



“Accent issue”: foreign accent syndrome following ischemic stroke

Vincenzo Di Stefano¹ · Antonella Maria Pia De Novellis¹ · Fedele Dono¹ · Marco Onofri¹ · Maria Vittoria De Angelis²

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Abstract

Background Foreign accent syndrome (FAS) is a rare syndrome associated with altered speech rhythm and prosody, which listeners perceive as foreign; cerebrovascular accidents, tumors and multiple sclerosis are reported as possible causes of FAS. The pathophysiology of FAS is not yet understood.

Case presentation A 68-year-old Italian man was admitted to the Emergency Department for non-fluent aphasia and dysarthria. Computed tomography (CT) scan did not show abnormalities; the patient was treated with systemic thrombolysis. A repeated brain CT and magnetic resonance imaging (MRI) confirmed an infarct in the left primary motor cortex and mild extension to cortico-subcortical frontal regions. In the following days he gradually improved, speaking Italian fluently with a typical German accent. In conclusion, FAS is a rare motor speech disorder, often related to cerebrovascular accidents involving critical regions in the dominant hemisphere. In addition, the present case adds further evidence to the role of the left primary motor cortex in modulation of prosody. In rare cases FAS can be the only sign of stroke or can appear after recovery from post-stroke aphasia.

Keywords Foreign accent syndrome · Stroke · Prosody · Speech disorders · Aphasia

Background

Foreign accent syndrome (FAS) is a rare acquired speech disorder characterized by changes or alterations in rhythm and prosody [1, 2]. Since its first description in 1907 by the French neurologist Pierre Marie [1], the interest about FAS has increased in the last decades, even though the underlying mechanisms remain unknown. In 1982, Whitaker coined the term “Foreign Accent Syndrome,” establishing specific diagnostic criteria [3].

Patients with FAS usually speak their native language with a non-native accent, which listeners perceive as foreign in the absence of any previous personal or family exposure to the foreign accent [4].

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✉ Vincenzo Di Stefano
vincenzo19689@gmail.com

¹ Department of Neuroscience, Imaging and Clinical Sciences, University “G. d’Annunzio” of Chieti-Pescara, Via dei Vestini, 66013 Chieti, Italy

² Department of Neurology, SS Annunziata Hospital, Chieti, Italy

FAS is usually caused by severe brain injury, such as cerebrovascular accidents, tumors, multiple sclerosis, neurodegenerative, and psychiatric diseases [5–9].

However, strokes involving the language-dominant hemisphere represent the most frequent cause of FAS [10].

As reported in Table 1, several sites of lesion in the left hemisphere had been reported in FAS patients, including cortical and subcortical regions, but the left basal ganglia appear to be the most common anatomic region involved [11].

Nevertheless, “functional” FAS has also been described in patients affected by psychiatric disorders in association with functional movement disorders [12]. In these rare cases, no organic cause can be found, but the variability of the accent during examination and the absence of a brain lesion in the dominant hemisphere support the diagnosis.

Alexander et al. described three phases in FAS progression: (1) the acute phase, when neurobiological symptoms and signs appear; (2) the lesion phase (3 weeks up to 3 months after stroke), when language disturbances disappear, without a sufficient compensation [13], and (3) the chronic phase (3 months post stroke), when FAS severity usually decreases due to spontaneous or post-therapeutic functional mechanisms [11]. All cases of FAS caused by an ischemic stroke can be found in Online resource 1 and Table 2 [4, 10, 14–44]. Hereby, we present the first case of an Italian patient developing FAS

Table 1 Overview of the most common site of lesion in the left hemisphere, which may cause FAS in patients reported in literature

Site of lesion in the left hemisphere (number of cases)	Authors and year of publication
Precentral gyrus (9)	Berthier 1991 (4 patients), Takayama 1993, Marien 2007, Bhandari 2011, Nishida 2011, Sakurai 2015
Postcentral gyrus (2)	Marien 2007, Karanasios 2011
Premotor cortex (5)	Graff-Radford 1986, Berthier 1991 (4 patients)
Inferior frontal gyrus (2)	Marien 2007, Sakurai 2015
Frontal operculum (2)	Moreno-Torres 2013, Munson 2005
Parietal lobe (9)	Blumstein 1987, Kurowski 1996, Kwon 2006, Marien 2007, Cohen 2009, Bhandari 2011, Karanasios 2011, Levy 2011, Roy 2012
Temporal lobe (5)	Blumstein 1987, Kwon 2006, Marien 2007, Karanasios 2011, Levy 2011
Broca's area (1)	Ardila 1988
Corpus callosum (2)	Hall 2003, Naidoo 2008
Internal capsule (10)	Damasio 1982, Graff-Radford 1986, Blumstein 1987, Gurd 1988, Kurowski 1996, Fridriksson 2005, Naidoo 2008, Coughlan 2004, Ryalls 2006, Scott 2006
Basal ganglia (11)	Damasio 1982, Gurd 1988, Seliger 1992, Kurowski 1996, Edwards 2005, Fridriksson 2005, Varley 2006, Marien 2007, Naidoo 2008, Levy 2011, Berthier 2015
Corona radiata (2)	Avila 2004, Tetsuo 2002
Pons (3)	Fridriksson 2005, Dankovicová 2011, Tran 2013
Insula (2)	Scott 2006, Moreno-Torres 2013

after an ischemic stroke involving the left primary motor cortex extended to cortico-subcortical frontal regions.

Case description

A 68-year-old right-handed man was admitted to our Emergency Unit for an acute episode characterized by difficulty speaking, with the inability to pronounce sentences and dysarthria. Neurological examination revealed motor aphasia and dysarthria, without sensory-motor deficits. Mental status was fully intact. Aphasia rapid test (ART) [45] confirmed motor aphasia. A National Institutes of Health Stroke Scale (NIHSS) score of 3 (9: 2; 10: 1) was estimated [46]. His past medical history revealed hypertension and mitralic valve surgery. Computed tomography (CT) scan was unremarkable at clinical onset. However, 24-h CT scan showed a subacute infarction, confirmed by brain magnetic resonance imaging (MRI), which demonstrated a left primary motor cortex infarct extended to cortico-subcortical left semiovale centrum (Fig. 1). In the acute setting, the patient was treated with systemic thrombolysis in accordance with current guidelines with a clinical benefit (NIHSS score of 0) [47]. Neurological examination showed a full recovery from aphasia with the appearance of a patient-referred new unrecognized “German accent.” He was aware of the acquired new accent and his relatives also confirmed that there was a change in his prosody. According to current diagnostic criteria, FAS was diagnosed [3]. One week from clinical onset, the Aachenner aphasia test (AAT) was within normal limits [48]. The patient underwent diagnostic workup for ischemic stroke, including cardiac rhythm monitoring, which detected atrial fibrillation. Anticoagulant therapy was prescribed and he was discharged at home with close follow-up. Further 6-month

and 12-month follow-up showed clinical stability with no aphasia or dysarthria, but persisting slight FAS. Online resource 2 shows FAS feature during reading, singing, and spontaneous speech after a month from stroke. Online resource 3 shows patient's speech features 1 year before stroke.

Discussion

We report the case of an Italian patient who developed a typical German accent after an ischemic stroke in the left primary motor cortex, extended to the cortico-subcortical region of the centrum semiovale.

At the moment, there is no complete model explaining the pathophysiology of FAS. It is still a matter of debate if FAS results from distorted articulatory planning and coordination processes or “hic et simpliciter” from the acquisition of a new accent [28, 49]. A further hypothesis foresees that FAS, dysarthria, and motor aphasia might establish a continuum among language disturbances [43].

Other authors hypothesized that the new accent comes after a long neural suppression due to a vascular accident with neural plasticity effects [2, 11, 21]. This theory finds and confirms the case by Pick, in which there was a previous exposure to the accent during the war [2] and in the case by Seliger, in which the patient was exposed to the accent in the childhood through his relatives [21]. In other words, the patient retains the accent, which has been encountered before, until a stroke makes it emerge as a “FAS.” In “polyglot cases,” FAS patients can be considered to have reverted to a previously learned accent.

Table 2 FAS cases in the premotor and precentral cortex reported in literature

Authors, year, age and gender	Site of lesion	Original accent	Acquired accent	Associated symptoms and signs
N. R. Graff-Radford et al., 1986 [15] 56 F	Left premotor cortex	American English (Midwestern)	Nordic	Mild pronator drift and impaired rapid alternating movements of right arm.
S. E. Blumstein et al., 1987 [16] 62 F	Left precentral cortex and subcortical white matter	American English (Boston)	Slavic (or generally European)	Right central facial paresis, mild clumsiness of right hand, increased right-sided reflexes.
M. L. Berthier et al., 1991 [19] 70 F	Left precentral cortex	Spanish (Buenos Aires)	Slavic	2-day episode of mutism, before FAS
M. L. Berthier et al., 1991 [19] 58 M	Left subcortical white matter of precentral cortex	Spanish	Slavic	Mutism, severe left faciobrachio-crural paresis with spared sensation and crossed aphemia.
M. L. Berthier et al., 1991 [19] 47 F	Left premotor cortex	Spanish (Buenos Aires)	Hungarian (Slavic)	Right hemiplegia.
M. L. Berthier et al., 1991 [19] 34 F	Left premotor cortex	Spanish	Slavic	Sudden severe headache at onset. Meningeal signs and a complete right third cranial nerve palsy.
J. Takayama et al., 1993 [22] 44 F	Left precentral cortex	Japanese	Korean	No data.
J. M. Gurd et al., 2001 [24] 47 F	Left inferior frontal corona radiata	English (North Yorkshire)	French	Headache at onset and impaired gait during FAS.
C. Avila et al., 2004 [26] 51 F	Left inferior frontal corona radiata	Spanish polyglot (French, English, and Catalan)	French	Right hemiparesis.
S. K. Scott et al., 2006 [32] 54 F	Left white matter of precentral cortex	Scottish	German, Polish, or South African	Aphasia at onset and right arm hemiparesis.
P. Marien and J. Verhoeven, 2007 [33] 53 F	Left precentral and inferior frontal cortices	Dutch	French or German	Moderate right hemiparesis and central facial nerve palsy.
H. S. Bhandari, 2011 [36] 55 M	Left premotor cortex	American (Texas)	Cockney (dialect of London)	FAS reversed after a tonic-clonic generalized seizure.
J. P. Roy et al., 2012 [41] 63 F	Left precentral cortex	Quebec French (Canada)	Alsatian or German	None.
Y. Sakurai et al., 2015 [44] 42 F	Left precentral and premotor cortices	Japanese	Chinese	Slight right hemiparesis and mild Wernicke's aphasia, after second stroke.

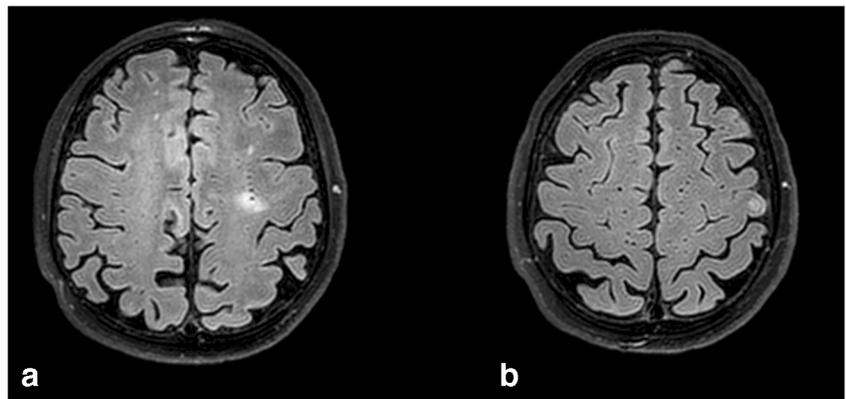
FAS, foreign accent syndrome

In Fig. 2, we report the list of the native languages (2a) and the percentage of acquired accents (2b) of patients with FAS described in literature. According to Pick and Selinger's theory, the acquired accent in FAS should reflect the exposure to most common languages in the world. On the contrary, the incidence of cases of FAS seems to involve people whose languages are not widely spoken. In fact, Slavic, German, and French accents account for more than 70% of FAS reported in literature (Fig. 2b). Although there are millions of people in the world that speak English as a second language, FAS cases with English accent account for less than 20% of all cases.

Moreover, it is difficult to define what we mean with the term "polyglot" because of different levels of exposure to the second or third language. In our case, the patient was an Italian who received formal language (English) instruction at school; he had not studied German and never lived or had a holiday in Germany. He developed a German accent in contrast with the previous exposure to English.

It is believed that linguistic and phonetic aspects can influence the expression of FAS. Probably, several factors affect the type of acquired accent; moreover, FAS is underdiagnosed

Fig. 1 MRI of the brain shows hyperintense signal in the premotor and precentral cortex in FLAIR sequences (a, b). MRI, magnetic resonance imaging; FLAIR, fluid-attenuated inversion recovery

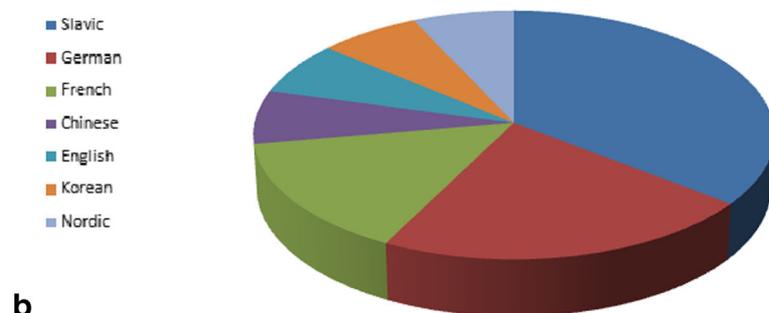
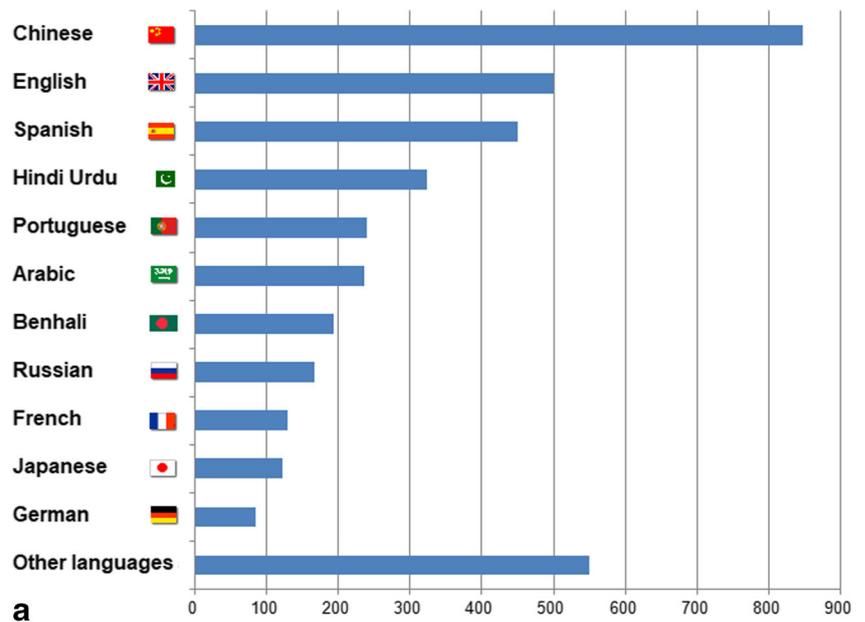


in many countries, thus explaining underreporting of this rare condition.

In clinical practice, hardly ever FAS represents an isolated sign, being more often associated with co-existing language disturbances. FAS is more frequent in women than in men and has been linked to frontal executive dysfunctions [11]. There are empirical evidences that primary motor and primary somato-sensory areas are responsible of initiation and

planning of speech and control intonation and articulation of the voice [50, 51]. The cortico-striato-thalamo-cortical network connects different structures including the putamen, thalamus, and premotor and sensory- motor cortices with bulbar nuclei, which allow lip, tongue, and laryngeal movements [52]: disruption of this pathway can cause an important disconnection between planning and production of speech, resulting in FAS [53].

Fig. 2 a The most common languages spoken in the world for millions of mother tongue people are listed. **b** The most common acquired accents in patients with FAS in literature



Probably, there is not a single region responsible for total speech control of intonation and accent, but most of patients present a lesion involving basal ganglia or premotor and precentral areas (Tables 1 and 2) [15, 17, 19, 22, 33, 36, 40, 42, 44, 49, 54, 55].

Recovery from FAS can be different from a patient to another: every patient should undergo a complete assessment of language skills with appropriate instruments and a follow-up of months and years post stroke, to detect all language improvements and to build the best speech therapies over time [56]. Moreover, FAS recovery mainly depends on the site of lesion [11]; paradoxical facilitation has been reported after cerebellar hemorrhage in patients with FAS [35, 43]. It has been hypothesized that maladaptive interactions between the two cerebral hemispheres could control rhythm and prosody and cerebellum can modulate such interhemispheric imbalance. Further evidence for a role of the cerebellum in motor speech planning emerges from functional neuroimaging [33].

Of interest, in contrast with previous literature [19], we observed that FAS was not present in our patient while singing (Online resource 2). Functional MRI studies described activation in the fronto-parietal areas and cerebellum during singing tasks [57, 58]. This finding corroborates the hypothesis, which attributes an important role to the cerebellum in modulation of patterns of speech in FAS patients with lesions in the left precentral cortex.

Finally, nowadays, many people have bi- or multi-language history, thus arising difficulties in meeting Whitaker's criteria. This issue arises important limitations on the application of criteria for FAS diagnosis. Most of the people, learning new languages, tend to retain their native accent, which may cause alteration of prosody [59], so FAS might only be due to pathological language mixing and clinicians should be aware of this aspect.

Conclusion

In conclusion, foreign accent syndrome is a rare motor speech disorder, often related to cerebrovascular accidents involving critical regions in the dominant hemisphere. Although many cases have been described, FAS remains a poorly understood condition.

FAS can be the only sign of stroke or more often occurs in association with neurological signs and symptoms. In our patient, FAS was induced by interruption of cortical-subcortical pathways that connect the precentral and premotor areas to the "language network." Lesions affecting regions that are critical for speech production may disrupt neural processes involved in the production of accent features. As a result of impaired speech regulation, other nodes of the motor speech network provide compensation by increasing their activities.

Clinicians should be aware that FAS could appear after recovery from aphasic acute phase of a stroke. In these patients, a complete and systematic neurological evaluation should be performed in order to achieve the correct diagnosis and to obtain the best outcome.

The present case adds further evidence to the role of the left primary motor cortex, especially in the modulation of prosody, although future research is needed to clarify the neurological, linguistic, and cognitive basis of FAS.

Contributors VD, FD, AMP D, and MVD provided clinical care to the patient. VD and AMP D drafted the first version of the manuscript, and all authors contributed to and have approved the final version of the manuscript.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical standards All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informant consent Obtained.

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