Neurological Aspects of Foreign Accent Syndrome in Stroke Patients

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1. Introduction

Foreign Accent Syndrome (FAS) is a relatively rare motor speech disorder characterized by speech errors which are perceived as a foreign accent by members of the same language community as the patient. The first case of FAS has traditionally been attributed to the French neurologist Pierre Marie (1907), who described a patient whose original Parisian French accent had changed to what was perceived as Alsatian French after recovery from anarthria. Since Marie's anecdotal description of FAS, the condition has been documented in at least 172 case reports.

Although the syndrome has attracted significant attention of the scientific community, little progress has been made towards a fundamental understanding of this disorder. Multidisciplinary research has neither been able to identify the pathophysiological substrate of this syndrome nor to identify a coherent system in the speech errors that may separate FAS unambiguously from other speech and language disorders such as apraxia of speech (AoS), ataxic dysarthria or even aphasia. The purpose of this review article is to present a synthesis of the available FAS data and to critically review the disorder on the basis of an analysis of cases reported since 1907. The focus will be on organic-neurogenic FAS.

1.1. Genesis of the FAS concept

On July 20, 1907 the French neurologist Pierre Marie presented four patients who had recovered from anarthria after a left hemisphere stroke involving the lenticular region (Marie, 1907). One of these patients was a right hemiplegic Parisian

man who had not been able to speak for nine years after suffering from a subcortical left hemisphere stroke. When the anarthria receded, he was not aphasic and was able to -as Marie puts it- express his thoughts in sufficient detail. He was also able to read and write (with his left hand). However, the patient's speech had changed into what was perceived as an Alsatian accent which he did not present before the insult:

> *'Next to this typical anarthric patient, you will see another one who, suffering from a right hemiplegia as well, remained without speaking for nine years. Now, he has recovered his language and can sufficiently express his thoughts; he writes quite well (with his left hand), and his inner does not seem to be affected. However, his anterior anarthria has some very clear repercussions, consisting especially of an outspoken Alsatian accent which he did not have before, because he is Parisian, and that only developed after he began speaking again' (p. 109, our translation)*

The regional accent change during recovery from subcortical left hemisphere stroke was not discussed and Marie did not identify any of the speech characteristics associated with the accent change.

The second landmark case was a patient of the Czech neurologist Arnold Pick (Pick, 1919) who described a 26-year-old Czech butcher who had developed a Polish accent after a left-hemisphere stroke. Pick explicitly addresses the determining speech characteristics to be both segmental (the softer articulation of the Czech fricatives) and suprasegmental (wrong placement of word stress). However, in contrast to Marie's (1907) report mentioning 'a kind of' regional accent, Pick is very clear about the foreign accentedness of his patient. Pick's (1919) description may be considered a less pure example of FAS after stroke. Indeed, the patient had been stationed for several months in Poland as a soldier during the First World War and also presented aphasic symptoms such as agrammatism, paraphasias, perseverations, alexia and agraphia. As such, the change to a Polish accent might also be regarded as a neurologically induced shift to a previously learned foreign accent (Polish) as part of bilingual or polyglot aphasia reflecting a selective disruption (at the phonetic level) of the cognitive-linguistic system to control the correct set of suprasegmental speech features of a particular language (accent).

The third landmark case was that of the Norwegian neurologist Monrad-Krohn (1947) who reported on Astrid L., a 30-year-old female native speaker of Norwegian. She developed a German accent after having sustained traumatic head injury to the left fronto-temporo-parietal region after having been hit by the fragment of a bombshell and falling down 'a steep incline of about 8 metres height' (p. 409). Reportedly, many Norwegians identified her accent as German and in post-war

Norway this almost painfully exposed the impact of such an accent change on the patient's identity in society. Due to the German occupation of Norway during the war, the patient "complained bitterly of constantly being taken for a German in the shops, where consequently the assistants would sell her nothing" (p. 410). According to Monrad-Krohn, this accent change was mainly caused by suprasegmental problems relating to the disappearance of the Norwegian tonal distinction. This led to the longstanding view that FAS is essentially an 'ataxia of the prosodic faculty'.

Contrary to Monrad-Krohn's (1947) view of FAS as a prosodic disorder, Nielsen & McKeown (1961) regarded the condition as a specific form of dysarthria characterized by a disturbance of the rhythm of speech. They explicitly drew attention to the highly subjective interpretation of the 'dysprosodic qualities' of the syndrome:

> *the dysprosody is simply interpreted as dysarthria unless it reminds the listener of some particular foreign language, and that therefore the interpretation is largely subjective. If the examiner did not have acquaintance with any language but his own, he would not see a simulation of some foreign language but only a peculiar form of dysarthria, a disturbance of rhythm (p. 159).*

Critchley (1962) for the first time distinguished two categorically different types of accents: 1) the intensification of a regional accent in aphasia due to a removal of inhibitory factors as the result of disease (see also Seliger et al., 1992) and 2) the appearance of something closely akin to a regional accent after a 'cerebro-vascular catastrophe' (post-aphasic dysprosodia). As an exception to the findings in the early literature which explicitly linked FAS to vascular or traumatic damage to the language dominant left hemisphere, Critchley (1962) reported two cases with an atypical etiology and a unique anatomoclinical profile. He described a 49-year-old woman who developed a regional accent in the context of a post-traumatic neurotic syndrome in the absence of any clinical evidence of a structural brain lesion. The patient's Welsh accent generated considerable interest in the media when she claimed damages in Court on account of 'the handicap' of a Welsh accent. Critchley's second patient was a 37-year-old woman who developed aphasic mutism followed by a regional Welsh accent. This patient is of special interest in that the accent did not result from left hemisphere damage, but from a right hemisphere stroke. Since this patient was reported to be right handed, she might be considered as a 'crossed FAS' due to reversed cerebral language dominance as reflected by the accompanying (crossed) aphasia.

Cole (1971) presented two patients in whom the causative lesion was located in the posterior fossa. Former editor-in-chief of Neurology, Robert Joynt, ironically commented on this unexpected finding, suggesting a crucial role of the cerebellum in the integration of motor speech characteristics and the pathophysiology of FAS:

> *it doesn't appear unusual that any of the brain mechanisms which are used to integrate motor acts may alter force, prosody, and rhythm of speech. It is well that Dr. Cole has pointed this out with posterior fossa lesions. However, I am enough of a romanticist to think that these subtle inflections and nuances of speech must ultimately stem from the cerebral cortex. For example, if Juliet had said in a monotone, ''Romeo, Romeo, wherefore art thou, Romeo?'' I doubt if Romeo would have done himself in for her. But then, I don't think Shakespeare wrote for the cerebellum. (p. 153).*

In 1982, Whitaker coined the term 'Foreign Accent Syndrome' to refer to the phenomenon in which a cerebral lesion induces a change of accent and for the first time defined the condition on the basis of four operational criteria: 1) the accent is considered by the patient, by acquaintances and by the investigator to sound foreign, 2) it is unlike the patient's native dialect before the cerebral insult, 3) it is clearly related to central nervous damage (as opposed to an hysteric reaction, if such exists), and 4) there is no evidence in the patient's background of being a speaker of a foreign language (i.e., this is not like cases of polyglot aphasia).

In 2010, Verhoeven & Mariën (2010a) made an explicit taxonomic distinction between three types of FAS: neurogenic, psychogenic and mixed FAS. In organic-neurogenic FAS the accent change is related to organic damage to the central nervous system (acquired, congenital or developmental) while this is not the case in psychogenic FAS: in the latter condition the accent is grounded in psychogenic issues. Mixed FAS occurs when a speaker with organic-neurogenic FAS further manipulates the accent to ensure greater accent consistency to create a more authentic foreign personality.

Over the years there has been substantial debate regarding the typology of FAS. The terms "psychogenic", "psychiatric", "psychosomatic", "functional" on the one hand, and "neurogenic" and "neurological" on the other, have all been used to denote different subtypes of FAS. This indicates that authors may be of the opinion that the different labels stand for different characterizations. Terms such as psychogenic, psychiatric and psychosomatic suggest a direct connection between FAS onset and psychiatric disorder. However, not all patients who develop FAS on non-neurological grounds have clearly attested psychiatric disorders. Also, some patients with neurological damage develop FAS as a functional disorder triggered

by a previous neurological event. Furthermore, the psychological symptoms may not always be substantial enough to lead to the diagnosis of a psychopathology after formal testing, even if the symptoms and their evolution are highly indicative of a non-neurological impairment. Classifying these cases as psychogenic may create the wrong impression that patients suffer from psychiatric disorders. We believe that much of the terminological debate can be solved by using the term functional to refer to (i) psychiatric cases, (ii) cases in which FAS is the result of a functional decompensation induced by a neurological disorder and (iii) cases where a psychological origin is agreed upon based on the presence of (formally attested) psychiatric symptoms, although these are not sufficient for the diagnosis of a well-defined psychiatric impairment.

Organic-neurogenic FAS patients on the other hand have suffered a neurological impairment, and the accompanying speech, language and/or cognitive deficits can be directly related to a vascular lesion (e.g. stroke), brain trauma, infectious disease (e.g. encephalitis), neuroinflammation (e.g. multiple sclerosis). Nevertheless, FAS has equally been reported in association with organic impairments that did not involve the CNS (TMJ surgery). Although it is not always straightforward to clearly distinguish between organic and functional cases – e.g. in cases of functional decompensation after neurological disorder – there are some elements that could be considered when interpreting individual cases such as onset latency, symptoms, symptom evolution and remission of symptoms (see also Keulen et al., 2016d, see also Baumgartner and Duffy, 1997).

Objectives

This article aims to review foreign accent syndrome (FAS) after neurological damage, with a specific focus on FAS occurring after stroke. To date, many case studies have been published, but a comprehensive literature review encompassing all the clinical aspects described over the past decades has been missing. This review focuses on the clinical presentation(s) of stroke-based FAS. This will be to the benefit of clinicians and researchers in understanding this disorder. Special attention is given to the demographic characteristics of the patients, the lesion locations, comorbid speech/language disorders, co-occurring cognitive impairments and remission. Where of interest, a comparison is made with functional FAS cases. In order to summarize the findings, a description of the "typical" organic-neurogenic FAS patient is given.

2. Methods

Publications on FAS were identified by means of searches in electronic databases (Medline, PsycINFO, Current Contents, Web of Science). In addition, the bibliographies of all obtained articles were scrutinized to identify additional references. Only first source information was analyzed; second- or third-line references to original contributions were not considered. This resulted in a survey of 172 FAS cases in total.

The quality of the collected data was variable as to the degree of reported details. Inclusion and exclusion criteria were applied to narrow down the corpus for further analysis. Inclusion criteria were defined as (i) the description of a patient in whom a change of accent was observed, and (ii) for whom there was a clear description of the associated etiology in order to avoid misinterpretation of the data. In order to improve the readability of this paper, we have avoided listing cases in the text to which particular statements apply. Rather, we have used single numbers between square brackets to represent series of FAS cases: the association of these numbers and the FAS cases they refer to can be found in table 8.1 in section 8 of this article (list A). The alphabetically ordered list B of table 8.1 enables the reader to identify the paper in which each FAS case is described. A full summary description of individual FAS patients can be consulted in the supplementary materials.

Regarding the exclusion criteria, it was decided to exclude cases reported in poster or oral presentations, conference proceedings [1][1] and unpublished theses [2][2]. Furthermore, cases were excluded if (i) there was an explicit statement that the etiology was unclear or could not unambiguously be identified as organic or as functional in the above-mentioned meaning by the authors [3], (ii) the etiology was not mentioned [4], (iii) there was doubt about the origin (‘suggestive’) [5]. Due to linguistic limitations, only articles published in English, Spanish, Norwegian, German or Portuguese were included. One case had to be excluded because a translation could not be obtained, i.e. Tokudo et al. (2015).

It should also be mentioned that some of the cases have been reported more than once [6]. When a case occurs in several publications, this is indicated in the the supplementary materials. When considering only ‘authentic’ cases fulfilling the inclusion criteria, the number of cases amounted to 112 [7]: these were published between 1907 and October 2016.

[1] Codes between square brackets correspond to a series of case studies that can be consulted in List A of table 8.1.
[2] It is important to note that some of the cases that were excluded based on this exclusion criterion, were later described in more detail in full-length articles in peer-reviewed journals. When a case occurs in several publications, this is indicated in the case list as well as in the supplementary materials.

3. Results

3.1. Demographic characteristics

The demographic, etiological, neuroradiological, speech/language and neuropsychological characteristics for all the cases were surveyed in detail. This survey can be consulted in supplementary materials. Cases marked with an asterisk in the supplementary materials are the ones that survived the corpus inclusion and exclusion criteria mentioned in section 2 (Methods) above.

Most patients with FAS are adults (97.32 %; 109/112): only three patients are children (<18 years) [8]. Seventy-six out of 112 patients are women (67.86%; 76/112), thirty-six are men (32.14%; 36/112). A binomial test indicates that the proportion of female FAS patients is significantly higher than the proportion of male patients ($p<0.0001$ - binomial test). The mean age of the FAS patients is based on calculations for 111 patients, as age was not mentioned for the patient described by Marie (1907). The mean age of the FAS patients is 48 years (range 7 to 88 years; SD: 14.59). The mean age of the female patient group (76/112) is 49 (range 24 to 88 years, M=49; SD=12.91), while the mean age of the male patient group (35/112) is 45 (range 7 to 76 years, M=46; SD=17.48). The age difference between men and women is not significant (Mann-Whitney *U* test, $U=1164$, $p=0.292$).

Hand preference is mentioned in 62 cases (55.36%; 62/112). Fifty-nine patients are right-handed (95.16%; 59/62) [9]. Only three patients are left-handers [10]. For the remaining 50 patients, handedness was not mentioned. Right/left hand dominance ratio is approximately 21:1. Handedness was formally assessed by means of a questionnaire or a handedness test in twelve of the right-handed cases [11] (19.35%; 12/62) and in one of the left-handed patients [12].

Twenty-six out of the 112 cases (23.21%; 26/112) are described as polyglots[3] at the time of FAS onset [13]. Most of these patients, however, learned their second or third language in school [14]. The patient described by Pick (1919) learned Polish while serving as a soldier. The case described by Seliger et al. (1992) retained some notions of the Irish brogue and its associated accent, because the patient had been exposed to this accent as a child through contact with relatives. The patients described by Roth et al. (1992) and Seliger et al. (1992) were described as instances of a “resurgence of accent” after long neural suppression due to a vascular incident and accompanying neural plasticity effects. The patient reported by Roth et al. (1992) was born in the Netherlands, but moved to the U.S.A. at a very young age. He

[3] The authors are aware that concepts such as ‘bilingualism’ and ‘polyglot’ can be defined in different ways. For the purpose of establishing this corpus all types (compact, coordinated, subordinated, balanced, dominant - Fabbro, 2003) were included, without making distinctions. We followed the authors' descriptions in detail.

adopted a Dutch accent after having suffered a stroke in the parietal area of the left hemisphere. The case described by Paquier and Assal (2007) was stated to be a 'polyglot', without any further specifications. The patient in Haley et al. (2010) was English-speaking but learned Spanish at an early age through contacts with Spanish-speaking friends. The remaining patients were raised bilingually [15]. Thirty patients were mentioned to be monolinguals [16] (26.79%; 30/112). For 109 patients (97.32%; 109/112;) the native language was explicitly documented in the case report, while this was not the case for three patients [17]. Nevertheless, inference from the information provided in the article demonstrates that the two cases described by Liu et al. (2015) spoke a local Chinese dialect which evolved to Mandarin (standard) Chinese after incurring a stroke.

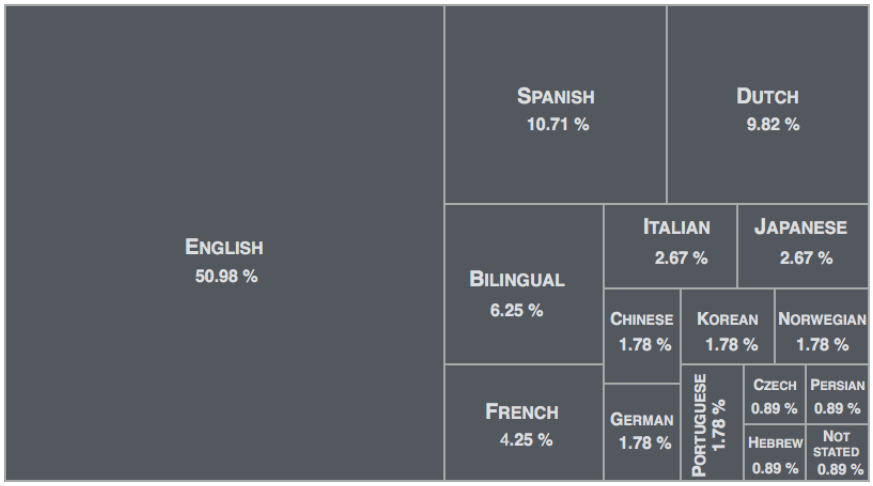

Figure 3.1. ***Plot of the different mother tongues in the FAS patients (n=112).*** No further specification of the regional variant is given as this was not always reported. Over 50% of the FAS patients included in the review had English as their mother tongue.

The majority of patients were either monolingual or (early) bilingual native speakers of English (54.46%; 61/112) [18] (fig. 3.1.).

In some FAS patients (12.5%; 14/112) there was evidence of previous contact with the accent that became the 'FAS-accent' [19], but this does not mean that these patients were fluent in the associated language. For instance, some patients had had previous contact with the accent due to language contact with relatives [20] or had received formal language instruction at school at a later age [21]. For example, the patient described by Ardila et al. (1988) had learned some elementary

English at high school, though he claimed never to have achieved fluency in the language. He mentioned that he could conduct a simple conversation in English. However, after incurring a stroke in Broca's area, he started speaking Spanish with what was perceived as an English accent. In the latter cases, and in the polyglot cases, the patients can be considered to have reverted to a previously learned "accent".

3.2. Etiology

77.68% of the FAS patients are instances of organic-neurogenic FAS, i.e. organic damage to the central nervous system (77.68%; 87/112) [22]. In 5 patients (4.46%; 5/112) FAS was acquired as a developmental speech impairment, implying that the patients had been speaking with a foreign accent before language development was completed [23]. Eighteen patients (16.07%; 18/112) developed FAS in the context of a psychological or psychiatric disorder [24], and can be considered as functional cases. Two patients described by Dilollo et al. (2014) developed FAS on an organic basis, without CNS damage, after temporo-mandibular-jaw (TMJ) surgery. Interestingly however, case 40 experienced psychological and psychosocial changes after the onset of her FAS, and travelled to England where she felt she would be more comfortable with the newly adopted accent. This case may fit the description of "mixed FAS": the accent was associated with TMJ surgery by the authors, but clearly reinforced due to subsequent psychological changes induced by a sense of change in the personal identity, albeit not after CNS damage.

In sixty patients (53.57%; 60/112) FAS was associated with stroke [25] (Table 3.1). Twenty-two of these are men (36.67%) and 38 are women (63.33%): women are affected by vascular FAS more often than men, though statistical comparison did not survive a binomial test (p=0.052). The mean age of the vascular group is 51 years (M=53, SD= 12.8 years, range= 24-76 years), but this excludes the case of Pierre Marie (1907) who was not specified for age. Men seem slightly younger (mean age=50 years, M=53 years, SD=14.40) than women (mean age=52 years, M=53 years, SD=12 years) but this difference is statistically not significant (t(59)=-0,447; p=0.656).

Table 3.1.

Summary of the frequencies of the different etiologies in the FAS patients included in the study corpus (n=112). Etiologies are grouped per FAS subtype. For functional FAS cases the terminology was adapted to DSM-V terminology. Most of these cases were subjected to a more in-depth discussion in Keulen et al. (2016d). No specification of type (bipolar I or II disorder) according to the DSM-V standards was provided (cases published before the 2013 revision of DSM).

FAS TYPE	ETIOLOGY	% (x/112)
ORGANIC - NEUROGENIC	Stroke	53.7 (60)
	Trauma	13.39 (15)
	Tumor	2.68 (3)
	Multiple sclerosis	2.68 (3)
	Primary Progressive Aphasia	1.79 (2)
	Paediatric Autoimmune Neuropsychiatric Disorder Associated with Streptococcus.	0.89 (1)
	Vaculitis	0.89 (1)
	Garcin's syndrome	0.89 (1)
	Vascular dementia	0.89 (1)
ORGANIC WITHOUT CNS DAMAGE – MIXED (?)	Temporo-mandibular jaw surgery	1.79 (2)
DEVELOPMENTAL	Developmental disorder	4.46 (5)
FUNCTIONAL	Conversion disorder (or functional neurological symptom disorder)	5.36 (6)
	Schizophrenia	2.68 (3)
	Obsessive compulsive disorder	1.79 (2)
	Bipolar disorder	0.89 (1)
	Post-traumatic stress disorder	1.79 (2)
	Borderline personality Disorder	0.89 (1)
	Manic episode (not reconcilable with any identifiable pathological condition)	0.89 (1)
	Suspected conversion disorder	0.89 (1)
	Depressive disorder (and suicidal ideation)	0.89 (1)

In 15 patients (13.39%; 15/112) FAS resulted from a cranio-cerebral trauma [26]. Together with stroke-based FAS, these cases make up the bulk of the etiologies. Four patients (3.57%; 4/112) presented with FAS in the context of an inflammatory disease such as multiple sclerosis (MS) [27] and vasculitis [28]. In three patients (2.68%; 3/112) FAS was identified during the course of a primary

neurodegenerative disorder [29]. In three other cases (2.68%; 3/112) FAS was associated with a brain tumor [30].

In one patient [31] (0.89%; 1/112) the infection causing FAS was identified as Paediatric Autoimmune Neuropsychiatric disease Associated with Streptococcal infection (PANDAS). The disease is known to mimic symptoms of (neuro/pedo-)psychiatric disorders such as Obsessive-Compulsive Disorder (OCD), Attention Deficit Hyperactivity Disorder (ADHD) and tics. In one patient (0.89%; 1/112) a multifactorial cause (trauma, viral infection: Garcin's syndrome) was suspected [32].

3.3. Lesion location in FAS

The lesion locations in the FAS patients are summarized in table 3.2 below. Table 3.2 shows that the majority of the patients with neurological damage have a lesion in the supratentorial left hemisphere (68.97%; 60/87) [33].

Table 3.2.

Classification of FAS speakers in terms of the location of structural brain damage. The asterisk indicates cases with vascular lesions. Numbers in the table refer to the case numbers in the supplementary materials.

LEFT HEMISPHERE			
Supratentorial	Infratentorial	Infra- and supratentorial	No further information
1, 3*, 6, 7*, 9*, 10, 12, 15*, 18, 19, 20*, 22*, 26*, 28*, 42, 43*, 44*, 46, 48*, 49*, 50*, 56, 65*, 71*, 75*, 84*, 87*, 89*, 91, 92, 94, 95*, 96, 97*, 103, 107*, 111, 113*, 114*, 115*, 116*, 117, 118, 123*, 127*, 137*, 139*, 141*, 142*, 143*, 146*, 147*, 150*, 153, 156*, 158*, 168*, 171*, 172*	35*, 154*	45, 72*, 73*	36*
RIGHT HEMISPHERE			
Supratentorial	Infratentorial	Infra- and supratentorial	No further information
8, 36*, 105*	101*	/	29*
BILATERAL			
Supratentorial	Infratentorial	Infra- and supratentorial	No further information
5, 16*, 119, 121, 11*, 93*	169	/	.

HEMISPHERE NOT INDICATED			
Supratentorial	Infratentorial	Infra- and supratentorial	No further information
53*	23*, 24*	57*	59*, 61, 62*, 63, 85*

Seventy percent of the patients with lesions in the supratentorial left hemisphere are stroke patients (70%; 42/60) [34] and 59.52% (25/42) of these patients are right-handed, 4.76% (2/42) are left-handed. Handedness is not specified in 35.71% (15/42) of the cases. Right hemisphere damage is rare and only occurred in FAS cases who suffered from stroke [35] (3.57%; 4/112). Cases with damage to infratentorial regions all suffered from stroke: all these patients are right-handed, and for case 154 handedness was not specified. For cases with infra- and supratentorial damage, only case 169 had not suffered a stroke. The patient's T2-Magnetic Resonance Image (MRI) showed hyperintensities at the infra- and supratentorial level and she was diagnosed with MS.

3.4. Lesion location in FAS associated with vascular damage

A number of studies have shown that the most appropriate etiology for establishing anatomoclinical correlations are circumscribed cerebrovascular lesions (including infarctions, hemorrhages and ruptured aneurysms with intraparenchymal damage) in the intermediate period ranging from about three weeks to about three months post-onset (Alexander, 1989; Mazzochi & Vignolo, 1979). The morphology of the lesion remaining the same, neurobehavioral symptoms in the earlier period (the acute phase; until approximately 3 weeks post-stroke) are often more severe than the lesion would suggest (due to the additional effect of diaschisis affecting ipsi- or contralateral brain areas, mass effect and perilesional damage (penumbra)). The effects of damage also depend on the affected brain area (Witte et al., 2000) and they may become less severe in the later period (the chronic phase; after approximately 3 months post-stroke) due to the opposite effect of functional compensation resulting from spontaneous recovery or therapy. Neurocognitive and neurolinguistic disorders presumably mirror the effect of the lesion most faithfully in an intermediate period – called the lesion phase of the stroke by Alexander (1989) – when the lesion is stabilized, associated neurobiological phenomena have disappeared and compensation is minimal

(between approximately 3 weeks and 3 months post-stroke) (Mazzochi & Vignolo, 1979).

To establish lesion behavior correlations for FAS in the present review, care has been taken to only single out the stroke cases (53.57%; 60/112). In vascular FAS patients, lesions were often situated in and around BA 4 and 6 (SMA, premotor area (PM) and motor strip) [36], the basal ganglia [37] and the left parietal lobe [38]. The lesion locations in Table 3.3 below are quite generic (first column). However, as the lesion descriptions were not always provided in sufficient detail in the case reports, an acceptable means of classification had to be found.

Table 3.3.

Lesion locations of vascular cases with FAS. The numbers in the second column refer to the case numbers in the supplementary materials. The asterisk (*) refers to a lesion located in the precentral gyrus, cases in bold were reported to have multiple lesion sites and occur at various places in the table; the cases in italics are the cases with pyramidal and extrapyramidal lesions.

Lesion location	Case references (see appendix corresponding cases)	% of vascular cases
Basal ganglia	***11***, ***16***, 26, ***36***, 43, 44, 48, ***49,*** 50, ***71***, 87, 95, ***97***, 115, 127, 141, 147, 151, 156	32
Frontal lobe (left)	3, 7*, 9, ***11*****, 15***, ***16***, **20***, **22**, ***49****, **89**, **96**, **139**, 142*, 143*, **146**, 150*, 172*	28
Parietal lobe (left)	**15, *16*, 22, 65, *71*, 75, 84, 89, 96, 97, 139, 139**	20
Insular region (left)	**20, 65, 96, 97, *113*, 139, 146,** 168	13
Temporal lobe (left)	**16, 65, 75, 84, 89, 97**	10
Pons	24, 35, 48, 154	7
Frontal operculum	**20, *113,*** 114	5
Temporal lobe (right)	***16, 36,* 85**	5
Cerebellum	23, 101	3
Frontal lobe (right)	***16,*** 106	3
Parietal lobe (right)	***36,* 85**	3
Corpus callosum	53	2
Insular region (right)	***11***	2

These case reports were then scrutinized in order to determine the time of FAS onset: 54 of the 60 case reports contained sufficient detail to associate FAS with either the acute, lesion or chronic phase: these were included for further analysis (90%; 54/60) [39]. Results indicated that 38 of the stroke patients (63.33%; 38/60) presented with FAS in the acute phase of the stroke [40], seven cases (11.67%; 7/60) developed FAS in the lesion phase of the stroke [41] and two cases (3.33%; 2/60) developed FAS in the late phase of the stroke [42]. For the remaining thirteen stroke patients (21.67%; 13/60) the occurrence of FAS onset could not be related to stroke onset [43]. Sixteen of the 38 patients (42.11%; 16/38) with FAS in the acute phase still had accented speech in the lesion phase of the stroke [44].

After verification of the imaging data, it was concluded that eight cases had damage largely restricted to the subcortical areas [45]. Eleven of the 16 patients were right-handed [46] (68.75%; 11/16), one patient was left-handed [47] (6.25%; 1/16) and for the remaining four patients, handedness was not specified (25%; 4/16). In fourteen patients in whom FAS persisted throughout the lesion phase, the condition resulted from a left hemisphere lesion [48] (87.5%; 14/16). The two remaining cases developed FAS after incurring an infarction in the body of the corpus callosum [49], and a hemorrhage in the right cerebellum [50].

3.5. Comorbid speech and language disorders

A general observation in the FAS literature is that the disorder seldom occurs on its own. In the stroke population [51], 41.67% (25/60) of the cases were mute in the acute phase before they developed FAS [52]. After mutism had receded, particularly aphasia, dysarthria and apraxia of speech were noted. Aphasia was noted in the acute phase in nineteen stroke cases [53] (31.67%; 19/60). Two cases developed aphasia in the lesion phase [54] (3.33%; 2/60). The case described by Pyun et al. (2013) was reported to suffer from aphasia. However, it was impossible to derive onset from the information provided in the case report: aphasia was formally confirmed one month post-stroke, but could equally well have developed in an acute stage.

Dysarthria was found in the acute phase in eleven stroke patients [55] (18.33%; 11/60), and in one patient dysarthric speech problems occurred in the lesion phase [56] (1.67%; 1/60). For the cases described by Cole (1971, case 1), Dankovičová et al. (2001) and Kurowski et al. (1996) dysarthria was also reported but a time of onset could not be accurately determined based on the case description. Apraxia of speech (AoS) was reported in the acute phase in five stroke patients (8.33%; 5/60) [57]. The cases described by Pyun et al. (2013) and Kuschmann et al. (2012, case 1= Kuschmann & Lowit 2015, case 1) were both

reported to suffer from AoS, but were only formally tested one month and twenty-six months post-stroke respectively. Hence, no exact time of onset could be determined on the basis of the information in the case report. Agrammatism occurred in 11 stroke patients [58] (18.33%; 11/60). Roth et al. (1997) [59] provide contradictory information in this respect, claiming that their patient was agrammatic (p. 551) without providing evidence for a grammatical disorder and stating that "the language of their patient was grammatically correct and lexically full" (p. 550). Due to the conflicting information this case had to be excluded. In three patients [60] agrammatism developed in the lesion phase (5%; 3/60). Two stroke patients presented with agrammatism without aphasia [61] in the acute stage (3.33%; 2/60), and one patient was described as producing “grammatical errors”, without any further information about the nature of these errors [62] (1.67%; 1/60). This patient was not aphasic and did not suffer any other comorbid speech or language disorder, except for a pre-FAS mutism. Alexia was also attested in four patients in the acute phase [63] (6.67%; 4/60). Agraphia was noted in the acute phase in five stroke patients [64] (8.33%; 5/60) and in the lesion phase in one patient [65] (1.67%; 1/60).

3.6. Cognitive impairments

In almost half of the vascular FAS cases [66] (45%; 27/60) neurocognitive findings were reported. In 33 out of the 60 vascular FAS cases (55%; 33/60) [67] cognitive functions were not investigated. Relatively few patients were identified to suffer cognitive impairments (15%; 9/60) [68].

Five out of the nine patients with identifiable cognitive impairments had some degree of acalculia [69]. For case 16 it was mentioned that the patient experienced difficulties with more complex calculations. Acalculia is typically associated with frontal or parietal lobe damage (dominant angular gyrus) (Kahn & Whitaker, 1991; Ardila & Rosselli, 2002). Case 97 had parietal damage and case 146 had damage affecting the frontal lobe, whereas for the remaining cases [70] the lesion location was not described in sufficient detail (case 72, 73): ‘left hemisphere’; case 116: ‘left middle cerebral artery territory’ (see table in appendix). Case 116 and 97 had isolated arithmetic deficits. For case 116, who also displayed agraphia, it is possible that the patient also suffered from (partial) Gerstmann syndrome (Benton, 1977, 1992).

Impaired working memory was noted in six patients [71]. In case 127, both digit and visual span were affected (tested at 1 month and 6 months post-onset): cognitive testing revealed a severe and sustained working memory deficit. For cases 72 and 73 no information was provided as to which tests had been used. For

cases 115 and 127 executive dysfunctions were mentioned, but test results were only provided for case 127. The latter case, who suffered an extensive basal ganglia hemorrhage, displayed impairments in attention (digit and visual span forward), executive functions, memory and non-verbal intelligence (digit and visual span backward; verbal learning test; Rey Complex Figure; Rey, 1941; 1964; the Trail Making Test; Reitan, 1958; the Wisconsin Card Sorting Test; Grant et al., 1993) and the Raven Coloured Progressive Matrices (Raven et al., 1956; Raven, 1976). Visuoconstructive abilities were intact (RCF copy). Cognitive defects remained present after intensive cognitive therapy (twice/week, 1 year; retested 12 months post-onset). Case 71 was diagnosed with a frontal lobe disorder following weak performance on the Luria 3-step test (Luria, 1970; 1980) (no scores available).

A general cognitive decline in the context of Schmahmann's syndrome (Manto & Mariën, 2015) was found in case 101, who developed FAS following a right cerebellar hemorrhage. The decline was reflected by pathological scores on all the cognitive tests administered one month post-stroke (Mini Mental State Examination (MMSE; Folstein et al., 1975), Wechsler Adult Intelligence Scale-III (WAIS-III; Wechsler, 1998), Repeatable Battery for Assessment of Neuropsychological Status (RBANS; Randolph et al., 1998), RCPM, WCST, TMT, the Middelheim Frontality Scale (De Deyn et al., 2005) and Frontal Assessment Battery (Dubois et al., 2000). The scores on the executive tasks had deteriorated when retested 6 months post-stroke (Stroop task; Golden et al., 1978; WCST, and TMT).

3.7. Remission of FAS

Analysis of the entire corpus reveals that the foreign accent receded in 24.17% (or 21/87) of the patients with CNS impairment [72]. For vascular FAS, remission of FAS was reported in twenty percent of the cases (20%; 12/60) [73] (range: 1 day - 3 years). In nine of these patients the lesion was situated in the frontal lobe, affecting the precentral area and frontal gyri [74]. Three patients who had basal ganglia involvement recovered from FAS [75]. For case 57 (a) pontine lesion(s) was withheld. By contrast, seven out of the 18 patients (or 38.89%) with functional FAS recovered from the condition during the follow-up period [76] (see also Keulen et al., 2016d).

Multiple chi-square tests were conducted in order to investigate: 1) whether the prognosis for patients with functional FAS is better than for those with (acquired) organic-neurogenic FAS (i.e. due to CNS damage), 2) whether there is a gender-related advantage in recovery and 3) whether there is an influence of the presence of

comorbid speech and/or language disorders on the remission of FAS symptoms. For the purpose of these analyses, only patients with organic-neurogenic FAS were compared to those with functional FAS, as too few patients suffer from the other taxonomic variants. This resulted in a total study group of 105 cases, 18 of whom suffered from functional FAS. 32 out of the 105 patients were men, 73 were women.

As far as the relationship between FAS subtype and remission is concerned, the chi-square analyses indicated that there was no significant association between the two variables (p=.243, Fisher's exact test). The same holds when the group with organic-neurogenic FAS is isolated χ^2(1, n=87) =.443, p=.506, as well as the stroke patients (p=.327, Fisher's exact test). As far as the second question is concerned, chi-square analysis revealed that there is no gender-related benefit regarding the remission of FAS: the relationship between gender and remission is not significant when collapsing all patients χ^2(1, n=105)=.494, p=.482. When looking only at patients with an organic-neurogenic FAS, the association could not be confirmed either χ^2(1, n=87)=.443, p=.506. The same goes for the stroke population (p=.712, Fisher's exact). Concerning the third question, a chi-square analysis revealed that there was no significant relationship between the presence of comorbid disorders and prognosis (remission) χ^2(1,n=105)=1.410, p=.235. The same conclusion holds when considering only patients with organic-neurogenic FAS χ^2(1, n=87)=.645, p=.422 and stroke patients (0.712, Fisher's exact). Furthermore, there was no significant relation between the occurrence of pre-FAS muteness and remission for the group of organic-neurogenic and functional patients taken together χ^{22}(1, n=105)=.001 p=.975. This was also the case when the patients with organic-neurogenic FAS X^2(1, n=87)=.021 p=.886 and stroke group χ^2(1, n=60)=1.714, p=.19 were investigated separately.

4. Discussion

4.1. Demographic findings and lesion location

FAS occurs more than twice as often in women (67.86%; 76/112) than in men (32.14%; 36/112) and when considering only the stroke population, this difference almost reaches significance. Interestingly, according to recent data by Thrift et al. (2017) world stroke incidence is consistently higher in men than women on a global scale. The age difference between women and men was not significant. Nevertheless, it is important to point out that the mean age of 51 years at FAS-onset in the stroke population (M=53, SD= 12.8 years, range= 24-76 years) is quite low compared to figures for other organic motor speech disorders. Flowers et al. (2013) investigated the mean age of first-ever acute stroke patients and found that the mean age of patients with dysarthria was 69.1 years (SD=14; n=92). For apraxia of speech, Ogar et al. (2006) obtained a mean age of 63.1 years (SD=13.3;

range: 31-79; n=18) for stroke patients in the late phase of stroke (+1 year post-stroke). Hickok et al. (2014) included 17 patients with AoS in a study on the relation between AoS and verbal short-term memory. The patients, all of whom suffered an ischemic infarction, were investigated in the acute stage and presented comorbid aphasia. Their study population had a mean age of 57.8 years (SD= 14.2).

It should be kept in mind that the low age range, which is in line with the age range in which functional speech and language disorders occur, and the fact that relatively more women than men suffer FAS - irrespective of the etiology - may well suggest that the organic-neurogenic group in this study contains at least a few functional or mixed cases (see also Baumgartner and Duffy, 1997).

The majority of FAS patients for whom handedness was reported were identified as right-handed: 95.16% (59/62). Crossed FAS in right-handed patients only occurs in four cases: three stroke cases [77] and one case in which the lesion was traumatic in nature [78]. The incidence of crossed stroke-based FAS (6.67%; 4/60) between 1907 and 2016 is slightly higher than the incidence of crossed aphasia which is estimated to be between 0.38% and 4% in the stroke population (Hécaen et al., 1971; Carr et al., 1981; Mariën et al., 2004). For the vast majority of the vascular patients FAS was associated with an isolated left hemisphere stroke (78.33%; 47/60) [79] (see also table 3.2). The four stroke patients with right hemisphere damage included two patients who suffered FAS and a comorbid (ataxic) dysarthria after damage to the poster fossa region (brainstem and pons), whereas the remaining two patients only suffered FAS.

Patients primarily had lesions affecting the SMA, premotor or motor strip [80], but also in or near Broca's area, affecting the inferior frontal gyrus (IFG) [81], the left center semi-ovale [82], the internal capsule with basal ganglia involvement [83], or restricted to the basal ganglia [84]. Others had lesions in the fronto-temporo-parietal lobe [85], the left temporal lobe [86], the left parietal lobe [87], or the fronto-parietal lobes [88].

A few vascular FAS patients had lesions largely confined to areas which at first sight only seem indirectly related to speech production disorders e.g. [89]. The patient described by Hall et al (2003), for example, suffered an ischemic infarction in the body of the corpus callosum, hence disturbing left and right hemisphere communication. FAS in this patient was mainly characterized by prosodic impairment, which the authors defined as a “linguistic aprosody” (p. 1551). Monrad-Krohn (1947) also characterized FAS in his patient as a dysprosody, but in his perspective, this seems to refer more strongly to ‘linguistic prosody’. The patient's musical prosodic abilities were unaffected and prosodic-linguistic problems were situated at the level of word accent rather than sentence intonation.

He furthermore qualified the patient's condition as "an 'ataxia' of the prosodic faculty" (p. 411). Whitaker (1982) considered the typical timing and target errors of his patient as analogous to ataxic dysarthria and speculated that the cerebellum might be functionally implicated, via anatomical connections with the inferior rolandic cortex which acts as a pivotal center in the speech production system.

4.2 FAS, AoS and ataxic dysarthria: the semiological resemblance

In over 40% of the stroke patients, FAS was preceded by a phase during which the patient was mute 52. Subsequently, FAS usually emerged in an acute phase (within 3 weeks after the vascular incident), sometimes together with an aphasia, dysarthria and/or AoS.

It is not always clear how a differential diagnosis between AoS, dysarthria and FAS was made and this may not always be self-evident: many of the clinical-perceptual characteristics of each of these disorders can occur in the others. In many cases, features typically associated with dysarthria or apraxia qualify as a FAS characteristics as well, i.e. as responsible for inducing the impression of a foreign accent: segmental changes affecting place and manner of articulation occur, which suggests an association with both AoS and dysarthria. However, it seems that the changes in organic-neurogenic FAS type are fairly consistent. Error-consistency, however, is primarily a feature of dysarthria. Consistency in FAS may be associated with the different hypotheses that have been proposed regarding the pathophysiology of FAS. The advanced tongue root hypothesis or muscular tension hypothesis by Graff-Radford et al. (1986) and Ingram et al. (1992) suggests an advancement of constriction location leading to a consistent pattern in changes resulting in, for instance, postalveolar consonants becoming alveolar(-like).

Arguably, FAS is also remarkably similar to the speech disturbances associated with lesions involving the superior paravermal region of the cerebellum, i.e. ataxic dysarthria (Kertesz, 1982; Ackermann et al., 1992; Mariën et al., 2001, Mariën et al., 2006). A slow and strikingly irregular articulation, a monotonous, staccato and scanned oro-verbal output, (mis-)articulations of vowels and consonants, and deficits affecting voice onset time (the time interval between consonant burst and vowel onset) and the production and discrimination of vowel length are commonly found in AoS, FAS and ataxic dysarthria (Mariën et al., 2001; Ackermann & Hertrich, 2000). Dysarthria occurred at approximately the same time as FAS in 13 out of the 15 vascular FAS cases: in 9 cases they both occurred in the acute phase [90], in one case they both occurred in the lesion phase

[91]. In another case, dysarthria preceded FAS [92]. Type of dysarthria was only specified for two cases [93]. However, it is very likely that the case described by Cole (1971) suffered an ataxic dysarthria, as is suggested by the remarks of Robert Joynt quoted above in the introduction (see section 1).

The most striking difference is of course the perceived foreign accent, which – to our knowledge – has not been reported in the context of (acquired) AoS or dysarthria. AoS and dysarthria were reported relatively infrequently in the corpus despite of often being associated with FAS. As the accent seems to be the single most distinctive characteristic compared to AoS and dysarthria, this is possibly one of the reasons for seeing FAS as a (mild) subtype of a planning and execution disorder in the likes of AoS and (ataxic) dysarthria.

Even from a neurobiological point of view it seems possible to associate FAS with both disorders. For instance, the (left) PM was identified as one of the most frequently impaired areas in the stroke patients with FAS in the current corpus. Findings by New et al. (2015) have demonstrated that this area is most likely a key region in the onset of AoS. They compared a group of stroke patients with AoS, without AoS (but with language disorders), and healthy controls. In their lesion overlap map, they found the highest overlap in the left hemisphere caudate (head), insula and premotor regions (BA6) for the patients with AoS. Within their selected regions of interest (comprising of the IFG, anterior insula and PM) especially the left PM was significantly more affected in the patients with AoS. In their resting state functional connectivity study, they found altered (in this case: reduced) connectivity between the left and right premotor areas in the group of AoS patients, which was negatively correlated to severity of AoS: the more negative the connectivity, the higher apraxia severity.

They evaluate their findings in the context of the DIVA model, which has been found particularly suited to explain the pathophysiology of AoS (see also Ballard et al., 2014). The DIVA model is a computational model consisting of a feedforward and a feedback loop which mutually inform each other in order to achieve repair in the case of articulatory breakdown (Tourville and Guenther, 2011). Via the feedforward control, one is aware of the phonological results of motor programs (predictive component) which are transferred to the articulator position and velocity maps. When the initiation maps in the (bilateral) supplementary motor area are activated, they release commands. These commands are to be interpreted as schemes containing plans for the position of the different articulators during speech, which are translated into codes for movements. The speech sound maps (situated in the premotor and inferior frontal cortex) contain

the speech motor programs and project to auditory and somatosensory cortices to inform about the articulatory results. When speech is articulated, the somatosensory and auditory-perceptual awareness activates the feedback loop. The latter updates the feedforward loop for correct target attainment. The right hemisphere premotor areas are an essential part of the feedback control map, from where control schemes can be sent to the cerebellum, before being translated into (corrected) position maps. According to New et al. (2015) the reduced bilateral PM connectivity could have led to deficient (pre)programming, or may have interfered with adequate compensation in their AoS patients, because (effective) reliance on the contralateral right hemisphere PM was reduced due to negative connectivity.

Moreno-Torres et al. (2013) emphasized the importance of a stable interplay between planning, executive and control networks in their study of a 44-year-old bilingual Catalan and Spanish female patient, who primarily displayed distortions of an executive nature, not planning. MRI showed lesions affecting the bilateral deep frontal opercular and insular regions. According to the authors the lesions were too discrete to cause an apraxia of speech. They hypothesized that the perceptual errors in their patient were the result of the distance effects the lesions (situated in the planning network) exerted on the executive network. In this respect it is also interesting to note that the patient had a non-native accent in both languages, which is expected if the errors are induced by neuromuscular articulation deficits. Transposing this information to the DIVA model, the increased metabolic activity in the PET scan, which was situated in the left superior temporal gyrus, the right middle IFG and left cingulate gyrus, could have been the functional correlate of the (flawed) error restoration via increased somatosensory feedback to the bilateral premotor cortices, motor cortices, inferior frontal gyri, possibly leading to FAS as an outcome.

A few years earlier, Scott et al. (2006) had argued that FAS may be the result of “a disconnection of the planning of articulation from motor control” (p. 370). Their patient was a 36-year-old right-handed Scottish female who suffered a stroke in the white matter near the precentral sulcus and anterior insula of the left hemisphere and developed a foreign accent described as German, Polish, or South African. Scott et al. (2006) stated that the role of the insula encompasses the integration of phonological features with suprasegmental features. The disruption of this process would entail FAS-like speech and can be seen as the consequence of a disruption between the motor strip and anterior insula, which means that they were possibly considering FAS as a disorder of both speech planning (such as AoS) and execution (such as dysarthria). In keeping with New et al.’s (2015)

findings, it doesn't seem straightforward to designate a single area as the substrate of a disorder. The connectivity between several regions within the speech network needs to be preserved and it seems that location of the altered connectivity, induced by a lesion, is decisive to symptom outcome.

Adopting a more holistic, connectivity-informed view in relation to (functional) neuroimaging studies has for instance confirmed that the cerebellum seems to be crucially involved in motor speech planning disorders (Mariën et al., 2006; Mariën & Verhoeven, 2007; Mariën et al., 2013; Keulen et al., 2017). In addition to a substantial amount of clinical and experimental evidence in support of a functionally lateralized linguistic cerebellum (Mariën et al., 2001 a,b) a number of cases – including the ones in the current corpus – indicate that posterior fossa structures can be directly implicated in FAS [94] (see also Keulen et al., 2017). Damage to these structures is usually more directly associated with executive motor speech disorders of the dysarthric type and/or mutism (e.g. Frim & Ogilvy, 1995; Kumral et al., 2002), but deprivation of cerebello-cerebral connections the premotor areas for instance, could lead to AoS-like symptoms.

Based on the suprasegmental and segmental characteristics in FAS patients, it seems that FAS should be seen as a dual component motor speech disorder with both articulatory planning and executive deficits explaining the shared characteristics with ataxic dysarthria and AoS, quite possibly depending on lesion type and extension as well as the functional connectivity changes induced by the (respective) lesion(s).

4.3. Cognitive deficits in vascular FAS

Stroke is often associated with some degree of cognitive impairment. Patel et al. (2002) estimated the prevalence of cognitive deficits in the stroke population between 11.6% and 56.3% and indicate that 38% of the stroke patients in their study (total: n=645) were cognitively impaired at three months post-stroke (MMSE scores: <24/30). In post-stroke aphasia, cognitive deficits are common, and a relationship has been found between aphasia severity and cognitive deficits (Lee & Pyun, 2014). This review shows that cognitive deficits in FAS were not systematically looked for. Cognitive impairments were only identified in nine out of the 60 stroke patients (or 15%). Eight of these patients presented with comorbid speech and language disorders [95]. This raises the question as to whether patients suffer more subtle vascular impairment than aphasic patients mentioned in previous studies, or inversely, whether FAS is a mild disorder generally arising in

patients whose brain (plasticity) allows them to better cope with vascular lesions.

The majority of the patients with cognitive deficits had lesions in the frontoparietal areas [96], for the cases described by Kuschmann et al. (2012, case 1 & 2) the lesions were not clearly specified and for the case described by Mariën et al. (2013) and Pyun et al. (2013), they were situated in the cerebellum and the basal ganglia respectively. Seven patients had problems with arithmetics [97].

Arithmetics requires intact short- and long-term memory functions, attention, and mental flexibility (further impaired in [98]; Alexander et al., 1986) and is subserved by mainly parietal and (pre)frontal cortices (see Rosenberg-Lee et al. 2011; Menon et al., 2000; Menon et al., 2002). Basal ganglia lesions, attested for [99], give rise to similar cognitive impairments as frontal lesions due to a network of fronto-subcortical loops (Casey et al., 2002, Delazer et al., 2004). The basal ganglia are implicated in a wide variety of cognitive functions, including executive functions, attention, visual perception, sequential processing and learning – which were variably disrupted in [100] – but also speech (Brown et al., 1997). These behavioural effects are demonstrated in vascular FAS cases with basal ganglia damage [101].

Interestingly, Reeves & Norton (2001), Blumstein & Kurowski (2006) and Schiff et al. (1983) related FAS to disruption of the cortico-striato-pallidal-thalamic pathway, consisting of many regions that cooperate in an integrated fashion to regulate many different cognitive functions. Although there are still many issues to be resolved, this circuit is clearly involved in various speech and language processes (production cerebellum was not included. Cerebellar lesions affecting cerebro-cerebellar connections, which involve the basal ganglia, can give rise to cerebro-cerebellar diaschisis and induce frontal-like cognitive and behavioural deficits due to functional disconnection with regions crucial to higher cognitive functions, but also in motor speech control (see Leiner et al., 1986, 1989, 1991, 1993; Middleton et al., 2000; Bailleux et al. 2008, 2010; Stoodley & Schmahmann, 2010). The most poignant example of the distant functional effect of cerebellar pathology is the case described by Mariën et al. (2013). Following a right cerebellar hemorrhagic stroke, neurocognitive testing in this patient revealed persistent deficits on a range of tasks[4]. Due to a constellation of executive, spatial, affective and linguistic deficits the patient was diagnosed with cerebellar cognitive affective syndrome (CCAS) or Schmahmann's syndrome (Schmahmann & Sherman, 1998; Manto & Mariën, 2015).

[4] For an overview of the results the interested reader is referred to Mariën et al. (2013)

4.6. Remission of FAS

In terms of remission, it was found that the foreign accent receded during follow-up in only 24.17% (or 21/87) of the patients suffering from organic-neurogenic FAS. This remission was due to natural progression. When considering only stroke cases, this figure decreases to 20%. For the stroke cases for whom remission was noted, FAS duration ranged from 1 day [102] to approximately 3 years [103]. For the cases with FAS resolution, all but three [104] had comorbid speech disorders [105]. None of these cases received therapy specifically targeting the FAS.

Garst and Katz (2006) have provided a quite elaborate overview of how they treat FAS patients. First and foremost, samples of spontaneous and repeated speech are collected over multiple occasions and these are analysed for error consistency. Linguistic and emotional prosody is assessed and especially prosodic stress and articulatory timing are targeted in therapy. It is suggested to use a mirror to increase visual feedback for articulator placement. Other authors have described different approaches: Dankovičovà and Hunt (2011) mainly focused on oromotor exercises in their patient. Moreno-Torres et al. (2013) suggest treating phonetic awareness and monitoring skills. They suggest that feedback training should be complemented by cholinergic drugs, since these have shown to be beneficial in treating some aphasic patients (Berthier & Pulvermüller, 2011). The patient in Pyun et al. (2013) did not remit completely, but did improve after intensive speech therapy administered 5 days per week. Therapy focused largely on sentential prosody. Jezek et al. (2015) give a detailed description of the treatment of their 57-year-old male patient who suffered vascular FAS. The patient received intensive treatment for six weeks, 1 hour per weekday. The following six weeks, the patient received another 30 minute session once a week. Therapy focused on prosody (including contrastive stress pattern use, and a dysprosody treatment developed for Parkinson patients), articulation (reading and repeating word lists, later also sentences and texts) as well as fluency. Articulatory training was further supported by electropalatogram (EPG) feedback. What is clear from the above is that treatment should be patient-tailored, specifically adapted to the needs and concerns of the individual, and this may well be the reason that so little information on therapy is available: the comorbid aphasia, apraxia of speech and/or dysarthria will be the primary focus as they are often perceived as the most invalidating.

Positive prognosis (remission as opposed to no remission) could not be associated with subtype, nor with the presence or absence of comorbid disorders based on the statistical analyses; not when taking patients of the two largest subtypes (organic-neurogenic and functional) together, and not when isolating the

stroke patients. This may have to do with a weak statistical power due to the small number of available reports and data, especially when subdivided according to subtype.

Although the difference in remission in organic-neurogenic compared to functional patients (38.89%) is not significant, it is interesting to note that Baumgartner and Duffy (1997) regarded swift remission during follow-up as a non-negligible factor in the differentiation between neurogenic and functional stutterers. In order to find out whether this difference also holds in FAS, more (detailed) data reports are necessary.

4.6. The prototypical FAS patient with organic-neurogenic impairment

The typical FAS patient seems to be a monolingual, right-handed female in her early fifties who has suffered a stroke or a cerebral trauma. The patient is mute in the acute stage, after which FAS emerges relatively quickly (in the stroke population usually within three weeks after the vascular incident). Lesions involve the premotor or motor cortex, basal ganglia but also areas of the brain that project to these regions, e.g. the cerebellum. FAS only rarely occurs as the only speech symptom and is often accompanied by aphasia (of the non-fluent type), dysarthria or apraxia of speech. Hence, FAS may initially be somewhat "covered up" by the more prominent effects of other speech and language disorders.

Patients themselves are aware of the accent (see e.g. Nielsen and McKeown, 1961, case 1; Critchley et al., 1964, case 3; Whitaker 1982; Blumstein et al., 1987; Coughlan et al., 2004; Avilà et al., 2004; Lewis et al., 2013), reports of anosognosia are rare (see e.g. Nielsen and McKeown, 1961, case 2; Ryalls and Whiteside, 2006; Christoph et al., 2004). Cognitive deficits are related to frontoparietal or basal ganglia damage and are variable in nature (although acalculia is regularly reported). Remission data is highly dependent on duration of the follow-up. For stroke patients, 20% of the reports mentioned a remission to a normal accent with durations ranging from 24 hours [106] to several years e.g. [107]. Etiology, gender and presence of comorbid speech/language disorders do not seem influence remission.

Although it seems that most of the reported patients are still in keeping with Whitaker's criteria, the question remains how to deal with the functional FAS and polyglot patients, who are disregarded in current criteria and nevertheless are being increasingly reported.

4.7. Whitaker's criteria

Although the criteria proposed by Whitaker (1982) are still widely applied in FAS research, they substantially narrowed the original concept of a change of accented speech by restricting the condition to: 1) monolinguals, with 2) acute CNS damage whose oral verbal output is 3) subjectively considered foreign on a purely perceptual basis by patients themselves, by acquaintances or by the investigator. Reviewing the literature on FAS since the first description at the beginning of the 20th century 27 cases were found which met all the Whitaker criteria for a FAS diagnosis [108]. This group represents 24.11% (or 27/112) of the total number of patients in this study. This group is small for several reasons.

Firstly, in Whitaker's definition the diagnosis of FAS strongly depends on the subjective perceptual interpretation of the speech by patients themselves, by acquaintances or by the investigator. As a result, much of the variability of perceived accents in perceptual FAS experiments follows from the lack of objective phonetic measures to semiologically identify FAS as a coherent and distinct motor speech disorder. As such, the diagnosis of FAS fundamentally suffers from subjective interpretation that crucially relies on the judgment of speech qualities based on the highly variable implicit saliency of segmental and suprasegmental speech features in interlocutors. As a result, FAS has been argued to reside in the ear of the beholder (Kurowski et al., 1996).

Secondly, several cases have been reported in which FAS did not clearly relate to central nervous system damage, or to an acute cerebral insult. Indeed, similar to Critchley's (1962; 1970) case 1, several other patients (20.54%; 23/112) [109] have been reported who developed FAS without demonstrable brain lesions on structural brain imaging with CT or MRI. Most of these fit the diagnosis of a functional FAS (16.07%; 18/112) [110] (see Keulen et al., 2016d for a review).

Thirdly, the last of Whitaker's criteria restricts FAS to patients with no evidence of being a speaker of a foreign language and this artificially restricts the condition to extremely small linguistic minorities of the world's population that stem from monolingual communities. This view on linguistic reality is too narrow as most of the world's population is bi- or multilingual. Twenty-six out of the 112 cases (23.21%) were described as polyglot[5] at the time of FAS onset [111]. For reasons of their bilingual or polyglot background, they do not meet Whitaker's criteria. However, as a change of accent and prosodic alterations might be an

[5] The authors are aware that concepts such as 'bilingualism' and 'polyglot' can be defined in different ways. For the purpose of establishing this corpus all types (compact, coordinated, subordinated, balanced, dominant - Fabbro, 2003) were included, without making distinctions. We entirely followed the authors' descriptions.

intrinsic symptom of pathological language mixing (Perecman, 1989), additional research is needed to discriminate multilingual patients with FAS from patients who developed an accent in the context of bilingual or polyglot aphasia, which would possibly exclude eight bilingual or polyglot vascular FAS cases, for which aphasic symptoms were noted [112].

Many of the remaining patients had knowledge of the language(s) and associated accents due to intense language contact (through, for instance, relatives), or a basic level of formal language education at school. Berthier et al. (2015) argued for the recognition of this disorder as a separate FAS variant. Indeed, in agreement with the hypotheses formulated by others such as Seliger et al. (1992), it could very well be that a disruption of a previously suppressed neural system leads to the surfacing of linguistic features the patient internalized at some point during linguistic development, but, for some reason, did not need and hence suppressed. Or, as Ojemann & Whitaker (1978) argued, languages learned at different ages are localized in anatomically distinct regions in the brain. This hypothesis has been at the center of debate for many years now and other confounding variables have complicated the issue: not only age of acquisition, but also type of instruction, and amount of language use play a key role in the functional organization of the polyglot brain (Kim et al., 1997; Dehaene et al., 1997; Hernandez & Li, 2007; Perani et al., 1998; Perani et al., 2003; Perani & Abutalebi, 2000).

5. Conclusion

This review has presented an overview of the demographics, lesion location(s), neurocognitive status, and remission data for a selection of case reports on FAS starting from Pierre Marie's paper. The status of FAS as a motor speech disorder was discussed and it was suggested that FAS is the result of an impairment affecting the planning and execution stages of the speech production process. Based on the reviewed characteristics, a description was given of the prototypical FAS patient, who adheres relatively well to Whitaker's criteria. However, the focus of this review was explicitly on vascular patients. In addition, the case reports in the corpus often did not include data on language background and did not assess the possibility of a (comorbid) psychiatric disorder, which underlines the limitations of this review.

It should be borne in mind that the majority of the cases defined as FAS or suggested to be FAS, comply with only one or two of Whitaker's criteria. It is suggested to broaden the definition of organic-neurogenic FAS to a dual speech disorder affecting both planning and execution in which segmental and/or suprasegmental changes lead to the impression of an altered accent by listeners

belonging to the same speech community, which can occur in the context of a variable etiology. Future research is needed to investigate the neurological, neurocognitive, and linguistic characteristics in detail and evaluate the possibility of comorbid psychiatric disturbances in order for therapy (development) to head in the right direction.

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8. Case codes

In order to make the article easier to read, series of FAS cases are referenced in the text by means of a single number between square brackets (LIST A, on the left hand side of table 8.1). The numbers listed after the numbers between square brackets in list A refer to the FAS cases that were identified in the literature search and which can be consulted in LIST B on the right and the supplementary materials.

Table 8.1
List of the series of patients referred to in the text (List A) and list of the single cases arranged by article reference (List B).

List A : Series of patients referred to in the text	**List B : Single cases listed by article reference**	
	Case number	***Publication in which the case is described.***
[1] 2, 17, 33, 34, 37, 105, 110, 112, 120, 136, 140, 145, 148, 160, 170 [2] 72-75 [3] 21, 66 [4] 132-135 [5] 51, 55, 126, 104, 149,144 [6] 27, 28, 29, 35, 36, 51, 50, 48, 60, 115, 66, 72, 73, 75, 96, 97, 99, 100, 107, 126, 163 [7] 1, 3-16, 18-20, 22-27, 28, 29, 35, 36, 40-46, 50, 48, 49, 52-54, 56, 57-59, 61-63, 65, 68-71, 72, 73, 75, 84, 85, 87-95, 96, 97, 99, 100-103, 106, 107, 111, 113-119, 121, 123-125, 127-130, 137, 139, 141-143, 146, 147, 150, 151, 153, 154, 155-158, 163,	1	Abel et al. (2009)
	2	Akhlagi et al. (2011)
	3	Ardila et al. (1988)
	4	Asogwa et al. (2016)
	5	Avila et al. (2004)
	6	Bakker et al. (2004)
	7	Berthier et al. (1991), case 1
	8	Berthier et al. (1991), case 2
	9	Berthier et al. (1991), case 3
	10	Berthier et al. (1991), case 4
	11	Berthier et al. (2015), case 1
	12	Berthier et al. (2015), case 2
	13	Berthier et al. (2016), case 1
	14	Berthier et al. (2016), case 2
	15	Bhandari (2011)
	16	Blumstein et al. (1987)
	17	Boatman et al. (1994)
	18	Carbary et al. (2000)
	19	Chanson et al. (2009)
	20	Christoph et al. (2004)
	21	Coelho and Robb (2000)

168, 169, 171, 172	22	Cohen et al. (2009)
[8] 33, 68, 100	23	Cole (1971), case 1
[9] 1, 3, 5, 7-10, 11-14, 16, 19, 20, 22, 25, 29, 35, 36, 48, 49, 50, 53, 58, 115, 65, 68-71, 72, 73, 75, 84, 87, 89, 94, 96, 97, 99, 101, 102, 105, 113, 114, 116-119, 121, 143, 146, 150, 151, 153, 156, 158, 163, 168	24	Cole (1971), case 2
	25	Cottingham and Boone (2011)
	26	Coughlan et al. (2004)
	27	Critchley (1962), case 1
	28	Critchley (1962), case 2
	29	Critchley (1962), case 3
	30	Critchley (1970), case 1
	31	Critchley (1970), case 2
	32	Critchley (1970), case 3
[10] 6, 115, 147	33	Croot et al. (2005), case 1
[11] 13, 14, 49, 53, 96, 97, 99, 100, 105, 151, 153, 163,	34	Croot et al. (2005), case 2
	35	Dankovičovà and Hunt (2011)
[12] 115	36	Dankovicova et al. (2001)
[13] 5, 13, 14, 19, 23, 24, 28, 52, 57, 65, 69, 71, 85, 89, 91, 96, 99, 101, 113, 119, 123, 137, 143, 147, 163, 168	37	Denes et al. (1995)
	38	Di Dio et al. (2006), case 1
	39	Di Dio et al. (2006), case 2
	40	Dilollo et al. (2014), case 1
	41	Dilollo et al. (2014), case 2
[14] 5, 14, 19, 57, 71, 85, 89, 91, 96, 99, 101, 163, 168	42	Edwards et al. (2005), case 1
[15] 4, 23, 24, 28, 65, 69, 113, 143	43	Edwards et al. (2005), case 2
	44	Edwards et al. (2005), case 3
[16] 1, 3, 11, 12, 16, 35, 50, 48, 59, 60, 61-63, 70, 72, 73, 75, 97, 100, 115, 116, 121, 125, 127, 141, 142, 153, 154, 158, 171	45	Edwards et al. (2005), case 4
	46	Edwards et al. (2005), case 5
	47	Edwards et al. (2005), case 6
	48	Fridriksson et al. (2005)
	49	Graff-Radford et al. (1986)
[17] 18, 92, 93	50	Gurd et al. (1988)
[18] 1, 4, 6, 15, 16, 22-26, 27, 28, 29, 35, 36, 40-46, 50, 48, 49, 52-54, 56, 58-63, 65, 71, 72, 73, 75, 85, 88, 90, 114-117, 121, 128-130, 137, 141, 143, 146, 147, 154, 156, 168, 171, 172	51	Gurd et al. (2001)
	52	Haley et al. (2010)
	53	Hall et al. (2003)
	54	Hoffmann (2008)
	55	Hwang et al. (2001)
	56	Ingram et al. (1992)
	57	Jezek et al. (2015)
	58	Jones et al. (2011)
[19] 3, 11, 27, 35, 42, 43, 70, 114, 117, 118, 124, 125, 155, 157	59	José et al. (2015), case 1
	60	José et al. (2015), case 2
[20] 11, 27, 35, 42, 43, 114, 117, 118, 124, 125, 155	61	José et al. (2015), case 3
	62	José et al. (2015), case 4
[21] 3, 70, 157	63	José et al. (2015), case 5
[22] 1, 3, 5-12, 15, 16, 18-20, 22-24, 26, 28, 29, 35, 36, 42-46, 48-50, 53, 54, 56, 57, 59, 61-63, 65, 71, 72, 73, 75, 84, 85, 87, 89, 91-	64	Kanjee et al. (2010)
	65	Karanasios et al. (2011)
	66	Katz et al. (2008)
	67	Katz et al. (2012)
	68	Keulen et al. (2016a)

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[24] 4, 25, 27, 52, 58, 69, 70, 88, 90, 102, 124, 125, 128-130, 155, 157, 163	71	Kurowski et al. (1996)
[25] 3, 7, 9, 11, 15, 16, 20, 22-24, 26, 28, 29, 35, 36, 43, 44, 48, 49, 50, 53, 57, 59, 62, 65, 71, 72, 73, 75, 84, 85, 87, 89, 95, 96, 97, 101, 105, 107, 113-116, 123, 127, 137, 139, 141-143, 146, 147, 150, 151, 154, 156, 158, 168, 171, 172	72	Kuschmann (2010), PFAS
	73	Kuschmann (2010), MFAS1
	74	Kuschmann (2010), MFAS2
	75	Kuschmann (2010), MFAS3
	76	Kuschmann et al. (2012), PFAS
	77	Kuschmann et al. (2012), MFAS1
	78	Kuschmann et al. (2012), MFAS2
	79	Kuschmann et al. (2012), MFAS3
	80	Kuschmann and Lowit (2015), PFAS1
	81	Kuschmann and Lowit (2015), MFAS1
	82	Kuschmann and Lowit (2015), MFAS2
	83	Kuschmann and Lowit (2015), MFAS3
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[29] 94, 118, 119	87	Lazaro-Perlado et al. (2013)
[30] 1, 103, 153	88	Lee et al. (2016)
[31] 63	89	Levy et al. (2011)
[32] 54	90	Lewis et al. (2013)
[33] 1, 3, 6, 7, 9, 10, 12, 15, 18, 19, 20, 22, 26, 28, 42-44, 46, 50, 48, 49, 56, 64, 65, 71, 75, 84, 87, 89, 91, 92, 94, 95, 96, 97, 103, 107, 111, 113, 114, 116-118, 123, 127, 137, 139, 141, 142, 143, 146, 147, 150, 151, 153, 156, 158, 168, 171, 172	91	Lippert-Gruener et al. (2005)
	92	Liu et al. (2015), case 1
	93	Liu et al. (2015), case 2
	94	Luzzi et al. (2007)
	95	Marie (1907)
	96	Mariën and Verhoeven (2007), case 1
	97	Mariën and Verhoeven (2007), case 2
	98	Mariën et al. (2006)
	99	Mariën et al. (2009), case 1
	100	Mariën et al. (2009), case 2
	101	Mariën et al. (2013)
	102	Marques (2015)
	103	Masao et al. (2011)
	104	Mendis et al. (2013)
[34] 3, 7, 9, 15, 20, 22, 26, 28, 43, 44, 48-50, 65, 71, 75, 84, 87, 89, 95, 96, 97, 107, 113-116, 123, 127, 137, 139, 141-143, 146, 147, 150, 151, 156, 158, 168, 171		
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	108	Moen (2006)
	109	Moen et al. (2006)
	110	Moen et al. (2007)
	111	Monrad-Krohn (1947)
	112	Moonis et al. (1996)
	113	Moreno-Torres et al. (2013)
	114	Munson and Heilman (2005)
	115	Naidoo et al. (2008)
	116	Nielsen and McKeown (1961), case 1
	117	Nielsen and McKeown (1961), case 2
[40] 15, 16, 20, 22, 24, 35, 43, 44, 48-50, 53, 57, 65, 73, 75, 85, 87, 96, 97, 101, 107, 113, 114-116, 139, 142, 146, 147, 150, 151, 154, 156, 158, 168, 171, 172	118	Paolini et al. (2013)
	119	Paquier & Assal (2007)
	120	Perera et al. (2012)
	121	Perkins et al. (2010), case 1
	122	Perkins et al. (2010), case 2
	123	Pick (1919)
	124	Polak et al. (2013)
	125	Polak et al (2013)
	126	Poulin et al. (2007)
	127	Pyun et al. (2013)
	128	Reeves and Norton (2001)
	129	Reeves et al. (2007), case 1
	130	Reeves et al. (2007), case 2
	131	Reeves et al. (2007), case 3
	132	Reuters (2015), case 1
	133	Reuters (2015), case 2
	134	Reuters (2015), case 3
	135	Reuters (2015), case 4
	136	Roque et al. (2012)
	137	Roth et al. (1997)
	138	Roy et al. (2012), case 1
	139	Roy et al. (2012), case 2
	140	Roy et al. (2015)
	141	Ryalls and Whiteside (2006)
	142	Sakurai et al. (2015)
	143	Schiff et al. (1983)
	144	Schroeder et al. (2016)
	145	Scianna et al. (2000)
	146	Scott et al. (2006)
	147	Seliger et al. (1992)
	148	Simon et al. (2001)
[41] 3, 9, 26, 73, 89, 137, 143	149	Tailby et al. (2013)
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[43] 7, 11, 23, 28, 29, 36, 59, 62, 71, 84, 105, 127, 141		
[44] 22, 35, 43, 44, 48, 53, 75, 96, 97, 101, 114, 139, 142, 147, 156, 168		
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[52] 3, 7, 9, 11, 29, 50, 57, 73,		

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[67] 20, 22, 23, 26, 28, 29, 43, 44, 50, 53, 57, 59, 62, 79, 84, 85, 89, 95, 105, 107, 114, 123, 137, 142, 143, 147, 150, 151, 154, 158, 168, 171, 172

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[72] 9, 10, 15, 20, 22, 42, 45, 57, 87, 91, 92, 93, 96, 97, 121, 142, 143, 147, 153, 169, 172

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150	Takayama et al. (1993)
151	Teymouri (2009)
152	Tokudo et al. (2015)
153	Tomasino et al. (2013)
154	Tran and Mills (2013)
155	Tsuruga et al. (2008)
156	Varley et al. (2006)
157	Van Borsel et al. (2005)
158	Van der Scheer et al. (2013)
159	Verhoeven and Mariën (2002)
160	Verhoeven and Mariën (2004)
161	Verhoeven and Mariën (2007)
162	Verhoeven and Mariën (2010a,b)
163	Verhoeven et al. (2005)
164	Verhoeven et al. (2013), case 1
165	Verhoeven et al. (2013), case 2
166	Verhoeven et al. (2013), case 3
167	Verhoeven et al. (2013), case 4
168	Verhoeven et al. (2013), case 5
169	Villaverde-Gonzalez et al. (2003)
170	Wendt et al. (2007)
171	Whitaker et al. (1982)
172	Whitty (1964)

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[108] 1, 3, 11, 12, 16, 35, 50, 48, 59, 61-63, 72, 73, 75, 97, 105, 115, 116, 121, 127, 141, 142, 153, 154, 158, 171

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Supplementary Materials :

Overview of published Foreign Accent Syndrome case reports. The current table only includes case reports published in national and international peer-reviewed journals, internal medical reports in national and internationally published hospital communications, conference proceedings, abstracts from oral and poster presentations (between 1907 - October 2016). The cases surviving the criteria for our review article are marked with an asterisk. The case reports are ordered alphabetically. Abbreviations are explained at the end of this document.

Case Reference Number	Age / Gender/ Handedness	Etiology	Lesion Location	Speech/language characteristics	Accent	Cognitive & Behavioral Symptoms
1 **Abel et al. (2009)***	60/F /R	Breast carcinoma metastasis	CT, MRI; 3 x 3 x 3 cm tumor lesion in the left anterior/inferior parietal lobe (postcentral gyrus), surrounding edema and mild mass effect Intra-operative cortical stimulation mapping (visual object naming): inconsistent hesitations during stimulation of the posterior superior temporal gyrus; no naming areas in the supramarginal gyrus or anterior angular gyrus	Onset: dysarthria and accented speech (FAS); immediate postoperative phase: increased dysarthria (communicated with pictures and writing); 3d post-operatively: WAB Auditory comprehension=60%, object identification=100%, reading comprehension=100%, writing=100%; with 2w postoperative: dysarthria receded; FAS persisted	American English → Swedish	Unaware of altered speech
2 **Akhlagi et al. (2011)**	40/M/R	Right temporo-occipital lesion	CT: right temporo-occipital ischemic lesion affecting the calcarine region.	Onset: mute, when speech recurred: BNT + Token test: only naming deficit, FAS, no dysarthria, no AoS	Farsi --> Yazdi/ Isfahani	NI
3 **Ardila et al. (1988)***	26/M/R	Infarction	CT-scan: Broca's area (left hemisphere)	At onset: mute with slow recovery of speech over the following weeks 1m post-onset: telegraphic style 2m post-onset: FAS, moderate agrammatism (moderate Broca's aphasia)	Spanish→ English-like	Normal

4 **Asogwa et al. (2016)***	34/F/NI	Schizophrenia	MRI, MRA: normal EEG: normal accent occurred when discharged and less psychotic (!)	Prosodic changes, consonant changes, and changes in vowels	American English → British English spontaneous reversal of accent at index episode of psychosis/stable(?)	NI
5 **Avila et al. (2004)***	51/F/R	Trauma	MRI: infarction right temporal lobe and left inferior frontal corona radiata MR angiography: right internal carotid artery obstruction secondary to dissection	at onset: mute with evolution to FAS selectively affecting L1 (Spanish), not L2 (French), L3 (English), L4 (Catalan), no aphasia for more than 2 years	Spanish → NI	NI
6 **Bakker et al. (2004)***	52/F/L	Multiple Sclerosis (for 20 y)	MRI: hyperintense WM lesions (left dorsolateral inferior frontal lobe, corpus callosum, left parietal lobe)	at onset: word finding difficulties, minor grammatical errors (no aphasia, dysarthria). four episodes of FAS in 5 years lasting 4 to 6 weeks: slurred and scanned speech, FAS (no dysarthria, apraxia)	Canadian English → Dutch	Decreased memory, labile emotions
7 **Berthier et al. (1991) (Case 1)***	70/F/R	Infarction	CT-scan: middle portion of the left precentral gyrus	At 48y: 2d mutism & FAS for one month; at 70y: mutism evolving in the acute phase to dysarthria & dysprosodia; 4w post-onset: normal fluency, mild dysarthria, abnormal prosody (FAS) but no aphasia	Spanish→ Slavic	Moderate bucco-facial apraxia (initial phase), normal cognition
8 **Berthier et al. (1991) (Case 2)***	58/M/R	Trauma	CT-scan: right fronto-parietal WM (beneath pre- and postcentral gyri)	5d after surgery: mutism, no aphasia (crossed aphemia) evolving to dysarthria, dysprosodia; At 2m post-onset: gradual development of FAS; At 1.5y: FAS still present	Spanish→Slavic-like	Normal
9 **Berthier et al. (1991) (Case 3)***	47/F/R	Hemorrhagic infarction	CT-scan: lesion involving left middle frontal gyrus	At onset: mute & aphasic (Broca's aphasia); At 3w: slow, hesitant speech with impaired articulation, melody and grammar); 1m later: functional communication, writing difficulties, FAS At 3m post-onset: FAS receded, still dysprosodic	Spanish→Hungarian -like	5m post-onset: normal
10 **Berthier et al. (1991) (Case 4)***	34/F/R	Trauma	Angiography: right posterior communicating artery & right middle cerebral artery aneurysm CT-scan: posterior superior margin of the left middle frontal gyrus	At onset post-stroke: mutism for 2d At day 2: effortful non-fluent speech, speech initiation defects, output limited to perseverations and single words, partial echo-answers, severe agraphia (transcortical motor aphasia) Post-surgery (surgery 20 days later): improvement for 2w but speech still dysarthric	Spanish → Slavic-like	Normal (2m after FAS receded)

				with staccato cadence & development of FAS After 3w: receded together with improvement of articulation		
11 **Berthier et al. 2015 (case 1)***	47/M/R	Stroke	CT: bilateral hemorrhages involving the left motor cortex and right insula-putamen region probably resulting from untreated hypertension or from sporadic cerebral amyloid angiopathy Neurological examination (8 months post-insult): revealed complete recovery of the right hemiparesis and improvement of language impairment	Acute: total mutism → verbal mutism (after 2 days) 20 days later: word and sentence comprehension: disturbed, expression and comprehension: normal, writing: disturbed. 8 months post- stroke: agrammatism, echolalia, moderate reading impairment, writing: normal, prosody: different/off, accent change	Cordobés (C-Argentina) Spanish → Italian/? → Argentinian without accent	Depression COWAT: impaired
12 **Berthier et al. 2015 (case 2)***	18/M/R	Cerebral abcess	CT and MRI: encapsulated abscess involving the L sensorimotor cortex with mass effect over the insular cortex and basal ganglia and perilesional oedema Post-surgical neurological exam: tongue deviation (right), 'pseudoperipheral' right facial and velum paresis Secondary to an incomplete anterior opercular dysfunction (Foix-Chavany-Marie)	Before surgery: emergency room with a 2-week history of fever, headaches, and vomiting. Upon admission: focal seizures affecting the right face and tongue with secondary generalization and on one occasion transient speech arrest After surgery: mute + aphonic + dysphagia Two weeks after surgery: fluent speech + Japanese accent Flat intonation	Guaranitico (N-E Argentina) Spanish → Japanese? → Argentinian without accent	COWAT: impaired
13 **Berthier et al. (2016) case 1***	27/M/R	Developmental FAS (Father: developmental stuttering)	MRI: venous angioma close to the head of the L caudate that crossed the medial frontal lobe white matter to drain into the superior sagittal sinus + mild dilatation of the L lateral ventricle DTI: no difference compared to controls, individual differences in some ROI's: Altered DTI-based parameters in the L hemisphere in the superior frontal gyrus, the middle frontal gyrus, and the anterior and posterior cingulate cortex	Slight segmental errors, esp. istakes on phonetic level: consonat strengthening, suprasegmental: ok, although amplitude differences were found in comparison of utterances of FAS with controls	Spanish --> French, American English, Argentinean, or Mexican Spanish, Rumanian, and Italian	OCD, depression, general anxiety, social phobia, PTSD, alexithymia, additional personality charateristics: hopelessness, apathy Impaired semantic fluency
14 **Berthier et al. (2016) case 2***	46/M/R	Developmental FAS	MRI: expanded perivascular spaces (EPVS) involving both insular cortices DTI: no differences compared to controls, individual differences in some ROI's, in the L superior frontal gyrus	Spatial and motor dysgraphia, fast speech rate (spontaneous speech), Consonant errors included nasalizations, denasalization, place and manner of articulation, vowel insertion, consonant insertion, consonant omission and metathesis	Spanish --> Spanish from Lerida (west Catalonia), South-America, Canary Islands, French, or "uncommon"	OCD, general anxiety, depression, additional personality characteristics: hopelessness, apathy Hayling test: abnormal, Rey - Osterrieth: delayed
15 **Bhandari (2011)***	55/M/?	Ischemia	Pre-seizure: CT: encephalomalacia (sign of ischemia) in the left parietal lobe, precentral	onset: 'foreign' accent, alterations of syllable	Texan English → Cockney English	Mental status: Montréal Cognitive Assessment: 29/30

			gyrus and middle frontal gyrus Following seizure: -Transthoracic echocardiogram - EEG - MRI: restricted diffusion in the left parieto-occipital region and in the left middle frontal gyrus, no mass lesion(s) - MRA: normal	structure, no change in tone, rate, pitch, no sound substitutions, perseveration, or echolalia, no difficulty with speech initiation, perseveration of syntax, no alexia, no agraphia, was able to copy a paragraph, no acalculia later that day: accent changed back to normal after the seizure, neurological status was otherwise unchanged		
16 **Blumstein et al. (1987)***	62/F/R	Infarction	CT-scan: 1) deep lesion to left lower sensory-motor cortex area; 2) small subcortical WM lesion deep left middle frontal gyrus and lateral to the putamen; 3) small left temporal gyrus lesion; 4) very small lesion in right middle frontal gyrus	Acute: sporadic agrammatism in speech, modest word finding difficulties, normal repetition, reading & writing; 9-14d: FAS	American English → Eastern Europe Accent (Slavic to French-like, Dutch of Scandinavian)	VIQ = 84; PIQ = 88, difficulties with complex calculations
17 **Boatman et al. (1994)**	52/M/R	Infarction	CT-scan: lesion of the left superior temporal gyrus extending to the parietal lobe	At onset: expressive aphasia, mild anomia, relatively intact comprehension Within weeks: acute aphasia receded leaving accented speech (dysprosody) After 6m: speech, including prosody within normal limits	American English (?) → Norwegian	NI
18 **Carbary et al. (2000)***	51/M/NI	Trauma	CT/MRI: pre-existing infarction of convolutions 2 & 3 of the posterior left frontal lobe	At onset: mute (catastrophic) with evolution to normal	NI → NI	Normal
19 **Chanson et al. (2009)***	39/F/R	Multiple Sclerosis	MRI: large L prerolandic WM lesion and many small lesions of the deep WM fMRI & DTI tractography: lesion near L corticospinal tract and facial motor area SPECT: R prerolandic, temporal and thalamic diaschisis; after 2m: normal	German accent (FAS) 2 days after subacute mild right facial palsy (no aphasia, dysarthria, apraxia); within a few weeks: remission FAS	French → German	Normal immediate memory, episodic memory, visuospatial abilities, frontal functions
20 **Christoph et al. (2004)***	28/M/R	Hemorrhage	MRI: left prerolandic cortex hematoma extending to the underlying white matter and involving the insula and frontal operculum Digital angiography: small AVM of distal middle cerebral artery	at onset: Broca's aphasia. in the following 2d: recovery of aphasia but FAS, fluent language, normal Token Test, normal BNT, normal fluency, normal oral and written comprehension, normal repetition, normal reading and writing after 1m: FAS completely disappeared	Portuguese→North American/Spanish/German/southern region of Brazil	Slight buccofacial apraxia and oral agility
21 **Coelho & Robb (2000)**	51/F/NI	Unknown	CT-scan onset: negative MRI-scan (>3d & 1m post onset): normal; EEG: normal	(At age 39) at onset: communicated through writing; After 2w: speech returned, left with pronounced accent that cleared after 4y (not formally	American English→French Canadian, Italian	Period of confusion Moderate nonverbal oral apraxia

				investigated) (At age 51) at onset: difficulty speaking; After 3d: slow and careful speech with mild word finding difficulties (able to read and spell); After 3w: inconsistent consonant and vowel distortions, WAB quotient=98, intact reading and writing		
22 **Cohen et al. (2009)***	58/F/R	Infarction	Stroke 1: MRI: stroke left frontoparietal junction After 3y: Stroke 2: MRI: R inferior cerebellar hemorrhage	Onset: aphemia; within hours: English with unlearned accent (FAS) for three years; 2nd stroke: remission FAS, normal speech	English with unlearned accent	NI
23 **Cole (1971) (Case 1)***	29/M/NI	Cerebellar anoxia	Clinically suspected cerebellar degeneration secondary to anoxia	Speech: dysarthric, slightly spastic and dysphonic	Ohian, Yiddish → Italian	No facial apraxia
24 **Cole (1971) (Case 2)***	58/F/NI	Infarction	Clinically suspected inferior pontine infarction	Acute: spastic, dysarthric and dysphonic	Ohian, Hungarian→Eastern European	Normal mental status, no facial apraxia
25 **Cottingham & Boone (2010)***	36/F/R	Minor TBI Conversion Disorder (?)	Motor Vehicle Accident CT: (head): normal Headaches 3 days after accident, facial numbness, weakness in R arm, speech difficulties: 10 days after accident Approx. 10 days post-onset: EEG, Brain MRA, MRI: normal, neurological examination: normal, but: AoS + left-sided give-way weakness (non-neurological sign), dysarthria Medical history: complex medical history, with several hospitalizations for symptoms not explicable by neurological cause (e.g. sudden hoarseness of voice)	Approx. 10 days post-accident: AoS and 'dysarthria' (?); BDAE & Dysarthria Profile: mild retrieval difficulties, hypernasality, halting, abrupt, telegraphic speech (inappropriate pronouns, subject-verb agreement, verb tenses), inconsistent grammatical errors 17-months post-MVA: patient reported reading problems (onset coincided with her 1st deposition in personal injury lawsuit) 22 months post-MVA: Speech characteristics: robotic speech, absent prosody, inconsistent difficulties with articulation, word-list learning task, dysfluent speech, omission of articles, prepositions, and plural endings, agrammatism	American English → Eastern accent (3 years after accident)	Behavior: 'overly earnest' and childlike, theatrical presentation. Cognition: (3 years post-onset) Finger tapping, Rey Word recognition + combination, Warrington Recognition Memory, WAIS-III, Rey-Osterrieth, RAVLT Effort, TMT, Stroop, Multilingual Aphasia Examination, BNT, verbal fluency (FAS), MMPI-2 - Impaired scores on WRAT 4 word reading, Stroop Test, MAE sentence repetition, verbal fluency, VIQ was 13 points lower than PIQ - passed for 10/13 indicators of feigned cognitive symptoms
26 **Coughlan et al. (2004)***	39/F/NI	Left internal capsule infarction	CT: left internal capsule infarction	Acute: dysphasia, dysarthria, dysphagia 3 weeks post-onset: amelioration of dysarthria, but onset of FAS: altered rhythm, stress patterns and prosodic features considered atypical for her accent + syllable-timed speech.	(Irish) English → French	NI
27 **Critchley (1962) (Case 1)***	49/F/NI	Minor trauma -> Neurosis	NI	NI	English→Welsh	NI
28 **Critchley (1962) (Case 2)***	48/F/NI	TIA	Left hemisphere	NI	French/English →French (drunken)	NI
29 **Critchley (1962) (Case 3)***	37/F/R	Stroke	Right hemisphere	At onset: mute, gradual production of single words, good comprehension, agraphia, alexia	English → Welsh	Character changes

				(dysphasia); 1y post-onset: FAS, adequate vocabulary, fluent speech, impaired writing (no dysarthria, aphasia or AoS)		
30 **Critchley (1970) (Case 1)**	This case report describes the same patient as in Critchley (1962) (Case 1)*. Cfr patient 27 for more details.					
31 **Critchley (1970) (Case 2)**	This case report describes the same patient as in Critchley (1962) (Case 2)*. Cfr patient 28 for more details.					
32 **Critchley (1970) (Case 3)**	This case report describes the same patient as in Critchley (1962) (Case 3)*. Cfr patient 29 for more details.					
33 **Croot et al. (2005) Case 1**	13/M/NI	Traumatic brain injury	NI	at onset: American accent (rhotic vowels and vowel quality) persisted approximately 5 months then disappeared 'overnight' after watching the movie 'Crocodile Dundee'	Australian English → American English	NI
34 **Croot et al.(2005) Case 2**	52/F/NI	Traumatic brain injury	NI	at onset: slurred speech, FAS. FAS diminished, but did not recede completely.	English → Middle or Eastern European	NI
35 **Dankovičovà & Hunt (2011)***	56/M/R	2003: Stroke 1 2005: Stroke 2	CT stroke 1: at 6m: normal MRI stroke 1: at 16m: small wedge-shaped area of porencephaly along anterior margin of the mid immediately left to the midline MRI stroke 2 (2008): posterior parietal atrophy superior to occipital sulcus	onset (2003): slurred speech; within a few days: improved speech but emergence FAS (no formal assessments); after 14 months: BDAE: poor non-verbal oral agility task (8/12), normal verbal agility (14/14); stroke 2 (2008): no significant changes of FAS	British English → Italian, Greek (patient grew up in Essex, to Greek mother who spoke English)	Normal except for non-verbal oral agility (BDAE), Apraxia test: transitive limb tasks (9/10), bucco-facial, intransitive limb and whole body tasks (10/10)
36 **Dankovičovà et al. (2001)***	43/F/R	Infarction	A CT scan: a sub-arachnoid hemorrhage (with evidence of bleeding around the basal system and right Sylvian fissure) and a large terminal carotid aneurysm. After neurosurgery: an extensive infarction in area of right MCA	Three weeks post-onset: mild dysarthria	English → Scottish	Impaired nonverbal agility
37 **Denes et al. (1995)**	28/F/R	Trauma	CT-scan: left frontal contusion; MRI-scan after 1y: bilateral hypodensities of the frontal WM	At onset: aphonia to dysphonia, dysprosodia, FAS (no aphasia or AoS) 1y post-onset: still FAS	Italian → English-like	NI
38 **Di Dio et al. (2006)**	This case report describes the same patient as in Critchley (1962) (Case 2)*. Cfr patient 28 for more details.					
39 **Di Dio et al. (2006)**	This case was also reported in Dankovičovà et al. (2001)*. Cfr patient 36 for more details.					

40 **Dilollo et al. (2014) Case 1***	46/F/?	TMJ surgery	MRI: inconclusive (artifacts of mandibular reconstruction: metal braces and implants form surgery)	Motor speech evaluation: no evidence of diminished strength or mobility of tongue, lips, jaw muscles. Timing and coordination of speech movements were normal, stress testing showed no reduction of accent over time, no speech deviations Three-year follow-up: still suffered from problems due to TMJ surgery (esp. difficulties with pronunciation of multisyll. words), Irish accent was still evident, but less consistently so, able to 'control the accent'	American English (Midwestern English) → (Northern) Irish accent	(Standardized tests not specified): battery that included tests examining visual and auditory learning and memory, executive functioning (planning, switching, word fluency – categorical and phonemic), an effort test, IQ test, a test that estimated pre-morbid IQ, academic achievement tests (spelling, arithmetic, reading comprehension), sensory-motor tasks, language tasks (sentence repetition, auditory and visual confrontational naming, visual receptive naming), personality tests (anxiety, depression, personality traits, psychopathology) *Results*: commensurate with expectations, above-expected up to very superior, no indicators of anxiety or depression were evident
41 **Dilollo et al. (2014) Case 2***	43/F/?	TMJ surgery	MRI: inconclusive (artifacts of mandibular reconstruction: metal braces and implants form surgery) Three-year Follow-up: migraines → MRI (hospitalized two times): no structural lesion(s)	Motor speech evaluation: no evidence of diminished strength or mobility of tongue, lips, jaw muscles. Timing and coordination of speech movements were normal, stress testing showed no reduction of accent over time, no speech deviations Three-year follow-up: mild word finding problems	American English (Midwest) → British English	Same as case 1 *Results*: mostly commensurate with expectations, performance on test of receptive vocabulary was almost one SD below expectation, executive functions, attention and concentration: below expectation, no indication of elevated levels of anxiety or depression Three-year Follow-up: no neurocognitive testing, but: patient complained of two episodes (not simultaneous with headaches) where she experienced 'complete memory loss' (now: leaves messages to herself in order not to forget)

42 **Edwards et al. (2005) Case 1***	70/F/NI	Traumatic hemorrhage	Left parietal/basal ganglia/internal capsule	acute: FAS: receded within 4 days (4 weeks follow-up), normal speech and articulation	English → Welsh	Confusion
43 **Edwards et al. (2005)** Case 2*	58/M/NI	Hemorrhage	Left basal ganglia	acute: Normal speech and articulation, FAS persisted (1 year follow-up)	English → Irish	NI
44 **Edwards et al. (2005) Case 3***	64/F/NI	Infarction	Left basal ganglia/internal capsule	acute: impaired articulation, agrammatism, paraphasias, FAS persisted (9 years follow-up)	Scottish → Dutch/ Swedish Russian/ German	NI
45 **Edwards et al. (2005) Case 4***	18/M/NI	Trauma	Multiple small left hemisphere contusions	normal speech and articulation, FAS receded at 3 weeks (3 months follow-up)	English → American	NI
46 **Edwards et al. (2005) Case 5***	53/F/NI	Trauma	Left motor cortex and subcortical contusions	Normal articulation, transient aphasia, agrammatism, paraphasia, FAS persisted (4 years follow-up)	English → Dutch	NI
47 **Edwards et al. (2005) Case 6**	This case is also reported in Gurd et al. (1988). Cfr case 50 for more details.					
48 **Fridriksson et al. (2005)***	45/M/R	Infarction	MRI: small lesion left putamen MRI tractography: nerve fibers extending from the internal capsule to the corona radiata spared fMRI: normal activations during overt picture naming, increased activity in central sulcus and ventral angular gyrus	at onset: severely slurred speech turned into FAS after 2 hours, at six weeks: no aphasia, no dysarthria, no apraxia, normal word finding, diminished but still distinct FAS, fluent speech	American English → French, Greek, British English	Memory, executive functioning: normal, increased sense of smell, taste and appreciation for musical harmony
49 **Graff-Radford et al. (1986)***	56/F/R	Infarction	CT-scan: Brodmann area 6 & WM anterior and superior to the head of the left caudate nucleus	At onset: dysarthria, sparse, labored speech, incomprehensible writing (transcortical motor aphasia) After some days: FAS 8 m post-onset: FAS and decreased oral word association and slow reading, lengthened utterance durations, pauses, inaccurate stresses, sporadic vocal shifts	American English → Nordic	NI
50 **Gurd et al. (1988)***	41/F/R	Infarction	CT-scan: lesion extending 1.5 cm lateral to the genu of the left internal capsule involving the lentiform nucleus	At onset: mute -> moaning within hours -> mild dysarthria & FAS (no aphasia); 8m post-onset: residual FAS, diminished verbal fluency	English → French/ German	Slight micrographia

51 **Gurd et al. (2001)**	47/F/R	Multiple Sclerosis?	MRI: bilateral frontal WM, left frontal corona radiata, left thalamus, cerebellar vermis	Transient aphasia (follow-up 16m)	North Yorkshire → French-like	Impaired nonverbal and verbal agility
52 **Haley et al. (2010)***	36/F/NI	Conversion Disorder	MRI: normal MR-angio: normal	onset: slurred speech, abnormal 'cadence'; 14d: fluent in naming and conversation but FAS; subsequent months: frequent relapses heavily to lightly accented, "child-like" speech; 4 months post-onset: FAS, formally excluded aphasia (BNT, BDAE, Cognitive Linguistic Quick Test), no dysarthria	American English → French, Spanish, Jamaican, Caribbean, African	NI
53 **Hall et al. (2003)***	53/F/R	Ischemic Infarction	MRI: body of the corpus callosum	at onset: fluent, staccato speech, disturbed melodic line, altered intonation, stresses and pauses, mild fluent aphasia, mild alexia, mild agraphia; after 1y: diminution of staccato speech, written and oral comprehension problems of complex sentences	English → French, French Canadian	No facial or limb apraxia, no tactile anomia
54 **Hoffmann (2008)***	63/F/NI	Cranial trauma (whiplash injury) or viral infection Garcin syndrome	MRI: at three occasions over a follow-up of 2 years = normal. CT: normal Angiogram: normal	Onset (1w post-trauma): intermittent voice alteration (French accent), no aphasia (normal BNT); after 3y: remission of accent, residual mild dysarthria with fluent speech and good comprehension	American English→French	MMSE = normal
55 **Hwang et al. (2001)**	40/F/R	Infarction?	MRI at onset: normal SPECT on day one: focal hypoperfusion defect left lateral temporal region and crossed cerebellar diaschisis MRI at 2y: normal SPECT at 2y: normal	No aphasia, dysarthria or AoS but minimal decrease of fluency (by self-report), tone errors, FAS; within 3d: almost complete remission of FAS; at 2y: occasional problems with tone in daily conversations	Mandarin → American English-like accent	Normal
56 **Ingram et al. (1992)***	56/F/NI	1) Hemorrhage, 2) Trauma	CT-scan: lesion involving left lentiform nucleus	At onset: aphasia and AoS After 1y: FAS and occasional paraphasias	Australian English → Asian, Swedish, German-like	Oral apraxia
57 **Jezek et al. (2015)***	57/M/	Stroke (Left MCA)	CT: Left MCA infarction, focal lesions in pons	Initially: mute → communication through writing After 2-3 hours: difficult articulation → Russian accent	German→ Russian, Slavic	NI
58 **Jones et al. (2011)***	39/F/R	Conversion Disorder with mixed	CT, MRI: normal EEG: normal	onset: mutism; 2-3d: slow rate, articulatory and prosodic abnormalities FAS (no dysarthria or neuromotor	American English → Jamaican	At 18m: severe impairment in several domains including memory, executive functions, language, fine motor skills; deficits in general cognition,

		presentation		deficits); at 20 months: FAS, profound muscular weakness (-4 SD inspiratory and respiratory strength); high-pitched, tremulous and breathy dysphonia in isolated tasks not in connected speech; variable hypernasality; severe right-sided lingual deviation with protrusion; speech sound distortions; tongue weakness; multiple prosodic abnormalities		attention, working memory, auditory comprehension (concerns regarding validity) MMPI: conversion-V profile; NEO-PI-R: very low range on neuroticism scale; SCL-90-R: elevation on the somatization scale (T=65); below 2 SD on STAI
59 **José et al. (2015, case 2)***	70/F/NI	Stroke (MCA)	NI	prosodic, rhythmic/syllable structure alterations and segmental distortions	English → French	NI
60 **José et al. (2015, case 2) = 105 = 106**	64/F/R	Subarachnoid hemorrhage (right frontal medial)	NI	Prosodic, rhythmic/syllable structure alterations and segmental distortions	English → Italian	NI
61 **José et al. (2015, case 3)***	47/F/NI	Cerebral vasculitis	NI	Prosodic, rhythmic/syllable structure alterations and segmental distortions	English → German/Polish	NI
62 **José et al. (2015, case 4)***	62/F/NI	Stroke (MCA)	NI	Prosodic, rhythmic/syllable structure alterations and segmental distortions	English → French	NI
63 **Jose et al. (2015, case 5)***	37/F/NI	PANDA related action dystonia	NI	Prosodic, rhythmic/syllable structure alterations and segmental distortions	English → Asian	NI
64 **Kanjee et al. (2010)**	This case is also reported in Naidoo et al. (2008). Cfr case 115 for more details.					
65 **Karanasios et al. (2011)***						
66 **Katz et al. (2008)**	46/F/R	Unknown	CT and MRI; moderate ventriculomegaly and frontal lobe atrophy	2004: slow, measured, dysarthric speech following an allergic reaction to iodine contrast; 2005: complaints of sounding foreign (FAS); lexical substitution; BDAE-3 subtests=normal; mild anomic aphasia); AIDS: single word intelligibility=90%, sentences =96%; ABA-2: mildly impaired diadochokinetic rate, polysyllabic utterance time and repeated trials subtest; moderate impairment for increasing word length (mild AoS); fatigue increased accent	American English → Swedish, Russian, Eastern European	Complaints of short-term memory difficulties and sustained attention to competing stimuli; MMSE=26/30; Trail Making Test=normal; D-KEFS=reduced cognitive flexibility and mild perseverations; normal limb and oral praxis
67 **Katz et al. (2012)**	This patients was also described in Katz et al. (2008). Cfr. Patient 66 for more details.					
68 **Keulen et al. (2016a)***	17/M/R	Developmental Apraxia of Speech	MRI: Normal SPECT: bilateral hypoperfusion distributed in medial prefrontal regions and both lateral temporal regions.	Several segmental (consonants and vowels) errors, suprasegmental changes	Dutch (Brabantine) → French	Above-average IQ Visuo-constructive competence and visuo-motor integration: problematic Abstract concept formation (executive functions):

			Decreased perfusion in the left inferior medial frontal region, right inferior lateral frontal region and right cerebellar hemisphere nearly reached significance.			pathological score on WCST
69 **Keulen et al. (2016b)***	40/F/R	Psychogenic (conversion disorder?)	2005: psychiatric ward (anxiety, aboulia, depressive symptoms) 02/2010: sudden FAS, stuttering, agrammatism, nuchal pains, athralgia, attention problems CT, MRI, EEG: normal 06/2010: Münchausen syndrome/conversion disorder? 07/2010: speech problems + gait problems: EEG: normal, MRI: normal 04/2011: psychiatric ward admission CT + EEG: normal agrammatism 08/2012: resolution of FAS after general anesthesia for appendectomy, agrammatism in writing only	2010: FAS, agrammatism, stuttering 2011: FAS and stuttering: diminished; agrammatism = still present 2012: FAS and stuttering resolved, agrammatism persisted in writing only FAS charactierzed by vowel and consonant changes as well as prosodic changes	French → Slavic, Russian, Romanian, Dutch	Verbal memory, logical memory (WMS-R) Rey Complex Figure (< pc. 10)
70 **Keulen et al. (2016c)***	33/F/R	Psychogenic: bordeline personality	12/2011: minor head trauma; CT: normal 1 week later: repeat CT + EEG: normal 11/2012: MRI: normal	No aphasia, no comorbid disorders but isolated morphological errors (articles) FAS characterized by slow speech rate, subtle segmental changes, accent fluctuated	French → Dutch	2012: WAIS: dissociation between verbal IQ (96) and performance IQ (120) Stroop: slow processing speed d2: slow processing + accuracy : pathological range 2014: WMS-R: dissociation between verbal memory index (74) and visual memory index (133), general attention index was in clinical range (70)
71 **Kurowski et al. (1996)***	45/M/R	Infarction	CT & MRI 2y post-onset; 1) left posterior supramarginal gyrus 2) extensive lesion lateral putamen, lateral part of anterior limb of internal capsule, frontal PVWM, anterior & superior to the head of the caudate nucleus	At onset: global aphasia 4m post-onset: mild Broca's aphasia, slight dysarthria 2y post-onset: normal formal language investigations, normal prosody, FAS	American English → British, Scottish, Irish, Eastern European-like	Slight frontal lobe impairment and impaired digit span; PIQ = 115; normal memory, gnosis, orientation, calculation, oral praxis
72 **Kuschmann** **(2010) PFAS**	61/F/R	2006: Stroke 1 (age 60), at 3w: Stroke 2	Stroke 1: Left hemisphere Stroke 2: ?	onset: slurred speech, no FAS; at 3w (stroke 2): FAS; following months: less pronounced accent	British English → French, Italian, Eastern European, Jamaican	
73 **Kuschmann** **(2010) MFAS1**	49/F/R	2006: Infarction (age 47)	Left hemisphere	onset: mutism for a few days; After gradual return of speech: FAS and aphasia	Scottish English → Italian, South African	
74 **Kuschmann (2010)**	This patient was also reported in Dankovičovà & Hunt (2011). Cfr patient 35 for more details.					

MFAS2						
75 Kuschmann (2010) MFAS3	53/M/R	2007: CVA	Left temporo-parietal infarction	onset: mutism; when speech returned: FAS (aphasia ?, AoS ?)	British English → Italian (as judged by native speakers of English) of English, Polish or Eastern European (as judged by native speakers of Italian)	
76 Kuschmann et al. (2012) Case 1*	This patient was also reported in Kuschmann (2010) as PFAS. Cfr patient 72 for more details.					
77 Kuschmann et al. (2012) Case 2 *	This patient was also reported in Kuschmann (2010) as MFAS1. Cfr patient 73 for more details.					
78 Kuschmann et al. (2012) Case 3.	This patient was also reported in Dankovičovà & Hunt (2011). Cfr patient 35 for more details.					
79 Kuschmann et al. (2012) Case 4*	This patient was also reported in Kuschmann (2010) as MFAS3. Cfr patient 75 for more details.					
80 Kuschmann & Lowit (2015) – case 1	This patient was also reported in Kuschmann (2010) as PFAS. Cfr patient 72 for more details.					
81 Kuschmann & Lowit (2015) – case 2	This patient was also reported in Kuschmann (2010) as MFAS1. Cfr patient 73 for more details.					
82 Kuschmann & Lowit (2015) – case 3	This patient was also reported in Dankovičovà & Hunt (2011). Cfr patient 35 for more details.					
83 Kuschmann & Lowit (2015) – case 4	This patient was also reported in Kuschmann (2010) as MFAS3. Cfr patient 75 for more details.					
84 Kwon & Kim (2006)*	71/F/R	Stroke	MRI: left temporoparietal infarction MR angiogram: normal	20m before: Wernicke aphasia, change of province accent noticed 2d after onset when she started to speak relatively fluent; after 20m: no dysarthria, no AoS, anomic aphasia (WAB=76.5), disturbed reading and writing, affective prosody intact, linguistic	Cholla-buk province accent→ Kwangwon province accent	Slightly depressed

				prosody impaired		
85 **Laures-Gore et al. (2006) Case 1***	64/M/NI	Series of strokes: in 1977, in 1998 and in 1999	1998 MRI: right posterior temporal parietal infarction and an older infarction in the left basal ganglia and left cerebellum 1999 CT: no new infarct	1999: acute aphasia, perceived as FAS; WAB = normal (quotient of 94.8), Apraxia Battery for Adults=mild AoS, no dysarthria	American English → Chinese, →Dutch, or Canadian accent	NI
86 **Laures-Gore et al. (2006) Case 2**	67/F/NI	?	MRI at age 59: no lesions	at age 43: acute change in speech =FAS at age 67: WAB Quotient = 97.5, substitution of 'yes' by 'si', Apraxia Battery for Adults=normal to mild AoS, no dysarthria	American English → Spanish or Jamaican accent	NI
87 **Lazaro-Perlado et al. (2013)***	46/M/R	Hemorrhage	CT: left cerebral hematoma, comprising the striatum and internal capsula CT with contrast: no active bleeding, no structural anomalies in the Circle of Willis, or arteriovenous malformations that can justify a hemorrhage,	onset: FAS, no aphasia 3 years later: remission	Spanish → Galician	Behavior: OCD with (motor) tics and hoarding behavior (predating FAS and stroke), Cognition: 2 m post-stroke: MMSE: 30/30, Addenbrooke Cognitive Assessment (Spanish): 92/100; Luria test: ok 6 m post-stroke: MMSE: 30/30, Addenbrooke Cognitive Assessment (Spanish): 95/100; Luria test: ok
88 **Lee et al. (2016)***	59/F/NI	Psychogenic FAS – post-traumatic stress disorder	Facial trauma (tripped): lost front teeth CT: normal MRI: area of right occipital white matter high signal; this was thought to be consistent with a perinatal event and not relevant either to the head injury or the current presentation Dental surgery (repair teeth after accident): accent change soon after anesthesia for dental surgery	Occasional segmental errors, speech sometimes sounded telegraphic, used German sounding words in her speech	Scottish English → Scandinavian, German, fluctuating accent 'German-like' - Scottish (reversal noted on two occasions), patient could also imitate posh English and Irish accent, after therapy: periods of remission interspersed with accented speech Accent 'reinforced' sense of altered identity	Addenbrooke' s Cognitive Assessment: normal sense of altered identity post-traumatic stress disorder
89 **Levy et al. (2011)***	42/M/R	Infarction	CT angiogram: left internal carotid dissection CT at 5d: massive left fronto-temporo-parietal lesion; at 3w: more discrete	Onset: non-fluent, agrammatic aphasia without AoS or dysarthria, formally tested with BDAE and BAT Follow-up (3 weeks): morpho-syntactic and lexical retrieval deficits, slow speech, hesitations and self-corrections. Largely intact comprehension in 3 languages but strong Hebrew accent in English and French Time of the study: residual aphasia with French production most impaired, followed by English. Production in Hebrew most preserved	Hebrew (native language) → stronger Hebrew accent in American English (learned at age 10) and French (learned at age 16)	NI

90 **Lewis et al. (2013)***	54/F/NI	Mania	CT (without contrast): during exacerbation: no structural lesions	FAS: changes affecting vowel and consonant articulation, intonation and melody	North Eastern Carolina American English → Caribbean English	Neuropsychological testing after recovery: no specific diagnostic pattern
91 **Lippert-Gruener et al. (2005)***	35/F/NI	Trauma	CT: traumatic hemorrhage left temporal lobe (cortico-subcortical)	at onset: amnestic aphasia to severe global aphasia in German (retained capacity to obey commands and to speak a few words in English) evolved to an amnestic aphasia (in German) and dysprosodia; at three months: minor amnestic aphasia and FAS, use of English and Spanish impossible, at six and 12 months: slight improvement; at two years: complete recovery	German → English (at three months)	None at three months
92 **Liu et al. (2015), case 1***	41/F/?	Trauma	CT: left temporal subdural hematoma, contusion in L temporal lobe MRI: confirmation of CT	NI	Accent lasted 10 days Local dialect → Mandarin?	NI
93 **Liu et al. (2015), case 2***	25/M/?	Trauma	CT: contusion in L temporal lobe and hematoma in dura mater (R hemisphere), diffuse axonal injury. MRI: refused by patient	NI	Accent lasted 1 month Local dialect → mandarin?	NI
94 **Luzzi et al. (2007)***	64/F/R	Nonfluent Primary Progressive Aphasia	MRI; baseline: normal; after 1y: mild left perisylvian atrophy SPECT after 1y: left frontotemporal hypoperfusion	Since about 3y: gradual development of Spanish accent, no aphasia, dysarthria or apraxia; after 1y: hesitant speech, phonological paraphasias, Spanish accent, mild agrammatism, mild to moderate impairments in naming, reading, writing and repetition. Normal oral and written comprehension (mild nonfluent aphasia)	Italian → Spanish	Baseline assessments: normal; after 1y: no change
95 **Marie (1907)***	NI/M/NI	Stroke	Left hemisphere involving the lenticular region	At onset: mute for nine years (anarthria); 9 years post-onset: FAS, no aphasia	French Parisian →Alsatian	NI
96 **Mariën & Verhoeven (2007) Case 1***	53/F/R	Infarction	CT/MRI: left hemisphere lesion involving inferior frontal gyrus, precentral gyrus, anterior insula, postcentral gyrus and supramarginal gyrus SPECT: left frontoparietal hypoperfusion extending to left temporal and adjacent subcortical regions, crossed cerebellar diaschisis SPECT after three years: unaltered supratentorial perfusion pattern, improved perfusion in the right cerebellum	at onset: verbal mutism for one day evolving to AoS and FAS (no aphasia); 11d –27d post-onset: apraxia of speech and FAS (no dysarthria nor aphasia); 36m post-onset: remission FAS, mild residual apraxia of speech	Dutch → French, Russian-like	Above average cognitive results (IQ, memory, language, praxis, gnosis, concentration, problem solving, executive functions)
97 **Mariën & Verhoeven**	61/M/R	Hemorrhage	CT: left hemisphere hemorrhage involving the	at onset: verbal mutism, global aphasia; after 6	Dutch -> North-	1m postonset: disrupted arithmetics

(2007) Case 2*			putamen, genu and posterior limb of internal capsule with extension to the posterior insula, the medial temporal lobe and the paraventricular white matter of the parietal lobe SPECT: 1m postonset: severe hypoperfusion left thalamus, lentiform nucleus, left medial and lateral temporal region and left motor cortex, crossed cerebellar diaschisis; 6m postonset: remission right cerebellar hypoperfusion, unchanged perfusional deficits at the supratentorial level	days: conduction-like aphasia and FAS; 1m postonset: conduction aphasia and agrammatism, FAS, very high speech rate, omissions and consonant deletions, harsh voice quality, syllable-timed and isochronous speech rhythm, groping; 6m postonset: near remission of auditory-verbal and written comprehension deficits, normal naming, residual conduction aphasia, improved speech rate, improved groping and speech rhythm, resolved FAS	African	
98 **Mariën et al. (2006)**	This patient was also reported in Mariën & Verhoeven (2007) as Case 1. Cfr patient 96 for more details.					
99 **Mariën et al.(2009)***	29/F/R	Developmental AoS	CT, MRI & SPECT; normal	Developmental apraxia of speech (normal oral praxis; aphasia formally excluded)	Native speaker of Dutch perceived with a French-like accent since early childhood	TIQ=88; VIQ=97; PIQ= 81; Verbal MQ=100; Visual MQ=74; normal frontal planning and problem solving; normal visual cognition; normal mood and behavior
100 **Mariën et al. (2009)***	7/M/R	Specific Language Impairment	CT, MRI, EEG; normal SPECT: hypoperfusion in the vermis (-1.96 SD), both lentiform nuclei (left -2.25; right -2.30 SD), left thalamus (-3.30 SD), bilateral occipital lobes	Non-fluent speech with phonemic paraphasias, telegrammatic style, agrammatism, normal receptive language skills and written language (SLI: phonological-syntactic syndrome) (normal oral praxis)	Native speaker of Dutch → French-like accent since early childhood	TIQ=124; VIQ=115; PIQ=128; normal visual cognition; normal praxis; normal gnosis; normal problem solving, normal concentration, working memory and verbal memory
101 **Mariën et al. (2013)***	71/M/R	Cerebellar stroke (right)	CT: hemorrhagic lesion in right CB, secondary hydrocephalus and bilateral calcifications in the globus pallidus SPECT: a significant decrease and aggravation of perfusion deficits bilaterally distributed in the prefrontal brain regions during follow-up MRI (6 months post-stroke): complete resorption of the cerebellar hemorrhage, atrophy of the right cerebellum (e) and restoration of the intraventricular volumes	onset: FAS (reversion to a previously learned accent) after 10d: akinetic mutism, after resolution: ataxic dysarthria + echolalia + FAS After 1m: spontaneous speech reappeared, FAS and foul language + foreign words (German in his Dutch) Posterior Fossa Syndrome	Dutch → German	1m and 6m post-stroke: depressed MMSE, pathological FSIQ, VIQ and PIQ (WAIS-III), pathological scores on all subtests of the RBANS, pathological scores on WCST, pathological scores on Stroop and TMT` 6m: especially decrease in executive and behavioral tests Cerebellar Cognitive Affective Syndrome (CCAS)
102 **Marques (2015)***	48/F/R	Dissociative motor disorder (associated with psychiatric problems - marital problems)	MRI: normal EEG: normal Laboratory exams: normal	NI	Portuguese → German (duration at least 4 months)	NI
103 **Masao et al. (2011)***	30/F/NI	Resection of oligoastrocytoma	CT (2011?): left fronto-temporo- parietal MRI + fMRI (axial T2-weighted) as well as	Pre-surgical (neuropsychological) examination: language was coherent and consistent, normal	Spanish (Mexico) → American English	NI

			tractography of pyramidal tracts (2011?): confirmation left fronto-temporo- parietal Antecedents: progressively intensifying headaches stretching over the left hemisphere	volume, average speed, logical thinking, Spanish native language, left hemisphere dominant for language (DICOTEST). Post-surgery outcome: right hemiparesis and transient changes in language, prosody characteristic of FAS 1 month post-surgery: Expressive language was coherent, logical and normal in its content. Changes in prosody with characteristics of an American English accent, segmental deficits, no aphasia, no dysarthria. 4 months post-surgery: accent persisted		
104 **Mendis et al. (2013)**	49/F/NI	Electrocution resulting in brain injury, loss of consciousness	MRI: no abnormalities	Language assessment at hospital: reduced lip movement, altered vowel articulation, altered voice quality, altered volume The second speech and language session: the patient was taking beta-blockers and was noted to have a significant increase in dysfluent speech, a reduced coordination of breathing and voice, and an increase in coughing. Assessment in a multidisciplinary voice clinic: inappropriately loud, un-coordinated voicing, monotone, flat-pitched voice, and inconsistent volume control, segmental distortions and hypernasality	Northern British English → Polish	Increased anxiety and muscular tension after loss of consciousness
105 **Miller & O'Sullivan (1997)**	64/F/R	Right anterior aneurysm (junction A1-A2) rupture (SAH)	NI	At onset: stutter-like (no aphasia, dysarthria or AoS)	English → Italian/Polish/ Czech	NI
106 **Miller et al. (2006) = 105***	This patient was also reported in Miller & O'Sullivan (1997). Cfr patient 105 for more details.					
107 **Moen et al. (1990)***	61/F/NI	Stroke	Left hemisphere	Acute; FAS (no dysarthria, no aphasia) A few months post-onset: staccato, deviant pitch patterns	Norwegian→English -like	NI
108 **Moen (2006)**	This patient was also reported in Moen et al. (1990). Cfr patient 107 for more details.					
109 **Moen et al. (2006)**	End 20s F/NI	MS/psychogenic?	MRI: large subcortical lesion in the right parietal hemisphere. Smaller lesions a.o. near Broca center.	At onset: very serious speech problems, (had to write to make herself understood). Deviant pronunciation of consonants and vowels. Weak voice, pain during swallowing, normal sensation in face. Duration at least 3 years and 5 months	Norwegian→ Russian	NI
110 **Moen et al. (2007)**	59/M/NI	Infarction	CT; left parietal infarction	At onset: aphasia and dysarthria After a few months: improved aphasia, mildly	Norwegian → unidentified accent,	Reduced psychomotor tempo and sustained attention, memory disorder (storage, recall,

				agrammatic, word finding difficulties, FAS (no formal indications for AoS), dysarthria (voice problems, reduced phonation and reduced pitch variation, monotonous)	possibly of Eastern Europe	working memory), oral apraxia
111 **Monrad-Krohn (1947)***	30/F/NI	Trauma	Left fronto-temporo-parietal	At onset: mute, complete aphasia with steady improvement to single words, agrammatism; 7m post-onset: dysphasia, word finding deficits, FAS, intact comprehension; 15m post-onset: improved speech, considerable dysphasia, groping, dysarthria; After 2y: fluent speech, paraphasias, mild agrammatism, paragraphias	Norwegian→German or French-like	Infantile, manic, no emotional control
112 **Moonis et al. (1996/1993 abstract)**	59/M/R	Trauma	EEG & MRI-scan: normal SPECT 5m post-onset: focal hypoperfusion left inferior dorsolateral frontal lobe & asymmetric response in caudate/putamen perfusion: left < right	2 & 18m post-onset: abnormal melodic line, mild articulation problems, FAS (no aphasia)	American English→French-like	Normal except for slowed Stroop performance
113 **Moreno-Torres et al. (2013)***	44/F/R	Stroke (Initially: classified 'psychogenic', diagnosis based in the fact that the initial 1,5 T MRI only disclosed small lesions 'unrelated to symptoms' and the brain PET was normal on visual inspection – analysis with SPM was not performed) Antecedents: In her 20's: Positive history of general lymphadenopathy due to toxoplamsa In her 30's: traumatic cervical injury Since adolescence: recurrent episodes of migraine with aura	Initial MRI (1,5 T): disclosed three small confluent infarctions involving the left intrasylvian region (frontal operculum + anterior insula) Initial PET: normal Approx. 17 months after first scans: MRI (3 T): small confluenct left hemisphere infarctions located in the inferior frontal gyrus and insula. Largest infarction involved the deep frontal operculum in junction with the ant. insular cortex. DTT: reconstruction suggested reduced intrainsular connectivity an also between areas of infarctions PET: L. hemisphere: significantly decreased metabolic activity in comparison to normal control subjects in cortical and subcortical brain regions implicated in speech processing (inf. frontal gyrus pars orbitalis, dorsal AIC, subgenual cingulated, temporo-polar region, lingual gyrus, putamen, insula,, medial/internal globus pallidus + cerebellum, reduced R. hemisphere metabolism was found involving the medial frontal gyrus, fusiform gyrus, parahippocampal gyrus.	17 months before current examination: R. herpes labialis followed by R. facial paresthesia and weakness involving upper and lower divisions of R. facial nerve Following days: decreased speech intelligibility during prolonged speaking, poor speech articulation, eventually mute, but could spell (minor spelling errors; letter transpositions): hypothesis of an anterior opercular syndrome (Foix-Chavany-Marie). Auditory + reading comprehension was intact, when anterior opercular syndrome resolved (1 week later) recovery of verbal output was slow (labored, hypophonic, …) 17 months after stroke (linguistic analyses in Spanish): Further testing: segmental errors, suprasegmental changes, linguistic and emotional prosody was disturbed in casual conversation	Catalan/Spanish → Czech/French	17 months after stroke (linguistic analyses in Spanish): WAB (98/100), spontaneous speech was fluent and abundant with normal volume, mild articulation struggling and abnormal rhythm with poor melody. WAB apraxia (60/60). PALPA (nonword minimal pairs= 54/56, word minimal pairs= 55/56, lexical decision= 159/160, word repetition= 23/24, and nonword repetition= 22/24) and semantic processing (spoken-word picture matching= 40/40) 17 months post-stroke: MMSE (30/30), WMS-III: average performance in word list learning (list A total = 38/48, list B = 6/12, short-term recall of list A = 10/12; recognition = 24/24), digit span score= 8, Rey Osterrieth Complex figure: copy (36/36), delayed reproduction (21.6/36), Trail- Making Test (A = 40s, B = 68.9s), Controlled Oral Word Association Task (45 nouns in 3 min),

114 **Munson & Heilman (2005)***	49/F/R	Infarction	CT: left middle cerebral artery distribution MRI: left frontal opercular lesion (BA 44)	at onset: mute, intact auditory-verbal and written comprehension, normal spelling, after several days: Broca's aphasia and FAS, at one year: fluent speech, mild word finding difficulties, misarticulation of sounds, dysprosody and persisting FAS	Midwestern-type American dialect → German (follow-up of three years)	NI
115 **Naidoo et al. (2008)***	50/F/L	Ischemic stroke	CT; admission: chronic microangiopathic ischemia; day 2: left internal capsule, left basal ganglia and frontal corona radiata lesion, MRI: 1m: anterior & posterior limb of internal capsule, superior portion of lenticular nucleus	Onset: slight dysarthria, mild word finding difficulties; 5d post-stroke: slow speech rate, imprecise consonants, monopitch, monoloudness, breathy voice quality, word finding difficulties; 14d: no change in speech; 1m: 100% speech legibility at the Assessment of Dysarthric Speech Test, BDAE: impaired recitation skills (1/17), 0 on BDAE melody task, word finding difficulties (BNT 6/15 with circumlocutions, hesitations, semantic paraphasias) changes in phonological segments and expressive prosody; 1.5m post-stroke: mild expressive deficits (word finding, expressive vocabulary, confrontation naming, semantic and phonemic verbal fluency); at 3m: greatly improved, word finding difficulties	Native Southern Ontario accent → Canadian East Coast accent (= regional variant)	at 1.5m: right-sided visual inattention, moderate inefficiencies in processing speed, mild reductions in mental control & working memory; mild executive dysfunction
116 **Nielsen & McKeown (1961) (Case 1)***	65/M/R	Thrombosis	Left middle cerebral artery	At onset: mute, motor aphasia, dysarthria, dysprosodia, apraxic agraphia	American English→Swedish	Moderate acalculia
117 **Nielsen & McKeown (1961) (Case 2)***	38/M/R	Trauma	Left hemisphere contusions	Acute: Non-specific dysarthria, grammatical errors	American English→Swedish	IQ above average
118 **Paolini et al. (2013)***	78/F/R	Primary Progressive Aphasia (nonfluent variant – PPA NFV)	MRI (no date): cortical atrophy, especially in the left temporal hemisphere SPECT: hypoperfusion of lateral frontal regions especially in the left hemisphere, as well as in lateral and medial temporal lobes	03/2008: language disorder characterized by dysprosodia, sporadic phonologic and semantic paraphasias (evolving over 2 years). Symptoms began with progressive change in loudness and pitch of voice, speech was perceived as having undergone a regional accent change. Language evaluation: agrammatic, dysprosodic, anomic spontaneous speech with phonologic errors and rare semantic paraphasias, prosodic comprehension impaired for affective and linguistic prosody, unaware of dysprosody, moderate naming impairment and low phonemic fluency. Normal semantic fluency, single word comprehension was good for nouns and slightly impaired for verbs. Sentence comprehension	Marche accent (central Italy) → Veneto accent (North-east of Italy)	Behavioral changes: disinhibition, impulsiveness, euphoria and irritability Neuropsychological Testing: MMSE: 24/30, follow-up MMSE (12 months later): 9.7; initially a mild frontal executive dysfunction was evident, no deficits in visual and verbal episodic memory, visuospatial abilities and object/people knowledge. Impairment in short-term memory was found with a low performance in the Digit Span test. Also showed bucco-linguo-facial apraxia 12-month follow-up: a relative preservation of time and space orientation, memory in daily

				was severely affected, repetition and writing of non-words were severely impaired, a mild deficit was found for words. Reading ability was good for both words and non-words. 12-month follow-up: severe worsening on oral production and comprehension 24-month follow-up: almost mute and oral comprehension was severely impaired, but able to read and to recognize familiar faces.		living, visuospatial abilities and selective attention. A mild executive deficit was also confirmed. 24-month follow-up: Her family members confirmed an overall worsening of her behavior with impulsivity, aggression and disinhibition.
119 **Paquier & Assal (2007)***	88/F/R	Binswanger disease (subcortical leukoencephalopathy- vascular dementia)	CT: profound subcortical vascular leukoencephalopathy, confluent lesions more pronounced bifrontally (L>R)	Recurrent episodes with spontaneous spelling aloud of auditory and visually perceived words and non-words, FAS, fluent speech, impaired denomination and auditory comprehension, relatively intact semantic knowledge (80%), partially preserved repetition, syntax, orthography, some semantic paragraphias, semantic errors in oral text reading, morphophonological paralexias, omissions of words, tendency to vocalize punctuations marks, normal oral spelling, code switching when speaking French	French→ Japanese	Not collaborative, resistant, irritable, agitated, anosognosia, suspicious (no hallucinations or delusions) MMSE=5/30; DRS= 26/144 but not severely demented, basic ADL preserved, partial orientation, utilization behavior, repetitive motor behavior, environmental dependency syndrome
120 **Perera et al. (2012)**	30/F/NI	Ischemic infarction	MRI: Ischemic infarction in the left corona radiata and left basal ganglia	Acute: vowel distortions, inconsistent consonant conversions and significant deviations in pitch pattern	English → German	NI
121 **Perkins et al. (2010)***	48/F/R	Trauma	MRI (25 days post-onset): abnormal signal intensities in corona radiata + bilateral centrum semiovale EEG: normal	onset: slurred speech 4 weeks post-onset: FAS	American English → Eastern European	NI
122 **Perkins et al. (2010)**	This patient was also reported in Fridriksson et al. (2005)*. Cfr patient 48 for more details.					
123 **Pick** **(1919)***	26/M/NI	Stroke	Left hemisphere	At onset: mute, aphasia -> fluent, hesitant, paraphasic, agrammatic output, disturbed repetition of unfamiliar names, alexia, agraphia; 4m post-onset: improved aphasia but more prominent FAS	Czech → Polish	Could not sing
124 **Polak et al. (2013)** **Case 1***	47/M/?	Refractory OCD (for > 25 years) - psychological + pharmacological treatment: no symptom relief	March 2006: 2 DBS electrodes → treatment MRI: pre-operative CT: post-operative No structural lesions (pre- & post-)	Deep Brain Stimulation: DBS after stimulation: speech change: frequently started speaking with an accent commonly used in his native region, accent change co-occurred with hypomanic episode - ! when stimulation parameters were increased to higher voltage: OCD symptoms decreased further, but hypomanic behavior and accent increased, linguistic changes were reported after the adjustments of parameters (until last follow-up in 2011)	Standard Dutch → Regional Dutch accent	- Yale – Brown Obsessive-compulsive Score: decrease from 33 to 18 after stimulation - After cognitive behavioral therapy: from 18 to 5 points

125 **Polak et al. (2013)** **Case 2***	65/M/?	Refractory OCD (> 50 years)	December 2006: 2 DBS electrodes → treatment	After DBS: the patient's pronunciation became very distinguished, more coarse language + swearing than before DBS and more hyperactive behavior - Hypomanic behavior seemed to be related to adjustment of the parameters, the induced accent remained unaffected by this readjustment - Accent remained present	Regional variant of Dutch → 'distinguished' Dutch	- Yale – Brown Obsessive-compulsive Score decreased from 33 to 30 after DBS - After cognitive behavioral therapy: further decrease from 30 to 8
126 **Poulin et al. (2007)**	74/M/R	?	MRI; asymmetric atrophy left temporal, frontal opercular/insular region (post-hoc) 18F-FDG PET: hypoperfusion bilateral in frontal, parietal and temporal lobes and focal deficit in the left anterior temporal lobe with prominence of the sylvian sulcus	January 2003 at consultation for follow-up exacerbation of bipolar disorder: FAS and agrammatism July 2005: acute exacerbation of bipolar disorder long-lasting FAS and agrammatism; no apraxia, dysarthria, aphasia, German and Spanish sounding non-words coming to his mind	Québec French→ Acadian French, English-like accent	Working memory deficit, executive dysfunctions
127 **Pyun et al. (2013)***	37/F/	Stroke	Brain CT (admission): intracerebral hemorrhage, left basal ganglia DTI (2 months after stroke): disconnected left arcuate fascicle at the mid-portion adjacent to the hemorraghic lesion. The number of fibers was decreased in the left arcuate fascicle Follow-up DTI: similar morphological features. fMRI*: picture naming task:* in comparison to controls, patient showed different places of activation. More dispersed and extensive. Antecedents: 6y prior to stroke: diagnosed with Moyamoya at the time of her first intracerebral hemorrhage	One month after stroke: K-FAST= 16/30, aphasic symptoms showed characteristics of mixed dysarthria + moderate degree of buccofacial apraxia and AoS (inconsistent articulation errors and ineffective self-correction), strange accent (high pitched sounds + elevated the terminal parts of her sentences even when speaking assertively) Speech and language were re-assessed 1m, 6m and 12m after onset: K-WAB initially indicated Broca's aphasia, moderately reduced auditory comprehension, semantic paraphasias and misuse of postpositions in spontaneous speech were observed during reading After speech therapy, AQ evolved from 52 (1 month) to 83.4 (6 months) and 89.4 points (12 months), phonetic analysis of speech, segmental and suprasegmental distortions	Korean → English	One month after stroke: MMSE= 23/30, full cognitive assessment showed moderate to severe impaired performance in attention, memory and executive functions and the impairment was worse in auditory and verbal tasks. Patient received computer assistive cognitive rehabilitation twice a week for a year. Follow-up: 12m after stroke: marked improvement in nonverbal cognitive functions including vigilance and nonverbal intelligence (RCPM). Performance executive functioning worsened.
128 **Reeves & Norton (2001)***	65/M/NI	Psychosis (Schizophrenia/ Parkinson)	MRI: normal	No aphasia, dysarthria or AoS	American English → British English	Delusions and hallucinations
129 **Reeves et al. (2007) Case 1***	30/M/NI	Psychosis (schizophrenia)	MRI; EEG: unremarkable	at onset: melodic sing-song accent, dropping of consonants, BNT=60, FAS; 6d after olanzapine was resumed: remission of FAS	American English → Jamaican	Disorganized thoughts, bizarre behavior, grandiose delusions; 9d after treatment: improvement
130 **Reeves et al. (2007) Case 2***	53/F/NI	Psychosis (bipolar disorder)	MRI; EEG: unremarkable	at onset: FAS, rising inflection at the end of phrases, omission of final consonants, no aphasia (normal naming repetition, grammar and comprehension); after 15d of medical treatment: started to lose	American English → European	Elevated mood with racing thoughts, auditory hallucinations, paranoid delusions; 15d after medical treatment: improvement

				the accent; at day 17: no accent		
131 **Reeves et al. (2007) Case 3**	?66/M/NI	Psychosis (schizophrenia)	MRI; EEG: unremarkable	from 30y onwards: several occasions of FAS during episodes of psychosis; fluent speech, no aphasia or impaired naming or grammar at 66y in nursing home: Episode 1: FAS with incorporation of British terms into vocabulary; elongated vowels, substitution of vowels, substitution of consonants; 4w after medical treatment: resolution of accent; after 2y: Episode 2: FAS with remission after 9d treatment; 6w later: Episode 3: FAS that receded after 9d of treatment	American English → British	Episode 1: hallucinations, delusions, paranoid delusions, agitation; after 4w of medical treatment: persistence of grandiose delusions; after 2y: Episode 2: psychosis with remission after 9d treatment; 6w later: Episode 3: psychosis
132 **Reuters (2015), case 1**	NI/F/NI	NI	NI	mainly prosodic changes: syllable-timed speech, continuation contours, voicing of typically mute consonants, word finding difficulties	German → French	NI
133 **Reuters (2015), case 2**	NI/M/NI	NI	NI	Both segmental and suprasegmental changes, esp. pitch curve and syllable-timed rhythm was reminiscent of French	German → French	NI
134 **Reuters (2015), case 3**	NI/F/NI	NI	NI	Esp. segmental distortions	German → Turkish	NI
135 **Reuters (2015), case 4**	NI/F/NI	NI	NI	Both segmental and suprasegmental changes	German → American English	NI
136 **Roque et al. (2012)**	?/M/?	Ischemic stroke	MRI: ischemic stroke in left pre-motor cortex	FAS, no aphasia, no dysarthria, no AoS Remission after 72 hours after induction of permissive hypertension	British English → Caribbean English	NI
137 **Roth et al. (1997)***	45/M/NI	Hemorrhagic stroke	CT-scan: left parietal lesion with extension into the ventricles	At onset: mutism evolving to Broca's aphasia and FAS after 2m (no agrammatism or anomia)	American English→Dutch	NI
138 **Roy et al. (2012) Case 1**	75/M/R	(See 81)	(See 81)	FAS characterized by vowel and consonant changes, prosodic alterations	Québec-French speaking → Acadian French (French spoken in the eastern provinces of Canada)	NI
139 **Roy et al. (2012) Case 2***	63/F/NI	Ischemic stroke	CT: hypodense lesion in the left sylvian area, involving the fronto-parietal cortex	FAS, mild Broca aphasia (mainly characterized by a reduction in spoken fluency) and mixed agraphia	Québec-French → Germanic (German/Alsatian)	NI

140 **Roy et al. (2015)**	53/F/NI	? unknown	MRI and CT: normal Diagnosis of PPA was initially made, but later removed	Fluctuating FAS (very mild to moderate-severe), more present with fatigue; mild anomia; reading difficulties due to attention deficit, segmental and suprasegmental changes, agrammatism	Québec French → ? 'foreign accent'	Fluency tasks, Stroop, TMT-A & B, Hayling test, Digit span (forward and backward)
141 **Ryalls & Whiteside (2006)***	57/F/NI	Infarction	MRI: small lacunar infarction in left internal capsule	at onset: mute over the following 2m: recovery of speech and development of FAS with 'Briticisms', appropriate intonation, normal BDAE and BNT, no aphasia	American English →British, Australian English	CLQT= criterion level, cognitive functioning within expected age range
142 **Sakurai et al. (2015)***	42/F/?	stroke	MRI: 4 days post-onset (April 2007): focal infarction surrounding precentral sulcus (upper inferior frontal gyrus (BA6) to precentral gyrus at level of lower middle frontal gyrus). SPECT: 19 days post-onset: blood flow reduction in L sup. front. gyrus, post. middle front. gyrus, sensorimotor cortex, ant. and post. Supramarg. gyrus, subcortically in the medial globus pallidus. Accent was lost in February 2009, when she suffered 2nd stroke in corona radiata	Language evaluation based on WAB (Japanese edition) 12 days post-onset. dysfluent spontaneous speech, halting, inconsistent speed, strange pitch accent, and intonation, shortening of word pronunciation, segmental distortions affecting vowels and consonants, suprasegmental changes, agrammatism (Broca's aphasia), deletions.	Japanese → Chinese/South Korean	NI
143 **Schiff et al. (1983)***	58/M/R	Stroke	CT-scan: lower half of left precentral gyrus with subjacent WM involvement	At onset: mute; After 7d: mild aphasia After 3 w: dysarthria, slow speech, dysprosody, FAS, telegrammatic utterances After 1y: resolution of FAS, permanent dysarthria	Portuguese/English → Chinese	NI
144 **Schroeder et al. (2016)**	42/F/A or R?	possible CNS lesion induced by TMJ surgery (born with prominent mandibular prognathism, TMJ pain as of age 14)	TMJ: oral and maxillofacial surgery was performed at age 42 CT, MRI, EEG (4 months after surgery/onset accent): normal	acute (post-surgery): word-finding problems, British accent, difficulty with reading	American English→ British accent	Post-surgical emotional change (no depression, no anxiety), feelings of raised irritability Significant dissociation between general memory (117) score and WM (85) ($p < 0.05$), verbal fluency (-2 SD), WM (-1.9 SD), letter-number sequencing (-1.9 SD), digit span (-2.2 SD), Trail Making Test (switching: -2.2 SD), Verbal fluency (letters): -2.9 SD, Grooved Pegboard Right Hand (-2.1 SD) (but: prior right radial nerve decompression surgery, date unknown)
145 **Scianna et al. (2000)**	68/F/NI	Infarction	Left parietal lobe	Acute(?): Mild hypokinetic dysarthria & FAS; After 5m: FAS receded	American English → Irish brogue	NI
146 **Scott et al. (2006)***	54/F/R	Infarction	MRI: small left hemisphere lesion in WM underneath the precentral gyrus dorsal and	at onset: mute, FAS when speech returned; at 2y: fluent speech but sound production and	Scottish → German, Polish or South	At 2 y: WAIS-R: VIQ=94, PIQ=98 ('low' scores for arithmetic and picture completion), verbal

			medial to the anterior insula	grammatical errors, normal naming, normal comprehension, normal repetition (ignoring the sound production errors), normal writing and spelling	African	recognition memory just above chance level, visual recognition memory slightly below normal limits, normal Weigl sorting test
147 **Seliger et al.** **(1992)***	65/F/L	Infarction	MRI-scan: left centrum semiovale lesion	At onset: dysarthria and FAS Within 48h: marked improvement of dysarthria After 2w: moderate accent After 3w: notably faded accent At 4m post-onset: almost normal	American English → Northern Irish-like	NI
148 **Simon et al.** **(2001)**	49/F/NI	12/1999: Trauma and Dissection left internal carotid artery 06/2000: brain aneurysm	1999: CT: normal; Repeat CT: after 3d: normal MRI: left internal carotid artery occlusion EEG: moderate right temporal lobe disturbance	at onset: stuttering, mild syntactic errors, mild word retrieval difficulties, FAS; 09/2000: stuttering, broken speech patterns, sounding Jamaican, normal BNT, normal word fluency, BDAE Cookie Theft picture impaired, prosodic and segmental abnormalities; at 10m post-trauma: significant improvement of fluency and near normalization of prosody	American English → Jamaican/Caribbean accent	Wechsler Memory Scale: normal, normal oral motor function
149 **Tailby et al. (2013)**	37/F/R	TIA? → Mixed FAS Antecedents: viral encephalitis at 19, pneumonia, gastric banding surgery, ovarian cysts, migraine (one of the migraine attacks involved left-sided weakness and 'loss of sight' in her left eye)	CT of brain, 04/2011 (admission): no acute event CT of brain, 3 days after admission: no acute event MRI (day 12): no acute event, T2 hyperinstensities lateral to anterior horn of left lateral ventricle and in the WM lateral and posterior to atrium of lateral ventricle. DWI (11 days after admission): no hyperintensities fMRI (11/2011): (lexical retrieval paradigm - in-scanner covert version of COWAT + verb generation paradigm): left hemisphere dominant language lateralization (although left-sided weakness), comparison with 32 controls shows that language representation did not significantly differ from that of neurologically normal controls	On admission, 04/2011: slurred speech, 24 hours later (speech pathologist): oro-pharyngeal dysphagia, moderately dyspraxic articulation, slowed and effortful speech, patient spoke of word-finding difficulties 48 hours later (speech pathologist): buccofacial dyspraxia was also noted (/ar/ for /a/) Day 9 of admission: French accent during first fluent conversation (during argument with mother) Following months: speech alternated between at least 5 different accents, could not control the accents and experienced them proprioceptively rather than phonetically, noticed changes in her use of language and pragmatics accompanying each accent 05/2011: no buccofacial or upper limb dyspraxia 08/2011: native Australian accent had returned and patient reported voluntary control over her accent (French/South African) 11/2011: reports developing a foreign accent whenever she speaks with someone who has a foreign accent, normal BNT, normal COWAT	04/2011: FAS 1: Australian to French, Between 04/2011 – 08/2011: would wake up with either a French or South African or New Zealander accent, 08/2011: Australian to French and South African (controllable!) 18/04-25/04: slow effortful speech 26/04-16/05: French 17/05-20/05: South African 21/05-25/05: French 26/05-02/06: New Zealand accent 03/06-16/06: French 17/06: South African 18/06-19/06: French 20/06-26/06: Russian 27/06-02/07: French 03/07-05/07: French in the morning, Australian in afternoon 06/07-17/07: context driven accent change	Day 4: ataxic gait Between 04/2011 – 08/2011: personality changes congruent with each of the consecutive accents 11/2011: neuropsychological testing: WAIS: VIQ : 85, PIQ: 96, lower ability in language-related cognition is reflected in the WART full scale of 83, new verbal learning and figural recall: within normal limits, processing speed, attention function, working memory and delayed recall were below normative standards.

150 **Takayama et al. (1993)***	44/F/R	Infarction	MRI-scan 4w post-onset: lesion posterior lateral aspect of the left precentral gyrus (middle fifth)	At onset: mute After 2d: FAS	Japanese → Korean-like	NI
151 **Teymouri (2009)***	53/F/R	Ischemic stroke	CT & MRI: left centrum semiovale lesions	Onset: mutism and anarthria, normal reading and auditory comprehension, then FAS?; first m after stroke: gradual improvement of dyslexia and agraphia (weakness in punctuation remained after 2y) a change to a talkative attitude, phonetic, phonemic and morphological difficulties, monotone intonation (variations at 1 y), long pauses between words, wrong stress placement, no sound assimilation	Persian→?	?
152 **Tokudo et al. (2015)** **no translation available**	There was no translation available of this article in Japanese.					
153 **Tomasino et al. (2013)***	50/F/R	Low Grade Tumor	MRI: tumor (2.7x2.2x2.5 cm) in left premotor cortex (BA 6, 44, 4) fMRI maps: 6-10d prior to surgery: lip & tongue movements; silent object naming, counting, sentence and pseudoword pronunciation DES: counting & naming	Onset: episodes of focal seizures with speech arrests lasting several minutes; After 1m: onset progressive FAS, reading difficulties and problems linking different parts of speech; Intraoperative mapping: no changes of FAS; Post-tumor resection: normal speech, no FAS, no dysarthria, no AoS, no aphasia (formally excluded via Token Test; verbal fluency tasks; BADA phonemic, lexical and syntactic tasks; Robertson Test)	Italian → Rumanian, Ukrainian/Russian, South African	NI
154 **Tran & Mills (2013)***	60/F/NI	Infarction	Initial CT: unremarkable MRI (3 weeks after onset symptoms): subacute infarction of the left hemi-pons and absent flow within the left vertebral artery concerning for occlusion MRA: head+neck re-demonstrated left pontine infarction without hemorrhage, areas of irregular and narrowing in the anterior circulation + diminished flow in the posterior circulation, most pronounced in the basilar artery. MRA with contrast: high-grade stenosis of basilar artery, fetal origin of the left posterior cerebral artery, and multiple areas of narrowing in both MCA's	Initial presentation at emergency dep.: speech change was thought to be secondary to a goiter impinging in the recurrent laryngeal nerve 3 weeks after onset symptoms: presented again to ED, complained of ongoing altered speech pattern On examination: spoke fluently Jamaican / Italian accent, ending most words with '-ah', BNT= 50/60, epenthesis, adding '-uh' to end or middle of words	African-American → Jamaican/Italian	NI
155 **Tsuruga et al. (2008)***	44/F/NI	Dissociative (conversion) disorder	MRI & SPECT; normal	Two transient episodes of aphonia; last period of aphonia evolved into FAS (more significant when talking to family); no aphasia	Japanese → Chinese	TIQ=101; VIQ=96; PIQ=107; Panic disorder one year after her father's death

156 **Varley et al. (2006)***	40/F/R	Stroke	MRI: infarction anterior portion left middle cerebral artery territory and hemorrhagic changes left putamen	at onset: nonfluent aphasia, AoS, good resolution of aphasia but FAS; at 2y: mild aphasia, comprehension and naming functionally intact, normal repetition, slow reading and spelling difficulties, occasional errors of morphology and syntax, FAS	South Yorkshire accent → Swedish	Difficulties in divided attention listening situations
157 **Van Borsel et al. (2005)***	32/F/NI	Minor head trauma/ Psychogenic cause	CT: normal	at onset: mute (by self-report), no aphasia, no verbal apraxia, no dysarthria, deviant articulation, dysprosody, telegraphic style and grammatical anomalies not typical for Broca or Wernicke aphasia, FAS; at one year: resolution of FAS (within 5 months post-onset)	Dutch → Eastern-European, Russian, Slavonic, Romanian, Spanish, French, German, Turkish, non-European	Depression with suicidal ideation
158 **Van der Scheer et al (2013)***	59/M/R	Stroke	MRI: ischemic infarction in left MCA territory and tissue loss in the right occipital lobe consistent with a small, older infarction	After surgery: mute, later his speech improved During neurological examination: no signs of aphasia, only speech rhythm changes, diadochokinesis test was difficult to perform (AoS could not be ruled out, but valid Dutch AoS test was not ready yet), Radboud Dysarthria Test: no dysarthria present Speech and language examination: speech rate is low (2.39 syll/sec), altered vowel articulation	Dutch → French, German, Asian, but in perceptual experiment: Arabic, Turkish, Surinam, Eastern Dutch accent	NI
159 **Verhoeven and Mariën (2002)**	53/F/R	Infarction	CT/MRI: left hemisphere lesion involving inferior frontal gyrus, precentral gyrus, anterior insula, postcentral gyrus and supramarginal gyrus SPECT: left frontoparietal hypoperfusion extending to left temporal and adjacent subcortical regions, crossed cerebellar diaschisis SPECT after three years: unaltered supratentorial perfusion pattern, improved perfusion in the right cerebellum	at onset: verbal mutism for one day evolving to AoS and FAS (no aphasia); 11d –27d post-onset: apraxia of speech and FAS (no dysarthria nor aphasia); 36m post-onset: remission FAS, mild residual apraxia of speech	Dutch → French, Russian-like	Above average cognitive results (IQ, memory, language, praxis, gnosis, concentration, problem solving, executive functions)
160 **Verhoeven and Mariën (2004) = 96 = 98 = 159 = 161**	This patient was also reported in Verhoeven and Mariën (2002). Cfr patient 159 for more details.					
161 **Verhoeven and Mariën (2007)**	This patient was also reported in Verhoeven and Mariën (2002). Cfr patient 159 for more details.					
162 **Verhoeven & Mariën (2010a,b)**	This patient was also reported in Verhoeven and Mariën (2002). Cfr patient 159 for more details.					
163	51/F/R	Psychotrauma/	1995 (onset) CT, EEG: normal	at onset (1995): stutter-like speech and FAS, no	Dutch → French	Above average cognitive results (IQ, memory,

Verhoeven et al. (2005)*		Conversion disorder 1995: conversion disorder (MMPI) 2003: DIS-Q: normal	2003 CT, MRI, EEG: normal	aphasia, dysarthria, apraxia of speech, after eight years: no aphasia, apraxia of speech or dysarthria, iterations, effortful speech and inappropriate pauses, FAS originating from lexical, grammatical and pronunciation characteristics, mixing and code switching (FAS in English but not in French)		language, praxis, gnosis, concentration, problem solving, executive functions)
164 **Verhoeven et al. (2013) Case 1**	This patient was also reported in Verhoeven and Mariën (2002). Cfr patient 159 for more details.					
165 **Verhoeven et al. (2013) Case 2 = 97**	This patient was also reported in Mariën & Verhoeven (2007) as Case 2. Cfr patient 97 for more details.					
166 **Verhoeven et al. (2013) Case 3**	This patient was also reported in Verhoeven et al. (2005). Cfr patient 163 for more details.					
167 **Verhoeven et al. (2013) Case 4**	This patient was also reported in Mariën et al.(2009). Cfr patient 99 for more details.					
168 **Verhoeven et al. (2013) Case 5***	24/F/R	TIA (2004) Stroke (2005)	CT (2004): normal CT (2004): normal CT, MRI (2005): ischemic lesion in the left insular region SPECT (2006): hypoperfusion in the left motor and insular region (-6.00 SD)	2004: acute onset motor speech symptoms evolving to anarthria within a few hours, normal oral and written comprehension After 12 hours: complete remission of motor speech symptoms (mild residual bradylalia) 2005: sub-global aphasia, evolving within a few days to a mild nonfluent aphasia with agrammatism, AoS and FAS 2006: agrammatism in oral and written language, residual AoS (scanned speech, groping, phonematic errors)	FAS native speaker of English, late bilingual of Dutch (moved to Holland at 8 years of age but had no accent in Dutch) → reversion to a previously learned accent (Birmingham accent affecting Dutch)	2004: verbal apraxia 2006: verbal apraxia, normal memory, normal IQ, normal executive functions, normal attention skills
169 **Villaverde-González et al. (2003)***	38/F/NI	Multiple Sclerosis	CT: two hypointense subcortical lesions in both frontal lobes MRI: several hyperintense lesions at supratentorial (periventricular) and infratentorial level	at onset: slight agrammatism, dysprosody resembling French, decreased speech rate (no dysarthria, no aphasia) after 3m: normalization	Spanish → French	No orofacial apraxia,
170 **Wendt et al. (2007)**	35/F/R	Ischemic Infarction	Left middle cerebral artery infarction at the age of 33	2y before current investigation: Broca's aphasia, minor word finding difficulties and mild problems reading aloud, auditory and reading comprehension unimpaired, no alexia, no AoS, no dysarthria, FAS; current examination: problem with rhythmic	German → Russian	NI

				speech production not characteristic of German or Russian (segmental and prosodic errors)		
171 **Whitaker (1982)***	30/F/NI	Stroke	Left hemisphere	At onset: muteness evolving to Broca's aphasia with AoS and agrammatism evolving to FAS; Some months post-onset: fluent output & FAS	American English→ Spanish	NI
172 **Whitty (1964)***	27/M/NI	AVM	Craniotomy: AVM in vicinity of the left rolandic fissure	At onset: mute for 7 hours; then slow, hesitant and slurred speech; 3d post-onset: normal speech except for FAS and some dysarthric slurring of words, (apraxic?) dysgraphia; 7w post-onset: normal speech, no FAS	English → German	NI

Abbreviations and test references:

18F-FDG-PET=18F-fluordeoxyglucose-positron emission tomography, ABA-2= Apraxia Battery for Adults-2 (Dabul, 2001); Addenbrooke Cognitive Assessment (Mathuranath et al., 2000); ADL= activities of daily living, AIDS= Assessment of Intelligibility of Dysarthric Speech (Yorkston & Beukelman, 1981); AoS= apraxia of speech, AVM= arteriovenous malformation, AQ= Aphasia Quotient, BA= Brodmann area, BADA= Batteria per l'analisi dei deficit afasici. BDAE= Boston Diagnostic Aphasia Examination (Goodglass et al., 2000), BNT= Boston Naming Test (Kaplan et al., 1983), CLQT= Cognitive Linguistic Quick Test (Helm-Estabrooks, 2003); CT= computed tomography, COWAT= Controlled Oral Word Association Task (Benton et al., 1994), d= day(s), D-KEFS= Delis-Kaplan Executive Function Scale (Delis et al., 2001); DRS= Dementia Rating Scale (Mattis, 1988), DTI= diffusion tensor imaging, EEG= electroencephalogram, F= female, fMRI= functional magnetic resonance imaging, Grooved Pegboard (Ruff & Parker, 1993), h= hour(s), Hayling test (Burgess & Shallice, 1997);IQ= intellectual quotient, K-FAST= Korean version of Frenchay Aphasia Screening Test (Pyun et al., 2009), L= left-handed, l= left, M= male, m= month(s), MAE= Multilingual Aphasia Examination (Benton et al., 1994); MCA= middle cerebral artery, MMPI-2: Minnesota Multiphasic Personality Inventory-2 (Butcher et al., 1989); MMSE= Mini-Mental State Examination (Folstein et al., 1975), MRA= magnetic resonance angiogram, Montréal Cognitive Assessment (Nasreddine et al., 2005), MRI= magnetic resonance imaging, NI= not indicated, NEO-PI-R= Neuroticism, Extraversion, Openness Personality Inventory-Revised (Costa & McCrae, 1992); OCD= Obsessive-compulsive Disorder; PALPA= Psycholinguistic Assessments of Language Processing in Aphasia PIQ= performance IQ, PVI= pairwise variability index, PVWM= periventricular white matter, R= right-handed, r= right, RAVLT= Rey Auditory Verbal Learning Test (Rey, 1964); RCPM= Raven Colored Progressive Matrices (Raven, 1976; Raven and Court, 1998); SAH= subarachnoid hemorrhage, SCL-90-R= Symptom CheckList-90 Revised (Derogatis, 1992); SPECT= single photon emission computed tomography, Stroop = Stroop Test (Golden et al., 1978), TIA= transient ischemic attack, VIQ= verbal IQ, VOT= voice onset time, TIQ= total IQ, TMT= Trail Making Test (Reitan, 1958), WAB= Western Aphasia Battery (Kertesz, 1982), WM= white matter, WAIS-III= Wechsler Adult Intelligence Scale III (Wechlser 1997); WAIS-R (Wechsler, 1981); Warrington Recognition Memory (Warrington, 1984); WMS-R= Wechsler Memory Scale-Revised (Wechsler, 1987), WRAT= Wide Range of Achievement Test (Wilkinson & Robertson, 2006), y= year(s), Yale-Brown Obsessive-compulsive Scale (Goodman et al., 1989).