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Foreign Accent Syndrome: a typological overview

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Introduction

Foreign Accent Syndrome (FAS) is a motor speech disorder which causes patients to speak their native language with an accent different from speakers belonging to the same language community: the patient lacks the ability to make the phonemic and phonetic contrasts of his/her native language and demonstrates suprasegmental alterations which cause listeners to perceive the accent as distinctly ‘foreign’. In 1982, Whitaker proposed 4 diagnostic criteria for FAS: 1) the accent is considered by the patient, acquaintances and investigators as foreign, 2) it is unlike the patient’s accent before the insult, 3) the accent is clearly related to central nervous system damage, 4) there is no evidence in the patient’s background of him/her being a speaker of a foreign language (pp. 197- 198). Although FAS of “acquired neurogenic origin” is the most common variant of the disorder, there exists a “developmental”, “psychogenic” and “mixed variant” as well (Verhoeven and Mariën, 2010).

In FAS of *neurogenic* origin the foreign accent is incited by a lesion affecting the central nervous system, often a stroke or brain trauma. However, FAS has also been attested in relation to MS (Villaverde-Gonzalez et al., 2003), tumor (Masao et al., 2011; Tomasino et al., 2013; Abel et al., 2009), as well as other pathologies affecting the CNS. *Developmental* FAS can be regarded as a subtype of neurogenic FAS; only here it is developmental in nature, affecting speech as it develops. In the *psychogenic* variant, a psychological/psychiatric disorder incites FAS, whereas the *mixed* variant is originally neurogenic in nature, but the accent change has such a profound effect on the patient’s psychological status that he/she internalizes it by further developing the accent in order ‘to create a more “believable” personality’ (Verhoeven and Mariën 2010, p. 600)

Methods

We examined the over 100 published authentic case studies stretching a period from 1907 to 2013 in order to present a general overview of the typology of FAS. The three main subtypes – *neurogenic* (with inclusion of the developmental variant), *psychogenic* and *mixed* FAS – are exemplified and compared by means of three illustrative case studies, including two new ones: a case of psychogenic FAS and developmental FAS.

RC is a 41-year-old, right-handed, monolingual English-speaking woman from the UK who started talking with a Polish/Croatian accent after a long period of migraine. JD is a 17-year-old, right-handed, monolingual Dutch-speaking Belgian boy who developed FAS in the context of developmental articulation disorder: childhood apraxia of speech. Both patients received a complete neuroradiological, neurological, neurocognitive and neurolinguistic work-up, which allows to further digress on the diagnosis, pathological substrate(s), comorbid speech- and language disorders, as well as the segmental and suprasegmental characteristics associated with FAS.

Results

Analysis of the literature reveals that 83% of the published cases ($n=89$) presented neurogenic FAS, whereas 12% of the patients ($n=13$) developed psychogenic FAS. Only 2% of the cases ($n=2$) match 'mixed FAS'. For three cases (3%) authors did not state a clear aetiology ($n=3$). With respect to the diagnosis, the criteria presented by Whitaker (1982) only hold for cases of *neurogenic* FAS. Whitaker's last criterion, concerning the 'second language history' of patients, seems to be outdated in a largely multilingual society. We know of FAS patients whom gave proof of a '*reversed accent*' (e.g. Roth et al., 1997; Verhoeven and Mariën, 2010; Levy et al., 2011).

Neurogenic FAS often occurs as a result of a stroke (87%, $n= 77$), or a brain trauma (13%, $n= 12$) commonly affecting the left prerolandic motor cortex, frontal motor association cortex or striatum of the language-dominant hemisphere (Lewis et al., 2013; Verhoeven and Mariën, 2010; Dankovičová et al., 2001). In psychogenic FAS, the motor speech disorder is often incited by a conversion disorder (Verhoeven et al., 2005; Jones et al., 2011; Haley et al., 2010; Tsuruga et al., 2008; $n= 4$), schizophrenia (Reeves and Norton, 2001; Reeves et al., 2007; $n=2$), bipolar disorder (Poulin et al. 2007; Reeves et al., 2007; $n=2$), mania (Lewis et al., 2013; $n= 1$) or an obsessive-compulsive disorder (Polak et al., 2013; $n=2$).

C had an unremarkable medical history. She underwent structural imaging (MRI), as well as a Tc- 99m-ECD SPECT, which both appeared normal. Neurocognitive work-up revealed that she had a normal IQ and normal neuropsychological profile. Clinical psychological investigations through administration of Dimensional Assessment of Personality Pathology-Basic Questionnaire (DAPP-BQ; Livesley and Jackson, 2009), Minnesota Multiphasic Personality Inventory-II (MMPI-2; Butcher et al., 1989) and Defense Style Questionnaire-60 (DSQ-60; Trijsburg et al., 2003) did not disclose any clinical syndrome (DSM-IV-TR Axis I) or personality disorder (DSM-IV-TR Axis II) that could be objectively asserted with enough certainty, although conversion disorder was suspected: four out of the six DSM-IV-TR criteria for conversion disorder were met (APA, 2000). The patient mentioned that she suffered from migraine attacks, which aggravated her accented speech and severely diminished speech fluency, as well as intonation. She did not present any supplementary comorbid speech or language disorders, such as dysarthria (e.g. Graff-Radford et al., 1986; Monrad-Krohn, 1947; Berthier et al., 1991), aphasia (e.g. Ingram et al., 1992; Ardila et al., 1988; Gurd et al., 1988) or apraxia of speech (e.g. Ingram et al., 1992; Aronson 1980) as often found in neurogenic patients, which further corroborated the hypothesis of a psychogenic origin.

JD's medical history was also unremarkable. Except for his articulatory development, all

developmental milestones were acquired within the accepted timespan. JD received speech- language therapy as of the age of five, and had always presented with a French accent. He underwent EEG, MRI and Tc-99m-ECD SPECT, but no abnormalities were detected. Neurocognitive work-up demonstrated verbal and performance IQ levels within the normal range. During neurolinguistic exam, JD gave proof of a deviant pronunciation, characterized by wrong accent placement (words), omissions and substitutions of consonants, and phonematic errors. Based on these clinical observations JD was diagnosed with a verbal dyspraxia.

Discussion

In this contribution we describe recently acknowledged FAS typology by means of two representative cases: a patient with psychogenic FAS and an example of developmental FAS. Our study provides evidence that the FAS criteria proposed by Whitaker are questionable and in need of review. Secondly, clinical evidence was found to confirm that FAS may occur on a developmental basis (Mariën et al., 2009). JD indeed presented FAS in a context of a developmental motor speech disorder, namely childhood apraxia of speech, as was determined after a clinical neurolinguistic investigation. As such, this case plays an interesting part in the on-going debate relating to the semiological resemblances between FAS and AoS; a hypothesis that has been supported by several researchers (Whiteside and Varley, 1998; Fridriksson et al., 2005; Mariën et al., 2009), but has also been contested (Pyun et al., 2013; Blumstein and Kurowski, 2006). For the case of RC the linguistic data, course and outcome of accent were highly indicative of a psychogenic aetiology, regardless of the fact that psychological testing could not lead to the diagnosis of a 'demonstrable' psychological/language disorder.

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