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**Functional Foreign Accent Syndrome in suspected Conversion Disorder: a case study**

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**Abstract**

**Background and objectives**

We provide a case analysis for a 28-year-old, native Dutch-speaking lady who developed Foreign Accent Syndrome (FAS), a few weeks after falling down the staircase. In addition to FAS, which gave the impression she spoke with a German accent, German(-like) words and structures occurred. Speech symptoms were aggravated by increased stress, fatigue or emotional pressure, and this triggered jargon speech. It was hypothesized her FAS and jargon developed on a functional basis.

**Methods**

An in-depth analysis of the patient's medical background, neuropsychological and neurolinguistic tests and psychodiagnostic exams were done. The patient participated in an fMRI experiment. In a syllable repetition paradigm, motor speech activations were compared to those of healthy individuals, to see whether they were altered, which would be expected in case of a neurological etiology.

**Results**

Medical history disclosed prior traumatic experiences for which she sought help, but no neurological incidents. Repeated neuropsychological and neurolinguistic tests showed deficits in recent memory and executive functioning. The patient demonstrated great difficulties with picture naming. Clinically, language switching and mixing as well as recurring jargon speech was found. Formal psychodiagnostic tests did not identify a clear disorder, but psychodiagnostic interviews were consistent with a DSM-5 conversion disorder. The fMRI study demonstrated that speech network activations corresponded to those found in healthy participants.

**Conclusion**

The clinical neurolinguistic characteristics, outcome of the fMRI experiment, together with the clinical psychodiagnostic findings were strongly indicative for an underlying functional etiology for the FAS and jargon speech, presenting as symptoms of conversion disorder.

**Keywords:** foreign accent syndrome, FAS, language mixing, language switching, jargon, fMRI, speech, accent, functional disorder, conversion disorder.

80 **1. Introduction**

81

82 Healthy polyglot individuals automatically use the correct language depending on the  
83 communicative/pragmatic context. Language or code mixing (e.g. within utterance  
84 mixing of different languages) and switching (i.e. between languages or codes in  
85 alternating utterances) can occur naturally when speakers are in the process of acquiring  
86 a new language, such as in young bilingual or polyglot children (1, 2), but also in  
87 adolescent and adult language learners, in the course of the acquisition process, as  
88 mentioned by Duran (3). In all these cases, language mixing and switching can be  
89 consequence of failed neural suppression of the language that was not targeted. In terms  
90 of choice for one or the other language in bilinguals, Abutalebi and Green (4) use the term  
91 "lexical competition" to refer to an ongoing "competition" between the L1 and L2, and  
92 the related control phenomena that are decisive in the selection process. They (see also  
93 Abutalebi and Perani (5)), as well as Hernandez (6) mention that the efficiency of  
94 inhibition mechanisms depends on several factors including the degree of proficiency,  
95 age of acquisition and computational load of the languages. They are hypothesized to  
96 influence the strength of the induced plastic changes in the neural networks in the brain  
97 subserving for instance cognitive control mechanisms, including language inhibition.  
98 Failed inhibition in language learners will not be interpreted as "pathological": the errors  
99 can be explained within the dynamic framework of the above-mentioned parameters.

100 Pathological language mixing and switching typically refers to the process of  
101 blended language use in bilingual or polyglot aphasic patients (7,8,9). Some researchers  
102 have argued that the basal ganglia play a crucial role in both language switching and  
103 mixing (8,10), whereas others have argued that switching is mainly subserved by frontal  
104 circuits (11,12) and mixing by the temporo-parietal lobe (11).

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105           Although typically described in the context of aphasia (e.g. 8), pathological  
106 language mixing and switching has been observed in a handful of cases with Foreign  
107 Accent Syndrome (FAS). One example is a 51-year-old woman with conversion disorder  
108 described by Verhoeven et al. (13) who switched between Dutch and French irregularly.  
109 Her Dutch lexicon, syntax and the pragmatics of her native tongue were all influenced by  
110 French. For instance, word order was French instead of Dutch. The case described by  
111 Reeves and Norton (14) (see also Reeves et al. (15)) suffered schizophrenia, and the  
112 patient switched from American English to a distinguished, formal register in *British*  
113 English whenever exacerbations were uncontrolled due to withdrawal from medication.  
114 In these cases, pathological language mixing and switching occurred within the context  
115 of an underlying psychiatric disorder. Nevertheless, these patients remained intelligible.  
116 So far, there has only been one report, by Gurnani and Horwitz (16), of a case in which a  
117 39-year-old woman produced “non-sensical speech” in the context of a FAS (the acquired  
118 accent was qualified as “Ukrainian”, although the mother tongue was not reported).

119           Jargon speech is usually associated with acquired aphasia (e.g. 17,18). Rohrer et  
120 al. (18) state jargon can in that case be semantic (language devoid of content), phonemic  
121 (production of words phonemically related to the target) or neologistic in nature (words  
122 and nonwords, semantically and phonemically not related to the target word). In these  
123 cases, the jargon aphasia has been suggested to be caused by a resource limitation deficit,  
124 a comprehension deficit, a feedback or production deficit, or a verbal monitoring deficit  
125 depending on the specific case presentation (19). In contrast to "*schizophasia*" or the  
126 "meaningless mixture of words and phrases characteristic of advanced schizophrenia"  
127 (20, p. 201) that can wax and wane, patients suffering from jargon aphasia retain  
128 persistent expressive problems (18). The language disorder does not fluctuate and the  
129 neurological symptoms will demonstrate a more linear resolution in case of remission.

130 Here we present a detailed work-up of 28-year-old female patient who presented with a  
131 sudden change of accent, which from time-to-time evolved to an unintelligible  
132 phonological jargon, in the absence of structural neurological damage.

133

## 134 **2. Case study**

### 135 ***2.1. Medical history***

136

137 The patient is a 28-year-old, monolingual female native speaker of Dutch from  
138 the Netherlands, with monolingual Dutch-speaking parents and an educational level of  
139 13 years. At school, she studied English for five years, received one year of French and  
140 two years of German language instruction. She worked as an ancillary nurse in a hospital.  
141 Her medical antecedents consisted of asthma and migraine. There was no indication of  
142 alcohol or drug abuse. She had previously experienced two miscarriages and was in the  
143 16<sup>th</sup> week of her first pregnancy when she developed FAS a few weeks after falling down  
144 a staircase (June, 2013). The fall did not impact the health of her child. Her proxies  
145 perceived her as a non-native speaker of Dutch, initially with a Turkish or Moroccan  
146 accent, later Belgian Dutch, the Dutch variant spoken in the northern part of Belgium.

147 Clinical neurological examination, laboratory tests and neuroradiological (MRI)  
148 investigations carried out after the fall were normal. A series of psychosocial stress factors  
149 were noted: the patient mentioned persistent emotional difficulties as a child after the  
150 death of her mother from a brain hemorrhage. She admitted to have suffered from this  
151 early loss for which she had been treated by a medical psychologist for a suspected post-  
152 traumatic stress disorder after her mother's death but could not remember the duration.

153 The patient consulted the hospital in November 2014, some six months after the  
154 fall, and complained about headaches, tension, stress, disoriented thoughts and behavior  
155 as well as attention difficulties. She mentioned that her accent was often perceived as

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156 Belgian Dutch, French or German. Occasionally, language mixing occurred (German),  
157 especially when she was stressed and she “could not cope with the large number of  
158 external stimuli”. The impact of stressors was very clear during the intake interview.  
159 When speech-language therapists entered the consultation room to attend the session, the  
160 patient’s speech deteriorated significantly and FAS symptoms aggravated. She developed  
161 a Belgian Dutch accent, then started to mix German and Dutch words and expressions,  
162 e.g. "*Danke, danke sehr, dokter*" ("Thanks, many thanks, doctor").

163 She claimed that she had had no regular contact with speakers of Belgian Dutch.  
164 However, her sister-in-law was German. Following the accent change, she developed  
165 loose thoughts and visual hallucinations which lasted for an unspecified number of weeks.  
166 She mentioned that she had also suffered from severely disoriented behavior which arose  
167 for the first time approximately two weeks after the fall. This distraught behavior  
168 continued throughout her pregnancy, and according to the patient, it was the result of  
169 increased stress and severe anxiety which she related to the death of her mother at a young  
170 age. The patient’s husband volunteered the same information in a separate interview.  
171 These behaviors were highly egodystonic and provoked a fear of insanity, also referred  
172 to in the literature as “phrenophobia” (21). The patient mentioned that she had not  
173 benefitted from previous psychological therapy and that she had never received  
174 medication to cope with her symptoms. At the time the patient visited the neurology  
175 department she was implicated in a disability litigation.

176

177 After the development of the current symptoms, she tried to continue her work but  
178 she could not concentrate long enough to stay in the profession. At the time of  
179 consultation, the patient parented her daughter (1,5 years old) and managed the  
180 household. She mentioned that she was easily over-aroused physically. During a last  
181 contact with the patient in April 2020, she mentioned that cognitive-behavioral therapy

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182 (CBT), acceptance and commitment therapy helped her, though not to such extent that  
 183 she had no more episodes of accent change or jargon, or that episodes were reduced in  
 184 duration. However, she felt she was more in control of her life by having learnt to set and  
 185 communicate her boundaries. The patient mentioned she now avoided crowded places,  
 186 and that the stress she experienced put pressure on her relationship.

187

188 **2.2. Neuropsychological investigations**

189 **Table 2.1.**

190 Overview of the neuropsychological test results of the patient at four instances.

191 Legend: BNT= Boston Naming Test (Kaplan et al., 1983); CAT-NL= Comprehensive  
 192 Aphasia Test-NL (Visch-Brink et al., 2014); Pc.= percentile; RBANS= Repeated Battery  
 193 for the Assessment of Neuropsychological Status (Randolph et al., 1998); RAVLT= Rey  
 194 Auditory Verbal Learning Test (Rey, 1964); SD= standard deviation; SS= Scaled Score,  
 195 TMT= Trail Making Test (Reitan, 1958); TOMM= Test of Memory Malingering  
 196 (Tombaugh, 1996); WAIS-IV-NL= Wechsler Adult Intelligence Scale IV, Dutch version  
 197 (Kooij and Dek, 2012); WCST= Wisconsin Card Sorting Test (Heaton et al., 2003);  
 198 WMS-R= Wechsler Memory Scale Revised (Wechsler, 1987)

199

Neurocognitive tests	03/11/2014	05/06/2015	24/4/2016	06/05/2016	MEAN (±1SD)
	(raw score or SS)	(raw score or SS)	(raw score or SS)	(raw score or SS)	
	SD or Pc	SD or Pc	SD or Pc	SD or Pc	
<b>Intelligence</b>					
<b>WAIS-IV-NL</b>					
Verbal comprehension	(83) -1.13 SD				100 (±15)
Perceptual reasoning	(85) - 1 SD				100 (±15)
Working memory	(92) - 0.53 SD			22	100 (±15)
Processing speed	(84) - 1.07 SD			76	100 (±15)
Total IQ	(81) - 1.27 SD				100 (±15)
<b>Memory</b>					
<b>WMS-R</b>					
Attention	(94) -0.4 SD	(87) -0.9 SD	(95) -0.3 SD		100 (±15)



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Visual memory	(84) -1.1 SD	(104) +0.3 SD	(109) +0.6 SD	100 (±15)
Verbal memory	(96) -0.3 SD	(76) -1.6 SD	(87) -0.9 SD	100 (±15)
General memory	(91) -0.6 SD	(82) -1.2 SD	(92) -0.5 SD	100 (±15)
Recent memory	(70) -2.0 SD	(76) -1.6 SD	(76) -1.6 SD	100 (±15)
<b>RBANS</b>				
Immediate memory	(83) -1.1 SD		(83) -1.1 SD	100 (±15)
Recent memory	(56) -2.9 SD		(52) -3.2 SD	100 (±15)
<b>RAVLT</b>				
Total learning score			(30) pc 1	
Recall			(2) pc 2	
Recognition			(22/30)	
<b>TOMM</b> (memory malingering)			48/50	
<b>Executive functioning and attention</b>				
TMT A	(29") pc 70		(36") pc 14	
TMT B	(42") pc >90		(67") pc 46	
Stroop test: Card 1	(48") pc 40		(66") pc 1	
Stroop test: Card 2	(55") pc 60		(79") pc 1	
Stroop test: Card 3	(80") pc 70		(107") pc 58	
d2-test				
Tn	(393) -1.2 SD		(546) +0.8 SD	
Tn-E	(371) -1.3 SD		(519) -0.79 SD	
CP	(146) -1.5 SD		(211) +0.05 SD	
WCST	(6) +0.38 SD			5.58 (±1.1)
RBANS	(97) -0.2 SD		(88) -0.8 SD	100 (±15)
Verbal fluency	(70) -2.0 SD			60.2 (±21.28)
<b>Language</b>				
BNT	(41/60) -3.19 SD	(44/60) -2.4 SD	(28/60) -6.7 SD	52.8 (±3.7)
RBANS	(108) +0.5 SD		(101) +0.1 SD	100 (±15)
<b>Visuospatial skills</b>				
RBANS	(100) ±0.0 SD		(96) -0.3 SD	100 (±15)
<b>Language</b>				
<b>Aachener Aphasia Test</b>				
<b>(Dutch)</b>				
Comprehension				
Auditory - Words	(24/30)			
Auditory - Sentences	(28/30)			
Reading - Words	(21/30)			
Reading - Sentences	(29/30)			

200

201 Legend: BNT= Boston Naming Test (22); CAT-NL= Comprehensive Aphasia Test-NL  
202 (23); Pc.= percentile; RBANS= Repeated Battery for the Assessment of  
203 Neuropsychological Status (24); RAVLT= Rey Auditory Verbal Learning Test (25); SD=  
204 standard deviation; SS= Scaled Score, TMT= Trail Making Test (26); TOMM= Test of  
205 Memory Malingering (27); WAIS-IV-NL= Wechsler Adult Intelligence Scale IV, Dutch  
206 version (28); WCST= Wisconsin Card Sorting Test (29); WMS-R= Wechsler Memory  
207 Scale Revised (30).

208

209 Overall, mnemonic capacities tested via the WMS-R remained stable over  
210 approximately 1,5 years, with relatively intact attention, and visual and verbal memory.  
211 Recent memory proved affected over three testing sessions. This was confirmed via  
212 testing on the R-BANS which showed severely impaired functioning. The patient scored  
213 remarkably worse on the TMT A & B and Stroop tasks (cards 1 to 3) during her fourth  
214 and last repeat neuropsychological investigation: whereas initially scores were largely  
215 within an acceptable range (except for Stroop card 2) in 2014, this was no longer the case  
216 in 2016. Scores on the d2-test (31), a measure of visual attention, concentration and  
217 inhibition, were (borderline) moderate in 2014. In terms of language assessment, visual  
218 naming (BNT) was severely impaired over three test sessions, with a very remarkable  
219 regression during the test session of April 2016, where she obtained only 28/60 (-6.7 SD).  
220 Errors (predominantly) consisted of neologistic (e.g. "escalator" was referred to as "wheel  
221 chair staircase"), semantic (e.g. "lion" instead of "sphinx") and visual (e.g. "branch"  
222 instead of "asparagus") mistakes. The CAT-NL revealed a weak performance on the oral  
223 picture description: grammar and syntax were severely impaired. During her last testing

224 session, issues were hence very specifically situated in the area of recent memory,  
225 attention, executive functioning, and naming and narrative skills.

226

### 227 *2.3. Psychodiagnostic examinations*

228

229 In December 2014, the patient underwent a psychodiagnostic assessment in the  
230 neurolinguistics and neuropsychology department of the hospital where she was initially  
231 seen as a part of the work-up. The tests consisted of the Minnesota Multiphasic  
232 Personality Inventory-2 (MMPI-2, 32), NEO-FFI (Neuroticism, Extraversion, Openness  
233 - Five Factor Inventory, 33), the Defense Style Questionnaire-60 (DSQ; 34), the  
234 Supernormality Scale (SS; 35), the Beck Depression Inventory-II (BDI; 36), the  
235 Depressive Experience Questionnaire (DEQ; 37), the List of Indeterminate  
236 Psychopathology (LIPP; 38), Rotter Incomplete Sentence Blanks (RISB; 39), as well as  
237 an exhaustive psychological examination (both individually and conjointly with her  
238 husband).

239 Her overall personality profile (MMPI, DSQ, DEQ) revealed a sensitive, people-  
240 pleasing, dependent personality without any indications for a psychotic structure or  
241 symptoms. On the contrary, there were more indications for a stable neurotic personality  
242 organization (40). The test results could not account for the psychotic behavior that had  
243 been noted in past. Malingering tests (the SS, LIPP and MMPI-2 validity indices)  
244 revealed a slight tendency for a naive positive self-presentation, but without significant  
245 effects on test results. The BDI-2, DSQ-60 and DEQ were presented twice (with a 2-week  
246 interval) to check for consistency in response patterns.

247 Individuals with similar profiles (Within Normal Limit, or "WNL"-profile with a  
248 3-6-0 pattern) are described as normal individuals, experiencing “transient situational  
249 distress” (41). Their mood is described as stable and happy (as reflected in a BDI-2 total

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250 score of 3 out 63). The patient did no longer mention any cognitive issues herself,  
251 although these were formally identified in neurocognitive testing. Although these code-  
252 types are associated with reported transient psychotic symptoms, it remains unclear  
253 whether her low scores warrant this association. The profiles did not suggest a strong  
254 potential for somatization or for a conversion disorder. Nevertheless, clear indications  
255 were found for a dependent personality. Through analysis of quantitative tests results,  
256 projective tests (RISB) and the clinical examination, the neurotic personality organization  
257 proved to be arguably immature.

258

259         It is important to mention that the test results did not allow a clear interpretation.  
260 The face-to-face examination, however, was more informative. During the first interview,  
261 the accent was subtle, only slightly noticeable when technical or peripheral topics were  
262 discussed, but when the conversation touched upon affective topics, her accent and her  
263 speech distorted rapidly. It changed back to normal when less emotional issues were  
264 discussed. During the interviews, she was pleasant, polite and very open. Although she  
265 did not recognize any psychopathology, suffering or mental pain at first, she (through a  
266 discussion with her partner) recognized her tendency to occult, repress and split affective  
267 experience roughly, and nearly dissociate when negative feelings became too threatening.  
268 She appeared interested in the assessment and remained hopeful about its results. When  
269 the feedback was provided (the same day) her foreign accent returned almost  
270 immediately. By changing the topic of the conversation and the number of individuals  
271 present during the conversation (her partner, staff) - and hence varying the emotional  
272 pressure, cognitive load and fatigue - quantitative distortions in her accent could be  
273 triggered quite easily until a jargon-like speech remained. It took significantly longer for  
274 the accent to disappear again.

275

276 In 2015, the patient was examined again. The psychiatric evaluation yielded no  
277 signs of a depressive disorder, anxiety disorder or obsessive-compulsive disorder, and no  
278 symptoms of a post-traumatic disorder or dissociative disorder were found. Additional  
279 information revealed a very strained relationship with her stepmother following the death  
280 of her mother. Psychosomatically, a change of accent was very noticeable: it ranged from  
281 Belgian Dutch to (flawed) German. This accent changed as a result of increased fatigue.  
282 Therefore, a diagnosis of FAS seemed appropriate at the time of psychiatric evaluation.  
283 Presentation was consistent with a DSM-5 (42) classification of “conversion disorder”  
284 with speech symptoms.

285

#### 286 *2.4. Phonetic description of speech*

287

288 We recorded two spontaneous speech samples: one with accented spontaneous  
289 speech and one with (phonological) jargon. There are various aspects of the patient’s  
290 speech which could account for the perception of a German accent. As far as the fricatives  
291 are concerned, the patient sometimes articulated the Dutch alveolar fricatives (/s/ and /z/) as postalveolar /ʃ/ (eg. [zwarə] “heavy” is pronounced as [ʃwa:rə], [slapə] “to sleep” is pronounced as [ʃla:pə]). The occurrence of the voiceless postalveolar fricative as the first element in word-initial consonant clusters (e.g. [ʃwa:rə]) and in word-final position (e.g. [ɔləʃ]) is a feature of German, not Dutch. Another factor contributing to the perception of a German accent is the devoicing of the labiodental fricative /v/ to /f/ (e.g. [fɛ:l], “much”), the alveolar fricative /z/ to /s/ (e.g. [sɛ:nə], “his”) and the velar fricative /ɣ/ to /x/ (e.g. [xəvɔlə], “to fall + past tense”). Although the devoicing of fricatives is very common in Belgian Dutch (43) this feature is consistent with the perception of a German foreign accent.

301 In terms of the vowels, there are two relevant observations. First, the patient often  
302 adds sjwa to the end of words and this may change the Dutch word into a German word  
303 (e.g. Dutch [zɔn] becomes German [zɔnə], “sun”). Secondly, it should be pointed out that  
304 the pronunciation of the diphthong [ɛɪ] has an onset which is too posterior and too open  
305 so that it sounds like [ɑɪ] (e.g. [mɛɪn] becomes [mɑɪn], “mine”). This no doubt also  
306 contributes to the perception of a German accent.

307

## 308 *2.5 Functional MRI*

309

310 To study the neurological substrates of the patient’s speech, functional MRI  
311 (fMRI) was carried out. The protocol was adapted from Brendel et al. (44) and consisted  
312 of one 30-minute run during which three conditions were presented in pseudo-  
313 randomized order: “pa-ta-ka” (=planning), “ta-ta-ta” (=no planning), and a visual control  
314 condition without articulation (=baseline). All three conditions consisted of a 2-second  
315 information screen showing which task should be performed (“pa-ta-ka”, “ta-ta-ta”, no  
316 articulation), a 2-second preparatory visual cue (black cross in the center of a white  
317 screen) and a 4-second window consisting of 10 flickering crosses at 2.5 Hz, or a 1.6-  
318 second window consisting of 4 flickering crosses at 2.5Hz. The patient was asked to  
319 repeat the syllables “ta-ta-ta” or “pa-ta-ka” synchronously with the flickering cross. The  
320 first flicker was the “GO”-signal. The stimuli were presented to the patient on a screen.  
321 The patient received articulation training before scanning in order to minimize  
322 (mandibular) movement artefacts. Stimuli were programmed in E-Prime (Psychology  
323 Software Tools, 2016).

324 fMRI analyses contrasted the long speech conditions (3 times “pa-ta-ka” and “ta-  
325 ta-ta”) with the visual control condition, analogous to the main analysis of Brendel et al.  
326 (44) (see appendix for acquisition specifications and analysis).

327

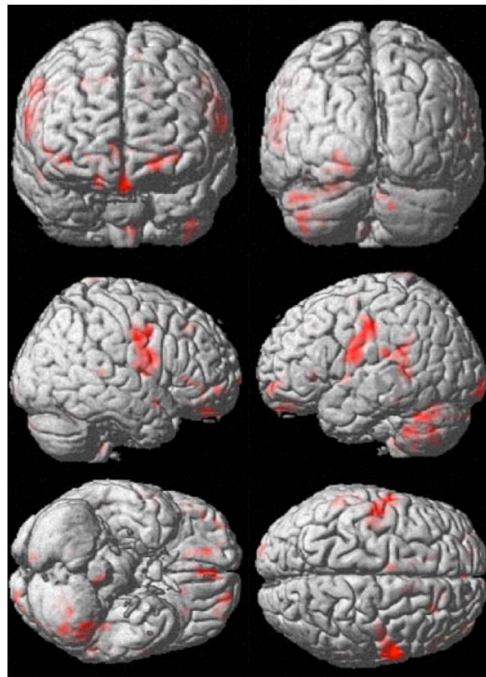
328 **Figure 2.1.**

329

330 Functional Magnetic Resonance Image (fMRI). Significant activation sites when LC >  
331 visual baseline. Significant clusters at uncorrected  $p < 0.001$  and #voxels  $> 10$ . All  
332 clusters with FWE-corrected  $p > 0.05$  and a peak with FWE-corrected  $p < 0.05$  are shown.

333 Activation sites can further be consulted in **Table 2.2.**

334



335

336

337

338 Results indicated no significant anomalies (see Figure 2.1). Table 2.2 lists the  
339 most important clusters found in our patient. Most of the clusters (in **bold**) were also  
340 found in the healthy participants of Brendel et al. (44). The most important clusters in the  
341 sensorimotor cortex (SMC) and the supplementary motor area (SMA) were activated,  
342 together with the left insula and the left superior temporal gyrus (STG). These areas are  
343 principally involved in motor planning and programming as well as motor preparedness  
344 (esp. SMA). Smaller clusters in for example the basal ganglia (essential structures

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345 mediating speech execution, as well as exerting a control function) were not reproduced,  
346 probably due to statistical constraints in a single-patient fMRI study. In the cerebellum,  
347 only left-sided activation was found in lobule VI, and in Crus I, an area that is more  
348 related to motor preparedness. In this patient, the activation extended also to lobule VIII,  
349 the second cerebellar sensorimotor area, and to Crus II, which is strongly associated to  
350 language processing.

351         Apart from a stronger left-sided lateralisation in the cerebellum, it seems the  
352 speech activations appeared to be very comparable to what was seen in the subjects  
353 without speech and language disorders who participated in the study of Brendel et al.  
354 Hence, this further confirmed the hypothesis that this patient developed her speech  
355 problems on a psychogenic basis.

356

### 357 **Table 2.2.**

358

359 Overview of activation sites in the speech network in the investigated patient during a  
360 syllable-repetition task. Activation sites represent LC > visual baseline. Significant  
361 clusters at uncorrected  $p < 0.001$  and #voxels > 10. All clusters with FWE-corrected  $p >$   
362 0.05 and a peak with FWE-corrected  $p < 0.05$  are listed.

363 Legend: <sup>1</sup>LCT corresponds in this case to the repetition of “pa-ta-ka” or “ta-ta-ta”  
364 synchronized with a visual stimulus; LCT = long click train, three times “pa-ta-ka” or  
365 “ta-ta-ta”; NCT = no click train, only the preparatory cue; SMA = supplementary motor  
366 area; R = right; L = left; SMC = sensorimotor cortex; IFG = inferior frontal gyrus; STG  
367 = superior temporal gyrus; CB = cerebellum; MFG = middle frontal gyrus; MTG = middle  
368 temporal gyrus.

369

370



Functional FAS in Conversion Disorder

Cluster	Peak	MNI coordinates {mm}			Area		
p (FWE-corr)	#Voxels	p (FWE-corr)	Z	x	y	z	
0.000	312	0.000	Inf	<b>-42</b>	<b>-18</b>	<b>39</b>	<b>L postcentral</b>
		0.000	7.50	<b>-60</b>	<b>-6</b>	<b>18</b>	
		0.011	5.11	<b>-48</b>	<b>-9</b>	<b>30</b>	
0.000	273	0.000	7.09	<b>45</b>	<b>-9</b>	<b>36</b>	<b>R BA6</b>
		0.000	6.93	<b>42</b>	<b>-12</b>	<b>39</b>	
		0.000	6.53	<b>60</b>	<b>3</b>	<b>21</b>	<b>R BA44</b>
		0.001	5.51	<b>51</b>	<b>-3</b>	<b>42</b>	<b>R BA6</b>
		0.003	5.35	<b>54</b>	<b>-6</b>	<b>30</b>	
		0.014	5.06	<b>63</b>	<b>0</b>	<b>9</b>	<b>R rolandic operculum</b>
0.000	318	0.000	6.51	<b>-39</b>	<b>-42</b>	<b>-45</b>	<b>L CB VIII</b>
		0.000	6.16	<b>-27</b>	<b>-60</b>	<b>-24</b>	<b>L CB VI</b>
		0.001	5.58	<b>-21</b>	<b>-69</b>	<b>-24</b>	
		0.003	5.33	<b>-51</b>	<b>-57</b>	<b>-33</b>	<b>L Crus I</b>
		0.004	5.31	<b>-48</b>	<b>-66</b>	<b>-30</b>	
		0.005	5.25	<b>-30</b>	<b>-60</b>	<b>-45</b>	
		0.014	5.07	<b>-18</b>	<b>-63</b>	<b>-21</b>	
		0.015	5.05	<b>-45</b>	<b>-66</b>	<b>-42</b>	<b>L Crus II</b>
		0.019	5.00	<b>-42</b>	<b>-54</b>	<b>-30</b>	
		0.027	4.94	<b>-42</b>	<b>-66</b>	<b>-51</b>	
		0.033	4.90	<b>-42</b>	<b>-63</b>	<b>-39</b>	
		0.033	4.90	<b>-33</b>	<b>-63</b>	<b>-42</b>	
0.000	137	0.000	6.11	<b>-57</b>	<b>-42</b>	<b>21</b>	<b>L STG/IPL</b>

		Functional FAS in Conversion Disorder				
		0.000	5.71	-57	-27	<b>21 L supramarginal gyrus</b>
		0.003	5.34	-57	-45	<b>3 L MTG</b>
0.002	32	0.000	6.11	-12	27	3 Corpus callosum
0.001	36	0.001	5.56	15	42	-18 R orbital SFG
0.000	55	0.004	5.29	-18	60	-9 BA11
0.000	59	0.010	5.13	0	45	-18 L rectus
		0.037	4.88	-3	54	-24
0.008	23	0.015	5.05	-6	-3	<b>63 L SMA</b>
0.007	24	0.019	5.01	-30	27	<b>3 L insula</b>
0.016	19	0.024	4.96	-3	-39	-51 L brainstem
0.016	19	0.029	4.92	<b>9</b>	<b>9</b>	<b>48 R SMA</b>
0.007	24	0.030	4.92	-42	-33	-18 L ITG

371

372 Legend: <sup>1</sup>LCT corresponds in this case to the repetition of “pa-ta-ka” or “ta-ta-ta”  
373 synchronized with a visual stimulus; LCT = long click train, three times “pa-ta-ka” or  
374 “ta-ta-ta”; NCT = no click train, only the preparatory cue; SMA = supplementary motor  
375 area; R = right; L = left; SMC = sensorimotor cortex; IFG = inferior frontal gyrus; STG  
376 = superior temporal gyrus; CB = cerebellum; MFG = middle frontal gyrus; MTG = middle  
377 temporal gyrus.

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382 **3. Discussion**

383

## Functional FAS in Conversion Disorder

384 We described a patient who experienced transient psychotic symptoms after a fall  
385 down the staircase and developed a FAS. Repeated neurological and neuroradiological  
386 investigations formally ruled out damage to the central nervous system (CNS). Moreover,  
387 an fMRI experiment demonstrated normal activations of the speech network during a  
388 syllable repetition task. Stress could rapidly trigger an accent change, language mixing  
389 and switching, and speech could turn into an incomprehensible phonological jargon.  
390 Neurocognitive deficits were attested and remained quite stable over the period of 1,5  
391 years. However, remarkably, performance on the naming task (BNT) regressed quite  
392 spectacularly, and inexplicably, during this time.

393

394 The onset of FAS after a traumatic experience has been previously reported in  
395 Verhoeven et al. (13), where the patient was nearly involved in a car accident as a cyclist  
396 and developed FAS after this experience. In Keulen et al. (45), the patient was hit by a  
397 car as a pedestrian, but did not suffer any structural CNS damage. A few months later, an  
398 abrupt change of personality is described. This led to the patient being fired from her job,  
399 after which a foreign accent suddenly arose. In Reddy et al. (46), the patient suddenly  
400 developed FAS after suffering from a (second) divorce. Antecedents of depression and  
401 suicidal ideation were present.

402

### 403 *Language mixing and switching*

404

405 Although a few FAS patients have been reported with pathological language  
406 mixing and/or switching, jargon seems to be a relatively new element in the clinical  
407 presentation of FAS. In 2001, Reeves and Norton (14, see also Reeves et al. (15)) reported  
408 a 65-year-old schizophrenic patient who presented with FAS concomitant with psychotic  
409 exacerbations after withdrawal from medication. The disordered speech in schizophrenia

410 was accounted for as a disturbance of inhibition mechanisms in relation to defective  
411 prefrontal neurotransmitter circuitry with associated executive dysfunctions (47). This  
412 balance was restored by administering dopaminergic drugs. The clearest example,  
413 however, of language mixing and switching in the FAS literature is the 51-year-old  
414 female Dutch patient described by Verhoeven et al. (13), also mentioned above. Her  
415 Dutch pronunciation was characterized by a French accent and her language showed  
416 syntactic and morphological features of French. In addition, she occasionally used French  
417 words or expressions. Importantly, the patient was a teacher of French as a second  
418 language. This familiarity with the accent and the lexical and grammatical characteristics  
419 of second language learners clearly distinguishes their patient from the current one.

420

421 The (phonological) jargon demonstrated by current patient has previously been  
422 associated with psychosis and psychopathy (48, 49). Most interestingly, FAS here could  
423 be triggered by conscious manipulation of the subject of conversation. It is not the first  
424 time that a link between conversationally related stressors and FAS has been attested. In  
425 Keulen et al. (50) the foreign-sounding accent of their native French-speaking patient  
426 disappeared when an emotional subject was introduced. Sudden resolution related to  
427 emotionally laden content has previously been argued to indicate psychogenic etiology  
428 (51).

429

430 *Stress, dopamine, FAS and jargon...*

431

432 In order to explain the fluctuations in the accent and the evolution towards jargon  
433 speech, it seems essential to evaluate what the effects of (emotional) stress on speech can  
434 be. Hansen and Patil argue that “[...] stress is a psychological state that is a response to a  
435 perceived threat or task demand and is normally accompanied by specific emotions (e.g.,

### Functional FAS in Conversion Disorder

436 fear, anger, anxiety, etc). These changes can affect speech behavior, even against an  
437 individual's will." (52, p. 109). How and whether stress causes (significant) impact on  
438 physical and/or psychological health depends a lot on an individual's resilience (53).  
439 Current patient repeatedly admitted her accent changed as a result of the presence of an  
440 increased number of stimuli, which she could not handle.

441

442         Interestingly, stress interferes with dopamine regulation: increased stress raises  
443 dopamine levels in the brain. Dopamine modulates prefrontal cortex inputs to the  
444 amygdala, and via the amygdala, to the hippocampus. This is one reason why elevated  
445 stress affects (esp. non-emotional) memory functioning: it has been shown in animals that  
446 stress causes structural changes in the hippocampus, e.g. via glutamate blockage (54)  
447 which causes long-term depression of synapses situated in this region, as well as  
448 impairing their long-term potentiation (55). In this respect it is interesting to note that  
449 patient demonstrated deficits with respect to memory and executive functioning.

450

451         The hypothesis of a dopamine dysregulation in FAS patients has also been raised  
452 in the context of hallucinations in schizophrenic and bipolar patients (15, 56). The case  
453 described by Reeves et al. (14, see also Reeves et al. (15)) switched language register and  
454 underwent an accent change in association with exacerbations, which were controlled  
455 after a pharmacological treatment including the dopamine antagonist olanzapine (20 mg).

456         Current patient did not suffer schizophrenia, and psychotic symptoms were only  
457 mentioned as fleeting symptoms in the history; but could no longer be corroborated in  
458 psychodiagnostic testing. The (fluctuating) accent change in relation to a self-perceived  
459 increase of stress(ors) in this patient may have caused subtle changes in neurotransmitter  
460 regulation that led to symptoms that were reminiscent of these functional FAS cases.

461

### Functional FAS in Conversion Disorder

462           Still, it seems clinical experience, combined with thorough analysis of a patient's  
463 antecedents, cognitive profile, psychiatric characteristics and behavioral abnormalities is  
464 still often judged insufficient to support the hypothesis of a psychogenic or functional  
465 origin. Nevertheless, the available psychodiagnostic tests often lack the power to allow  
466 for an unequivocal diagnosis. For many FAS cases a formal diagnosis as “functional” has  
467 proven problematic. Lee et al. identified several characteristics that help to distinguish  
468 functional and structural FAS cases (57). These include previous motor disturbances  
469 causing the maladaptive speech response, inconsistencies in accent production, the  
470 adoption of unusual mannerisms in speech, and the speech disturbances being transient  
471 and reversible. In this case, one can wonder whether motor disturbance led to the fall  
472 from the stairs after which the foreign accent syndrome developed. All the other  
473 characteristics were present in this case. In the literature, foreign accent syndrome has  
474 already been classified as, or in conjunction with conversion disorder, on several  
475 occasions (13, 58, 59).

476

477           The relation between conversion disorder and functional foreign accent syndrome  
478 definitely warrants further exploration in research. The effects and possibilities for  
479 treatment also necessitate more in-depth studies. In this respect, for current patient, it is  
480 especially noteworthy that a large-scale epidemiological study has shown that childhood  
481 trauma does have a negative impact on conversion disorder therapy outcome (60) and it  
482 was mentioned earlier that patient claimed to not have benefited significantly from  
483 previous therapy. In a very recent paper by Birdsey and Millar (61), CBT was also  
484 proposed to a woman suffering FAS on a psychogenic basis. Although CBT in that case  
485 study helped to reduce distress caused by the disorder as it did for this patient, it was not  
486 reported CBT helped to diminish the FAS and associated accent change itself.

487

**4. Conclusion**

489

490 A case of FAS is described in which jargon speech was observed. Although an  
491 underlying mental disorder, such as schizophrenia, was not formally objectified by  
492 psychodiagnostic tests, the atypical dissociations within the neurolinguistic  
493 manifestations strongly indicated a functional etiology, such as conversion disorder.  
494 Fluctuations of the speech symptoms can be induced by manipulating stressors. It is  
495 concluded that the neurolinguistic profile, symptom evolution and fMRI results are not  
496 suggestive of a neurological disorder, but rather with a functional FAS.

497

**Author contributions**

499 Manuscript drafting: SK, PM, JV, CVDF-C, LDV, LDP, TD; Neuropsychological and  
500 neurolinguistics exams: SK, PM, LDV, LDP, TD; fMRI study: SK, PM, RB, KVD, PVS,  
501 HR, JDM, FVO; Manuscript editing: all authors. Manuscript revision: SK, LDV, CVD-  
502 C.

503

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505

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508

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510 (FWO)

511

**Ethical considerations**

513

514 Ethical approval was obtained from the Ethics Committee for the Social Sciences and  
515 Humanities of Antwerp University, Belgium, SHW\_18\_26, CME Vrije Universiteit  
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517

518 **Conflict of interest**

519 The authors have no conflict of interest to declare.

520

521 **References**

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688 **Annex I: acquisition and analysis specifications for fMRI experiment (section 2.5)**

689

690 *A) Acquisition*

## Functional FAS in Conversion Disorder

691 Functional and structural MRI were conducted on a 3T GE scanner (Discovery  
692 MR750w) equipped with a standard 24-channel head coil. A BOLD sensitive T2\*-  
693 weighted single shot spin echo (SE) planar imaging (EPI) sequence (TE/TR: 70/3000ms;  
694 FA: 90deg) was used resulting in voxel dimensions of 1.875 x 1.875 x 4 mm<sup>3</sup>  
695 (interleaved) with 1mm gap, matrix = 128 x 128. Twenty-seven axial slices were acquired  
696 per volume, 200 volumes and 2 dummy scans per run. An additional SE-EPI sequence  
697 was obtained in the opposite left-right phase direction to be used as a fieldmap to correct  
698 the EPI-distortions. Three runs of 10min were used. In addition, an axial T1-weighted  
699 FSPGR BRAVO (176 slices) was taken to use for co-registration and normalization.

700

### 701 *B) Analysis*

702

703 fMRI data were unwarped using the top-up tool of fsl<sup>1</sup> and further analyzed using  
704 SPM12 software ([www.fil.ion.ucl.ac.uk/spm](http://www.fil.ion.ucl.ac.uk/spm)). After slice timing and motion correction,  
705 the unwarped functional images were registered to the T1 weighted anatomical dataset.  
706 The anatomical image was then segmented and the forward deformation field was used  
707 to normalize the functional images to MNI (Montreal Neurological Institute) space. The  
708 registered functional data were smoothed spatially with a Gaussian kernel with a full  
709 width at half maximum (FWHM) of 6 x 6 x 6 mm<sup>3</sup>. Eight conditions (“pa-ta-ka” LC; “ta-  
710 ta-ta” LC; “pa-ta-ka” SC; “ta-ta-ta” SC; visual control LC+SC; “pa-ta-ka” NC; “ta-ta-ta”  
711 NC; instructions) were modeled as separate regressors and combined with the 6  
712 movement regressors in the general linear model (GLM).

713 Similar to Brendel et al. (44), the general speech network was assessed by  
714 contrasting both LCs with the visual baseline condition. An initial uncorrected threshold

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<sup>1</sup> Andersson JL, Skare S, Ashburner J. How to correct susceptibility distortions in spin-echo echo-planar images: application to diffusion tensor imaging. *Neuroimage*. 2003;20(2):870-888.

### Functional FAS in Conversion Disorder

715 of  $p < 0.001$  at the voxel level was used to detect activations. Only clusters with more  
716 than 10 voxels were included in the table (see Table 2.2.). Activations were considered  
717 significant if the family-wise error (FWE) corrected p-value was smaller than 0.001 at  
718 the cluster level and smaller than 0.05 at the voxel level (see figure 1).

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