FAS literature is why patients' speech is perceived as foreign, and not disordered (Ingram, 1992; Miller et al., 2006; Moen, 2000; Varley et al., 2006). For instance, Ingram (1992) had listeners rate the intelligibility, disorderedness, and foreignness of pre- and post-morbid speech samples. Although post-morbid speech was perceived as neither less intelligible, nor more disordered than (presumably) normal pre-morbid speech, it was rated as sounding significantly more foreign.

Numerous aspects of speech, both segmental and suprasegmental, have been implicated in an attempt to resolve this puzzling situation. Alterations in prosody have been reported since as early as Monrad-Krohn, 1947, with a wide range of distortions noted, including vowel epenthesis (frequently schwas, /ə/) (Blumstein et al., 1987; Ingram, 1992; Katz et al., 2008; Miller et al., 2006; Varley et al.,

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