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Effects of regiolects on the perception of developmental foreign accent syndrome

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ABSTRACT

Foreign accent syndrome (FAS) is a relatively rare speech motor disorder in which the pronunciation of an affected speaker is perceived as distinctly foreign by listeners of the same language community. Because of various close semiological resemblances with apraxia of speech, FAS has been hypothesized to be an apraxia subtype. In 2009 two cases of developmental FAS (dFAS) were described in whom the disorder was detected in an early stage of their speech-language development in the absence of brain damage or mental illness. In the present study, two listening panels consisting of 30 native speakers of two regiolects, Dutch and Flemish, evaluate the spontaneous speech of two native Flemish-speaking boys with suspected dFAS, three native Flemish-speaking children diagnosed with developmental apraxia of speech (dAoS), two bilingual children (L1 = Flemish, L2 = French or English), and six native Flemish-speaking children with typical speech-language development. Whereas the Dutch panellists were not able to distinguish the different groups, the Flemish listeners accurately identified the children with dFAS and the bilingual children. None of the listeners were able to discern between dFAS and dAoS. The latter finding supports the assumption that the two speech disorders not only share similar semiological and perceptual characteristics but also a common pathophysiological substrate. Although it is not always identified by listeners of the same language community but is by speakers of the same regiolect, in addition to FAS resulting from brain damage or a psychological disorder, dFAS appears a distinct form of apraxia of speech resulting from developmental deficits.

1. Introduction

1.1. Description

Foreign accent syndrome (FAS) is a relatively rare speech motor disorder in which the pronunciation of the affected speaker is perceived as distinctly foreign by listeners of the same language community. Monrad-Krohn (1947) considered FAS to be a suprasegmental disorder of linguistic prosody, i.e. manifesting in an unfamiliar tone, rhythm, timing, and intonation, giving the speech output, though syntactically and idiomatically correct and comprehensible, an unusual and foreign quality. Still, same-language speakers are often not able to identify the exact geographical location of the accent (Fridriksson et al., 2005).

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The French neurologist Pierre Marie (1907) was the first to report on the phenomenon, describing a French-speaking patient from Paris who, after recovering from anarthria due to a lesion in the subcortical left hemisphere, spoke with an Alsatian accent. After Marie's brief communication, a handful of descriptions of similar observations followed. It was not until 1982 that the first diagnostic criteria for FAS were proposed by Whitaker: 1) the accent is deemed foreign by the patient, the patient's relatives, and examiners/researchers; 2) the patient had a different accent before the injury, 3) the accent can clearly be linked to damage to the central nervous system, and 4) there is no reason for the patient to have the accent based on his background. Various descriptions of the characteristics of FAS speech are available. The majority of studies mention segmental issues, such as adaptations of vowel length, substitutions, omissions of phonemes, and suprasegmental issues such as unusual stress in words or sentences (Poulin, Macoir, Paquet, Fossard, & Gagnon, 2007).

FAS mostly occurs in combination with other speech or language disorders, as is the case with many language impairments. Describing 25 FAS patients, Aronson (1990) found 70% to also suffer from apraxia of speech (AoS), aphasia, or dysarthria.

1.2. FAS or AoS?

Even after more than a hundred years of multidisciplinary research, we still lack a coherent model of deficient speech that distinguishes FAS from AoS. As the two disorders share speech characteristics, various researchers (e.g. Fridriksson et al., 2005; Mariën, Verhoeven, Wackenier, Engelborghs, & De Deyn, 2009; Whiteside & Varley, 1998) posited FAS to be a mild or specific form of AoS caused by a disruption of speech motor planning processes. AoS is a neuromuscular speech disorder in which the precision and consistency of speech movements are affected due to an inability to properly and smoothly convert phonological knowledge to verbal motor commands (Rosenbek, 1999). Based on their acoustic analysis of two recovered FAS cases, Perkins, Ryalls, Carson, and Whiteside (2010) concluded that FAS and AoS share overt similarities. In both conditions, speakers clearly substitute consonants and vowels rather than producing unclear, disrupted speech, as is the case in, among other speech disorders, dysarthria. Moreover, in both types of affected speech same-language listening panels perceive the accent as non-native.

1.3. Aetiology

Whitaker (1982) described FAS as an acquired neurological speech disorder following demonstrable brain damage, which is indeed the case in 83% of adult cases (Keulen, Verhoeven, Bastiaanse, & Mariën, 2014). Although the exact pathophysiological mechanisms subserving FAS remain to be elucidated, several brain areas are often implicated in FAS. Most FAS patients recorded in the literature developed FAS due to focal vascular damage (stroke) in the perisylvian speech area, the frontal motor cortex, or the striatum of the language-dominant hemisphere (Dankovicova et al., 2001). However, various other aetiologies have also been proposed, including head trauma (Edwards, Patel, & Pople, 2005), brain tumours (Buentello-García, Martínez-Rosas, Cisneros-Franco, & Alonso-Vanegas, 2011; Tomasino et al., 2013), multiple sclerosis (Villaverde-Gonzáles, Fernández-Villalba, Moreno-Excribano, Alias-Linares, & Garciá- Santos, 2003), epilepsy (Cole, 1971), and dementia (Paolini et al., 2013). As the speech motor symptoms characterising FAS strongly reflect disrupted articulatory planning and coordination, the cerebellum has been suggested to be involved in the pathophysiology of both FAS and AoS (Cole, 1971; Mariën & Verhoeven, 2007; Monrad-Krohn, 1947; Whitaker, 1982; Whitty, 1964). Mariën et al. (2009) added that the cerebello-cerebral network may also be implicated in articulatory planning and thus possibly in both FAS and AoS.

Apart from a variety of acquired neurological aetiologies, Verhoeven and Mariën (2010) distinguished three other possible causes of FAS. Firstly, in several cases a psychogenic cause was suspected to lie at the core of the speech problems (psychogenic FAS). After close inquiry no (visible) brain damage or any organic brain disease could be objectified, with strong indications of a psychological or psychiatric disorder remaining (Haley, Roth, Helm-Estabrooks, & Thiessen, 2010; Tailby, Fankhauser, Josev, Saling, & Jackson, 2013; Van Borsel, Janssens, & Santens, 2005; Verhoeven, Mariën, Engelborghs, D'Haenen, & De Deyn, 2005). Secondly, both Ryalls and Whiteside (2006) and Verhoeven and Mariën (2010) described a combined variant of neurogenic origin (mixed FAS). In each case the woman concerned had acquired the new accent after brain injury but had subsequently perfected it to boost her personal credibility. Thirdly, and though rare but the most relevant for the present study, FAS has also been documented as a developmental speech disorder in children.

Only a very limited number of studies have reported on the brain areas involved in developmental apraxia of speech (dAoS). Although structural changes are often only visible at a (sub-) microscopic level, Liégeois and Morgan (2012) in their review report that mainly the basal ganglia, perisylvian and rolandic cortices bilaterally as well as the cerebellum are involved in the genetic-idiopathic variant. However, dAoS may equally develop in association with (rolandic) epilectic disorders (Horn et al., 2010; Scheffer, 2000) and in association with metabolic disorders (galactosemia) (Shriberg, Potter & Strand, 2011; Liégeois & Morgan, 2012). In developmental FAS, the link with a clear neurobiological substrate is, much as is the case for dAoS, less evident. Developmental foreign accent syndrome (dFAS) has not been associated with structural deficits at the supratentorial or infratentorial level (Keulen et al., 2016; Mariën et al., 2009). However, a SPECT study performed by Keulen et al. (2016) demonstrated a hypoperfusion at the level of the left inferior frontal region, the bilateral medial prefrontal regions, the bilateral temporal regions and the right cerebellum. The affected regions hence correspond quite well to the brain regions inducing apraxic-ataxic-like speech in patients with acquired brain damage.

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