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Revisiting the boundaries of different altered accents profiles

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Key words:

Accent, Altered Accent Profiles, Foreign Accent Syndrome, Neurotransmitter, Speech Production Network

This article is dedicated to the memory of Professor Peter Mariën

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Abstract

A speaker's accent is regarded as an essential aspect of their identity to the extent that its abnormal modulation may have several negative consequences. Although the so-called foreign accent syndrome (FAS) is the best-known impairment, other altered accent profiles (AAPs) are likely to be more common and often go undiagnosed. Despite significant progress during the last two decades, several consequences of AAPs have yet to be systematically evaluated. Moreover, a common finding in several previous reports is the incompleteness of diagnostic work-up and the rarity of studies reporting longitudinal evolution and treatment approaches. In disorders with presumed low prevalence like AAPs, it is imperative to carry out a comprehensive analysis, documenting not only the altered accent profiles but also the neural correlates and associated disorders affecting cognition, behaviour, quality of life, and outcomes after treatment. This article highlights how to move forward in the multimodal evaluation of altered accents by integrating information from different sources.

1. INTRODUCTION

Speakers differ in the number of languages they master, and, in the accents, they convey. There are more than 7,000 living languages (“Ethnologue, Languages of the World,” 2024); hence, it is tempting to speculate that the number of accents worldwide is even higher. While many studies have focused on language, research on accents has primarily been viewed as an accessory to language, thus attracting less interest in the neuroscientific community until recently. That is surprising because accents are a critical component of people's identity to the extent that social adscription to specific groups and judgments about others are often based on accent (Labov, 2006). In addition, attempts to voluntarily alter accents in non-native adult speakers to prevent discrimination are not always successful (Carlson & McHenry, 2006; Tan et al., 2021). Furthermore, accent can be altered in medical, neurologic, and psychiatric conditions (Mariën et al., 2019). The emergence of a persistent undesired accent is not without consequences, as it usually affects speakers psychologically and socially (Lippi-Green, 1994; Miller et al., 2011; Monrad-Krohn, 1947).

The original description of an undesired accent change associated with brain damage is attributed to Pierre Marie (1907), who described the case of a man whose Parisian native accent had changed to Alsatian after a stroke. Although this case is often cited as the first case of foreign accent syndrome (FAS)¹, in the strict sense of the word, what Marie described was a change in regional accent. Since this pioneering report, several variants of FAS have been described (*see below*), yet owing to the presumed rarity of FAS variants, the differential characteristics of these discrete syndromes are only beginning to be identified (Barreto & Ortiz, 2020; McWhirter, Miller, et al., 2019; Romö et al., 2021), not without fruitful controversies on classification (Berthier & MorenoTorres, 2019; McWhirter, Miller, et al., 2019; McWhirter, Stone, et al., 2019).

In this article, we propose to replace the term Foreign Accent Syndrome (FAS) with the more generic term Altered Accent Profiles (AAPs), a terminological change that concurs with the suggestion made by Blumstein and Kurowski (Blumstein & Kurowski, 2006)², who recommended finding a more generic designation to supersede the term FAS (*see also* Kurowski et al., 1996). Hence, the label FAS should only be applied to those speakers who develop a manner of speaking in one or more languages resembling a foreign accent. There are reasons for adopting the label AAPs. On revising the literature, it was found that the FAS label is commonly applied to classify accent changes even though the affected individual shows, for example, a change in regional accent or the return to a previously non-used accent that already belonged to the person's repertoire (Levy et al., 2011; Roth et al., 1997; Seliger et al., 1992). Also, the new designation has some advantages over the traditional one. Using a broad term may cover *all* new cases, even if it can be accommodated to include situations where another accent replaces the native regional accent. Moreover, the designation of AAPs could be

¹ Foreign accent syndrome is a rare condition in which a person develops speech patterns that are perceived as a foreign accent (Blumstein et al., 1987).

² Blumstein and Kurowsky (2006) rightly highlighted that persons with FAS do not acquire the phoneticphonological production of a given actual foreign or regional accent; instead, their verbal output only "sounds like" as if they were native of several different languages.

advantageous because it focuses on the many factors implicated in the native-like accent change and not exclusively on the precise speech disturbance.

At the same time, this new term raises questions requiring attention and discussion. The first question has to do with the definition of the term accent. The second question is how AAPs should be analysed. As far as the first question is concerned, an accent has been described as the collection of pronunciation characteristics which typify individuals or groups of speakers. It is a 'way of speaking' exhibited by some people, not others. Although this definition of accent is very much rooted in the phonetics of speech (segmental and suprasegmental characteristics), accents potentially reveal a wealth of evidential information about the identity of speakers, such as their physical characteristics (weight, height, gender, age), their personality, state of health, social status, geographical origin, and language background (Abercrombie, 1967; Bestelmeyer et al., 2015; Laver & Trudgill, 1979; Lyons, 1977; Verhoeven, 2002). Accents have to do with how an individual or group of individuals pronounce a language, and accents make up the vocal identity of speakers. Accents can be assumed to be informative, so it may be helpful to consider accents against a semiotic framework (Abercrombie, 1967; Laver & Trudgill, 1979; Lyons, 1977; Verhoeven, 2002). Based on Abercrombie (1967), Lyons (1977) and Laver & Trudgill (1979), it is helpful to distinguish different types of indexical information:

(1) Individuating markers are those that characterize individuals. They inform about a speaker's age, weight, height and gender.

(2) State markers, those characteristics that reveal that the speaker is in a particular state: emotional (fear, anger, sexual arousal, readiness), state of health (healthy or suffering from a cold), state of intoxication (under the effects of drugs and alcohol).

(3) Group markers, which identify individuals as members of a particular group they belong to (in-group) and others not belonging to that group (out-group) (Bestelmeyer et al., 2015). Lyons distinguishes between region-identifying (geographical background of the individual), status-identifying (social status), and occupation-identifying (profession).

Each of these aspects is informative in that they may make listeners aware of something they were unaware of (Lyons, 1977: p.33), irrespective of the speaker's intentions. Based on this taxonomy, it is tempting to define accents more narrowly as the ensemble of linguistic and phonetic characteristics which mark speakers as belonging to a particular group (group markers). AAPs such as neurogenic FAS represent speech disorders in which speakers cannot encode their habitual group markers. It is relevant that losing this ability may have perceptual consequences for the attribution process by listeners, which may cause additional emotional distress in the affected individual. In contrast with neurogenic FAS, psychogenic FAS would not be a speech deficit proper. Instead, it would be secondary to underlying psychiatric disorders that may alter the patient's group identity. Thus, psychogenic FAS may reflect conscious or unconscious and variable successful attempts to produce the group markers of this new identity.

A second crucial question is how AAPs should be assessed. Since current knowledge suggests that different interactive processes are involved in the genesis of AAPs, implementing a transdiagnostic approach may enable an analysis of these disorders from a much broader perspective (DiLollo et al., 2014; Miller et al., 2011; Moreno-Torres et al., 2013; Tomasino et al., 2013). Some initiatives in this direction have been taken (Gurd & Coleman, 2006; Keulen, 2017; Moreno-Torres et al., 2016). This article integrates information from different

perspectives to analyze the key similarities and differences in AAPs associated with various neurologic, psychiatric, and medical disorders. Regardless of aetiology, AAPs probably share neurobiological mechanisms, thus making the boundaries between clinical presentations fuzzy. That encourages the development of a transdiagnostic framework for examining evidence of neural and behavioural signatures of AAPs (Craske, 2012; McFayden et al., 2022). The article concludes by formulating several predictions that need confirmation and some outstanding questions for future research.

2. DIAGNOSTIC CRITERIA

The first attempt to operationally define AAPs was made by Whitaker (1982), who focused on the emergence of a foreign accent only in association with structural damage to the central nervous system (CNS). Whitaker (1982) coined the term FAS and proposed the four diagnostic criteria: (i) awareness of the affected person, relatives and physicians that the newly acquired accent sounds foreign; (ii) the new accent is different to the native dialect; (iii) the accent change results from brain damage; and (iv) the affected person is monolingual without previous contact with the newly acquired foreign accent (Whitaker, 1982: pp. 196-198). Although these criteria were used until recently to classify FAS ((Ryalls & Miller, 2014)), they should be considered too restrictive (Verhoeven & Mariën, 2010: p. 600). To reconcile discrepancies, consider that other forms of altered accents had not been described when Whitaker proposed criteria for diagnosing FAS. Changes in regional accents early described by Marie (1907) were wrongly defined as FAS, whereas some real cases of FAS antedating Whitaker's diagnostic criteria had been classified as dysprosody or altered melody of language (Critchley, 1964; Monrad-Krohn, 1947; Whitty, 1964). During the following decades, several cases of AAPs not fully meeting Whitaker's criteria have been reported (*Keulen, 2017,*), thus encouraging Verhoeven and Mariën (Verhoeven & Mariën, 2010) to modify and expand Whitaker's criteria to describe new variants. Notably, the requirement of monolingualism (iv) was considered too restrictive and had to be abandoned: it is estimated that at least 50% of the world population can effectively communicate in more than one language ((Grosjean, 2019). Many countries have a strong tradition of second language learning from a very young age onwards, and the requirement for monolingualism was considered unrealistic. In the second instance, the requirement of the foreign accent to be rooted in organic damage to the central nervous system (iii) was relaxed to accommodate for psychogenic FAS, which often does not have any demonstrable CNS aetiology (but see Dadario et al., 2023).

The existing scientific evidence shows that AAPs are heterogeneous in most signatures, thus resisting applying a straightforward taxonomy for evaluating accent changes, their neural basis, and comorbidities. Therefore, classifying AAPs into subtypes requires substantial work, and proceeding with this approach is only sometimes feasible because cases that combine more than one variant exist. More research on AAPs should be done because, by their rare occurrence, these disorders may be qualified as “orphan” disorders, thus hampering the progress in diagnosis and treatment. However, unwanted changes in accent might not be as rare as previously assumed. Public awareness, knowledge and identification of AAPs are gradually increasing, and this is reflected by the increased number of anecdotal reports appearing on social networks, where affected speakers witness the impact of living with an altered accent

(<https://www.youtube.com/watch?v=JF56cDpB0r0>). That underscores the need for further research in this area.

3. CLINICAL VARIANTS OF ALTERED ACCENT PROFILES

From a taxonomical perspective, three types of AAPs have been described: neurogenic, psychiatric, and mixed (Keulen, 2017; Verhoeven & Mariën, 2010). These subtypes, each with unique characteristics and challenges, are important in neurology, psychiatry, speech pathology, and neurolinguistics and are briefly described below (*for a review, see* Keulen, 2017; Mariën et al., 2019).

3.1 Neurogenic

3.1.1 Acquired

The term acquired neurogenic AAPs is used here to distinguish them from neurogenic developmental AAPs (*see* Section 3.1.2). Reviewing the various aetiologies of neurogenic AAPs is beyond the scope of this article, but stroke and traumatic lesions prevail (*see* Keulen, 2017; Mariën et al., 2019). The diagnosis of acquired neurogenic AAPs usually implies a close temporal relationship between the emergence of an accent change and well-defined focal brain lesions or neurodegenerative diseases (e.g., primary progressive aphasia; Alzheimer’s disease) affecting accent production (Luzzi et al., 2008) or reception (Fletcher et al., 2013; Hailstone et al., 2012). However, clinical observation indicates that the affected individuals do not always perceive the temporal coincidence of a lesion and the onset of accent alterations (Moreno-Torres et al., 2013). The patient’s impaired recognition of accent changes usually delays diagnosing AAPs and accessing therapeutic interventions. Such initial lack of knowledge of accent changes may be due to concurrent deficits affecting accent speech perception, audiovisual feedback, phonological awareness, and error detection and monitoring (Adank et al., 2015; Gauvin et al., 2016; Nozari & Novick, 2017) resulting from damage to discrete components of the speech production network (SPN).

Despite the diverse aetiologies of AAPs (Mariën et al., 2019), it seems that, with few exceptions (Akhlaghi et al., 2011; Hill et al., 2023; Karanasios et al., 2011), AAPs often result from discrete compromise of one or more components of the left SPN, and rarely after the involvement of the right SPN (Berthier et al., 1991; Critchley, 1964; Dankovičová et al., 2001; González-Alvarez et al., 2003; Laures-Gore et al., 2006; Priftis et al., 2020). Cases of neurogenic AAPs associated to small stroke lesions, have already been considered “pure” in the sense that the patients display no additional speech and language deficits (Gurd et al., 1988; Takayama et al., 1993) or are associated with rapidly reversible impairments (e.g., Coughlan et al., 2004; *see* Section 3.1.1). Nevertheless, the typical association of small lesions with AAPs does not preclude that altered accents can also emerge after large hemispheric lesions, particularly during the recovery process from non-fluent aphasia when the accompanying reduced fluency, phrase length and functional communication come out (Berthier et al., 1991; Monrad-Krohn, 1947; van der Scheer et al., 2014; Whitty, 1964). Frequently, however, residual accent changes are often overlooked in patients with persistent nonfluent aphasia, perhaps because residual speech may be very fragmentary to identify accent changes (Critchley, 1964).

It is also expected that the emotional aftermath of accent change is overshadowed by the negative impact of aphasia on patients' personal lives. In this connection, the timelines for recovery from stroke aphasia and AAPs may differ, with a faster improvement of language deficits than the return of regional accent to its premorbid characteristics (Berthier et al., 2015). Such recovery dissociation has been linked to the presumed limited adaptive neuroplasticity for accent (Simmonds et al., 2011). Evaluating the presence of AAPs in non-recovered aphasic individuals is challenging because speech-language therapy can also be tailored to treat abnormal processing of distorted segmental and prosodic speech features responsible for accent changes.

Future studies should also attempt to disentangle whether the neural mechanisms underpinning AAPs differ in cases with large lesions compared to others with smaller lesions. More research on this issue is needed and it should examine the neural processes (e.g., compensatory reorganization, individual variability in accent processing) influencing differences in evolution and response to treatment (Fridriksson et al., 2005; Varley et al., 2006). Analysis of neuroimaging data in neurogenic AAPs suggests that the longitudinal evolution depends more on the location than the size of the lesion (Berthier et al., 1991, 2015; Higashiyama et al., 2021). Indeed, the involvement of the inferior and middle parts of the left precentral gyrus is consistently associated with the long-standing persistence of accent changes (Berthier et al., 1991, 2015; Graff-Radford et al., 1986; Higashiyama et al., 2021; Schiff et al., 1983). However, it is relevant that large lesions are not invariably associated with poor outcomes (e.g., Whitty, 1964), perhaps because crucial components of the SPN (e.g., left precentral gyrus) already associated with longlasting changes in accent are not permanently damaged.

3.1.2. Developmental

Cases of developmental AAPs are starting to be recognised. The usual form of presentation is that parents of some toddlers with developmental speech and language disorders note that their children are unable to acquire a native-like accent (Berthier et al., 2016; Keulen, Marién, et al., 2016; Mariën et al., 2009; Rambathla & Rao, 2013; Vares González, 2015). In some cases, the awareness of speaking with a non-native accent is delayed until adolescence or early adulthood (Berthier et al., 2016; Mariën et al., 2009). This form of AAP has also been classified as neurogenic, thus implying that it results from functional or structural CNS involvement. The few reported individuals with developmental AAPs had neuroimaging evidence of developmental brain anomalies involving components of the SPN, which interfere with adaptive neural plasticity (Berthier et al., 2020; Sokolowski & Levine, 2023). Such impaired restorative neural plasticity could explain the long-term persistence of accent changes into adulthood and the lack of response to speech-language therapy (Keulen, Marién, et al., 2016; Mariën et al., 2009). Similarities between developmental and acquired AAPs in speech features have been identified (Berthier et al., 2016; Keulen, Marién, et al., 2016) as well as in functional brain anomalies (cortico-cerebellar network dysfunction - Keulen et al., 2017).

Psychological consequences of developmental AAPs are not always present (Keulen, Marién, et al., 2016; Mariën et al., 2009), yet their long-standing evolution plus their embedding in other disabling speech and language developmental disorders may induce

emotional disorders (anxiety, depression and social withdrawal) (Berthier et al., 2016). Therefore, further work is needed to investigate whether this group of psychiatric disorders represents a maladaptive reaction to the prolonged consequences of living with AAPs (self-acceptance, isolation, academic failure, unemployment) or whether the wellknown link between developmental speech disorders and dysregulation of emotional adjustment plays a role (Dietrich et al., 2012; Gunn et al., 2014; Karukivi et al., 2012; Snowling et al., 2006). Since AAPs may be much more common in populations with developmental speech-language disorders than in neurotypical individuals (Keulen, Marién, et al., 2016), comprehensive multimodal studies in individuals with autism spectrum disorder, specific language impairment, and speech disorders (apraxia of speech, stuttering, cluttering) could be very informative.

3.2 Psychiatric, psychogenic, functional and related designations

Theoretical explanations of AAPs have understandably been construed in the framework of impaired programming or execution of segmental (phonetics, phonology) and suprasegmental (prosody) features and cognitive function (control of motor speech processing) (Moen, 2000; Varley et al., 2006). This testing approach, however, does not address the influence of a general alteration of well-being in the pathogenesis and functional mechanisms underlying AAPs. Therefore, more research on AAPs' psychological precursors or consequences is strongly required. In the last few years, there have been an increasing number of cases showing isolated or recurrent episodes of AAPs in patients with psychiatric disorders (psychosis, schizophrenia, bipolar disorder, and conversion disorders) (Asogwa et al., 2016; Jones et al., 2011; Lewis et al., 2012; Poulin et al., 2007; Reeves et al., 2007; Reeves & Norton, 2001; Verhoeven et al., 2005). Essential information comes from individuals with schizophrenia and bipolar disorder who markedly change their accents coinciding with psychotic exacerbations activated by the withdrawal of neuroleptics (Reeves et al., 2007; Reeves & Norton, 2001). The nativelike accent often returns (Asogwa et al., 2016), in parallel with the attenuation of psychotic symptoms after re-instating drug treatment (Reeves et al., 2007; Reeves & Norton, 2001). In this sense, unfavourable outcomes may occasionally be informative concerning the origin of the underlying processes implicated. The above scenario raises the possibility that speech dysfunction is not always the primary source of AAPs. Instead, such compelling evidence suggests a permissive role of decompensated psychotic symptoms (delusions, hallucinations, grandiose ideas) in the functional disinhibition of SPN's components underpinning AAPs.

Partially based on their psychological precursors or consequences, these presentations of AAPs are generally designated as psychogenic (Keulen, 2017; Lee et al., 2016; Van Borsel et al., 2005) or functional (Demichelis et al., 2022; Lee et al., 2016; McWhirter, Miller, et al., 2019; Romö et al., 2021). These terms are used interchangeably to refer to those cases not showing structural CNS damage (Demichelis et al., 2022). Nevertheless, these labels have also been used in cases with structural CNS involvement and clinical features suggestive of a functional disorder (McWhirter, Miller et al., 2019)³. Lastly, the label idiopathic has recently been used to refer to AAPs in patients showing no gross structural CNS damage (Demichelis

³ *Functional neurological disorder* is defined as an impairment in brain networks that leads to various symptoms, including speech output difficulties (Jungilligens et al., 2022).

et al., 2022) but with atypical connectivity patterns in language and sensorimotor functional networks (Dadario et al., 2023).

Since there are still no well-defined boundaries between neurogenic, psychogenic, functional, and idiopathic AAPs, further work is required (Berthier & Moreno-Torres, 2019; Dankovičová et al., 2001; McWhirter, Miller, et al., 2019; McWhirter, Stone, et al., 2019). What seems apparent is the inappropriateness of introducing additional diagnostic labels until more precise distinctions between them are established. A simple way to bring order into the current lack of consensus on the categorical classification of AAPs is to consider that they often appear in conjunction (Section 3.3). Following this line of thought, recent research initiatives seek to shed some light on establishing benchmarks for differential diagnosis (Barreto & Ortiz, 2020; Cuoco et al., 2024; McWhirter, Miller, et al., 2019; Romö et al., 2021). For instance, Romö et al. (2021) evaluated distinctive segmental features in subjects with neurogenic ($n = 12$) and functional ($n = 5$) FAS-AAPs. Findings revealed that schwa⁴ insertion prevailed in participants with the neurogenic type, whereas a greater degree of variability in /r/ realisations was more frequent in the functional subtype. Although these results are very informative for the studied groups, participants with psychiatric diagnoses were not tested (Romö et al., 2021). Examining psychiatric comorbidity in the most extensive case series of individuals self-reporting FAS so far ($n = 49$), the same group of researchers (McWhirter, Miller et al., 2019) found similar frequencies of anxiety and depression in participants with probably-possibly functional ($n = 39$) and probably structural (neurogenic) ($n = 10$) (*see* Barreto & Ortiz, 2020; Berthier & Moreno-Torres, 2019; McWhirter, Miller, et al., 2019).

When a role of psychogenic or functional factors is suspected, scrutiny of personality traits (Berthier et al., 2016), pre-existing medical illnesses (Broderick et al., 2023; Cotelli et al., 2020; Olguner Eker et al., 2023) and surgical interventions (Corbett et al., 2022; Polak et al., 2013; Schroeder et al., 2016) is required because they may act as potential contributors to AAPs. In this case, collaborative interdisciplinary research and clinical care are recommended (Mavroudis et al., 2024). The fact that "functional neurological disorders", including AAPs, are now interpreted as multi-network impairments indicates that modern neuroimaging techniques and other biomarkers should be included in the evaluation toolkit to enhance diagnostic precision (Jungilligens et al., 2022; Mavroudis et al., 2024).

3.3. Mixed

From the data mentioned above, it seems that AAPs may adopt a hybrid identity (Berthier & Moreno-Torres, 2019), with affected individuals presenting with an overlap of accent change and reduced psycho-social adjustment, that justifies the designation of "mixed" for such cases (Verhoeven & Mariën, 2010). Existing combinations classified as mixed AAPs are diverse and include: (i) the circumstance when speakers with a neurogenic variant voluntarily modulate the newly acquired accent to comply with their idiosyncratic desires (Fontana et al., 2024; Mariën et al., 2019); (ii) speakers with a neurogenic variant who develop an adjustment disorder with anxiety, depression or both (McWhirter, Miller, et al., 2019); and (iii) speakers with previous

⁴ The term schwa refers to the weak vowel sound in some syllables that is not emphasized

psychiatric disorders who develop AAPs in conjunction with medical (Olguner Eker et al., 2023) or neurological illnesses (Lázaro-Perlado et al., 2013; McWhirter, Miller, et al., 2019), deep brain stimulation interventions to alleviate treatment-refractory psychiatric disorders (Polak et al., 2013), or abrupt pharmacological treatment withdrawal (Reeves et al., 2007; Reeves & Norton, 2001). These and other possible situations would imply that AAPs can trigger emotional reactions to deal with the undesired accent change, or conversely, being vulnerable to psychological problems or suffering full-blown psychiatric disorders (conversion disorder, schizophrenia, bipolar disorder) can put the individual at a higher risk for developing AAPs.

It is intuitively apparent that cases of AAPs presenting with mixed symptomatology can be shared amongst vulnerable populations (Berthier et al., 2016; Romö et al., 2021). Therefore, it is crucial to ascertain whether people of diverse ethnic backgrounds, migrants, children with disabilities, individuals with a history of psychiatric disorders, and sexual and gender minorities are at high risk of developing AAPs. Although it seems that most forms of AAPs are associated with decreased general psychological well-being, it remains to be explored whether different presentations of AAPs show specific behavioural correlates that helped their classification and selection of personalised treatments. In support, preliminary evidence suggests that post-stroke neurogenic FAS can improve by combining audiovisual feedback training and expression modelling of emotional prosody with the cholinergic modulator donepezil (Berthier et al., in preparation; Moreno-Torres et al., 2013). Moreover, neuroleptic drug treatment reverts FAS in decompensated schizophrenia and bipolar disorder (Reeves & Norton, 2001), and cognitive-behavioural therapy would help in AAPs related to conversion disorders (Birdsey & Millar, 2020). Altogether, this preliminary information instantiates Gurd and Coleman's early proposal (2006: p. 424) advocating personalised approaches for assessing and treating AAPs.

In summary, we have reviewed the evidence that AAPs can have similar symptoms regardless of the aetiology, suggesting that affected persons share a functional or structural biological basis (Turner & Husain, 2022). Hence, this argument reinforces the utility of applying a transdiagnostic approach to assessment.

4. The Neurobiology of Accent

Over the past two decades, there has been a growing interest in the neurobiological analysis of accents. This field holds significant promise for understanding the brain's role in accent processing. This research has been instrumental in unravelling the intricate anatomy and properties of the SPN (Basilakos et al., 2018; Castellucci, Guenther, et al., 2022; Möttönen et al., 2018; Simmonds et al., 2011; Simonyan et al., 2016; Valeriani & Simonyan, 2021).

The behavioural and neural underpinnings of accent processing are beginning to be identified (Adank et al., 2015; Moreno-Torres et al., 2016), and a further conceptualization of networks subserving speech perception, planning, and production (Castellucci, Guenther, et al., 2022; Castellucci, Kovach, et al., 2022) has been initiated. Neuroscientific breakthroughs have come from lesion studies (Berthier et al., 2015; Higashiyama et al., 2021; Katz et al., 2012; Moreno-Torres et al., 2013; Tomasino et al., 2013), brain imaging in neurotypical subjects (Simonyan et al., 2016; Valeriani & Simonyan, 2021), biologically-plausible

neurocomputational models (Tourville & Guenther, 2011), neurophysiology (Cuadros et al., 2023) and from analogies of vocal learning in human speech and bird song (Simmonds, 2015; Zhang et al., 2023).

Recent neuroimaging data start to show that structural lesions of the SPN components induce neurogenic AAPs (Higashiyama et al., 2021) involving some hubs (primary motor cortex, insula) which may be essential for the occurrence of AAPs and, perhaps, its long-lasting course (Berthier et al., 1991; Higashiyama et al., 2021), whereas involvement of other SPN components (e.g., supplementary motor area) contribute to AAPs but may not be necessary or sufficient for their development (Berthier et al., 2015; Higashiyama et al., 2021). Detrimental or beneficial changes in accent may also be secondary to disconnection of bidirectional cortical-cerebellar networks of the SPN caused by local structural damage of the left frontoparietal cortex and remote effects in the activity of other components (diaschisis) (Bostan & Strick, 2018; Cohen et al., 2009; Keulen et al., 2017; Priftis et al., 2020; Silveri, 2021). Paradoxically, recovery of cerebellar perfusion (Mariën et al., 2006; Mariën & Verhoeven, 2007) or lesions in the cerebellum (Cohen et al., 2009), a component of the SPN distant to the original lesions, may resolve neurogenic FAS.

Multimodal neuroimaging in AAPs subsumed under the headings psychiatric, psychogenic, functional, or idiopathic usually do not show structural lesions (Keulen, Verhoeven, et al., 2016). However, one case series study of idiopathic AAPs revealed functional connectivity alterations within language and sensorimotor networks in left frontal regions and between subcortical structures (Dadario et al., 2023). Although more neuroimaging studies are needed in these AAPs, it seems that changes in accent result from structural and functional damage to the CNS, and the responsible lesion-dysfunction usually affects the SPN (Kurowski et al., 1996). It is tempting to speculate that discrete damage to one or more components of the SPN may be associated with different variants of accent change. For example, this effect leads to the disappearance of one or more segmental and prosodic characteristics of the native accent (e.g., Farsi), which are replaced and mixed with speech features of another regional accent (e.g., Yazdi) (Akhlaghi et al., 2011). At present, there is little information on whether changes in accent after brain damage or dysfunction result from removal of inhibitory factors (Critchley, 1964; Roth et al., 1997; Seliger et al., 1992) coupled or not with a compensatory effect by increasing the demand in other subcomponents of the SPN when crucial components implicated in the control of planning or execution of accent are not available (Cohen et al., 2009; Fridriksson et al., 2005; Mariën & Verhoeven, 2007). Paying attention to these potential mechanisms is necessary because it would be possible to modulate the activity of structures participating in the recovery of AAPs with drugs (Moreno-Torres et al., 2013) and non-invasive brain stimulation (NIBS) (Cohen et al., 2009).

5. The influence of altered neurotransmitter activity

The activity of the SPN and its connected emotional and cognitive networks is modulated by neurochemistry (Fuertinger et al., 2018; Turk et al., 2021). Advances in this field would provide a better understanding of the accented speech perception and production mechanism in healthy subjects. They can likewise inform future neurobiological modulation (e.g., pharmacotherapy,

NIBS) in cases of AAPs with evidence of neurotransmitter dysfunction (e.g., dopamine, acetylcholine). Analysis of these issues could be informative for individuals with psychiatric disorders (e.g., schizophrenia, bipolar disorder, and somatoform and conversion disorders) who develop AAPs (changes in regional accent) *de novo* (Poulin et al., 2007; Verhoeven et al., 2005) or after discontinuing neuroleptic drugs (Reeves et al., 2007; Reeves & Norton, 2001; Strous et al., 2003). In the same vein, neurotransmitter dysfunction needs to be investigated in people in whom neurogenic AAPs induce adjustment disorders with psychiatric manifestations and reduced quality of life. Hence, attention is also required to examine the role of neurotransmitter activity in accent changes, as neurochemistry is implicated in the modulation of speech and language (Dávila et al., 2023; de Boer et al., 2020; Jung et al., 2017; Li et al., 2020) in bilinguals and polyglots (Heinemann & Assion, 1996; Saito, 2019; Sajid et al., 2020). Available evidence from AAPs (change in regional accent) (Asghar & Whitty, 2020) as well as FAS (Reeves & Norton, 2001) and regression to mother tongue in polyglots (Heinemann & Assion, 1996; Saito, 2019) during psychotic and bipolar disorders exacerbations due to neuroleptic withdrawal are usually followed by rapid resolution of foreign speech and language switching when treatment with neuroleptics (dopamine antagonists) is reinitiated (Reeves & Norton, 2001).

6. Predictions and outstanding questions

6.1 Predictions based on present evidence

Predictions in neuroscience can guide research, enhancing confirmation. Therefore, several predictions can be formulated based on recent advances in studying accents in neurotypical individuals and others with AAPs.

AAPs are more frequent than previously assumed. Underdiagnosis of an AAP is expected unless the person is specifically asked about it. Hence, enquiring about people with acquired and developmental speech and language deficits, psychiatric disorders, and vulnerable populations about AAPs may enable the identification of overlooked cases, particularly those with subtle symptoms or AAPs mixed with other disorders (aphasia, apraxia of speech, dysarthria).

AAPs are heterogeneous in most of their presentations and are still considered rare. Hence, concerted action in multisite research centres can unveil hidden cases, enabling the recruitment of more homogeneous groups for research.

Future cases of AAPs should be evaluated using a transdiagnostic approach. That involves collaborating with professionals and researchers who can apply various ancillary methodologies. Such an approach can provide a more comprehensive understanding of AAPs.

Mixed variants of AAPs are probably the most frequent forms of presentation. That justifies the use of comprehensive multimodal testing and developing treatments based on personalised medicine.

Cross-sectional analysis of AAPs is exciting but less valuable than longitudinal studies aimed at understanding their natural evolution and their response to treatment.

Most case studies of AAPs have not provided information on treatment. It is essential to investigate personalised treatment interventions. While speech-language therapy may be the preferred treatment, the potential benefits of audiovisual feedback training and strategies tailored to treat speech and voice disorders need to be explored. Furthermore, the potential efficacy of behavioural therapies in psychiatric cases requires further study. Available evidence suggests limited or no efficacy of speech-language therapies in developmental AAPs. Therefore, the adjunctive contribution of neurobiological interventions (such as drugs and NIBS) should be explored.

6.2 Unsettled questions

Recent research has provided important breakthroughs and raised several questions that should be addressed. These findings can significantly impact our understanding of accent alteration and speech disorders. Only some of the questions are outlined below. • Does increased general awareness of AAPs by media exposure artificially inflate psychogenic and functional cases to request consultation in primary and specialist care?

Is undesired accent change a consistent, stable phenomenon? Or may it be fluctuating (present only for some words, phrases, or circumstances) or focused on a single output modality (spontaneous speech but not reading)?

What mechanisms are underlying the alternation between two or more different accent changes (foreign and regional accents)?

Where are the boundaries between the natural evolution of a native accent and the acquisition of non-native accents in healthy people and AAPs?

Is there little room for adaptive neural plasticity in long-standing cases of AAPs? Which constraints are in place for the limited neural plasticity of neurogenic AAPs? When lesioned, are distinct components of the SPN more crucially tied to AAPs' persistence than others? Can an altered traffic flow between inhibitory and excitatory subcomponents of the SPN hamper recovery?

Are specific features of AAPs resulting from increased recruitment of interconnected neural systems to compensate for damage-dysfunction to the SPN? That being the case, which are the suitable therapeutic interventions to exploit the vicarious activity of these regions?

How do we treat the aftermath of living with an AAP? Improving speech alterations, providing psychosocial support to cope with the undesired accent change or both?

7. Conclusion

This article has attempted to discuss FAS from a more general perspective than before, and it was suggested that the term be replaced with a more general designation, AAPs, to accommodate the different presentations of undesired accent changes in patients with neurological or functional disorders. The implications of this for a more correct diagnosis and

treatment were discussed. Different types of AAPs were distinguished, and the relationship between the native accent and the AAPs was also described. Moreover, the potential role of AAPs' neurobiology and neurotransmitter dysfunction was reviewed. Finally, several predictions and hypotheses were formulated which could shape the future of AAPs research.

Declaration of Interest

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CRediT authorship contribution statement

Marcelo L. Berthier: Investigation, Conceptualisation, Writing Original Draft - Review and Editing. Ignacio Moreno-Torres: Conceptualisation, Writing and Editing. Jo Verhoeven: Conceptualisation, Guadalupe Dávila: Conceptualisation, Writing and Editing.

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