

## Disentangling a Functional Speech Disorder in the Context of another Neurological Disease: A Case Report

### Perturbação Funcional da Fala no Contexto de outra Doença Neurológica: Caso Clínico

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#### ABSTRACT

This clinical case describes the communication profile of a functional speech disorder. A 48-year-old woman was admitted to the emergency service due to sudden changes in speech, generalized dystonia and gait ataxia. Magnetic resonance imaging showed multiple supra and infratentorial inflammatory lesions involving the posterior occipito-temporal lobes bilaterally, frontal convexity bilaterally, insula and paramedian frontal cortex on the left, diencephalon, rhombencephalon. On the third day, a speech pathology evaluation was conducted, revealing inconsistencies in speech: repetitions of initial sounds, without characteristics of an acquired stutter, a clenched articulation, not justified by an orofacial motor disorder, an inconsistent pattern in voice and prosody. Three days later, another assessment was carried out, showing a significant improvement in speech intelligibility. There was a marked variability in speech features defects found over the days, as well as in their severity. These behaviors appear simultaneously with a speech pattern that is not justified by any of the neurological lesions found.

**Keywords:** Conversion Disorder; Psychophysiologic Disorders; Speech; Speech Disorders

#### RESUMO

Este caso descreve o perfil comunicativo de uma perturbação funcional da fala. Uma mulher de 48 anos foi admitida no serviço de urgência com alterações súbitas da fala, distonia generalizada e ataxia da marcha. A ressonância magnética mostrou múltiplas lesões supra e infratentoriais inflamatórias envolvendo lobos occipitotemporais bilateralmente, ínsula e córtex frontal paramediano esquerdos, diencefalo e rombencéfalo. No terceiro dia, foi avaliada em Terapia da Fala, revelando inconsistências: repetições de sons iniciais, sem características de gaguez adquirida, articulação cerrada, sem defeitos de motricidade orofacial, flutuações na voz e prosódia. Passados três dias, foi efetuada nova avaliação, verificando-se uma melhoria significativa na inteligibilidade do discurso. Existe uma flutuação dos defeitos de fala encontrados ao longo dos dias, bem como da sua gravidade. Estes comportamentos surgem em simultâneo com um padrão de fala que não é justificado por nenhuma das lesões neurológicas.

**Palavras-chave:** Fala; Perturbação Conversiva; Perturbações da Fala; Perturbações Psicofisiológicas

#### INTRODUCTION

Functional speech disorders (FSD) are a subtype of functional neurological disorders. They can be characterized by speech manifestations that resemble those observed in other neurological disorders but cannot be fully explained by the underlying neurological or mental condition.<sup>1</sup> Some studies of FSDs suggested that the main manifestations include dysfluencies (pauses, hesitations and repetitions) articulatory errors, dysphonia (changes in vocal quality), and atypical prosody (changes in rhythm and melody).<sup>2</sup> The characteristics associated with motor speech disorders and FSD often overlap and can co-occur.<sup>2,3</sup> The literature regarding the identification and clinical evaluation of these disorders remains limited. Detailed case reporting is essential, as it provides valuable insights that enhance diagnostic accuracy and improve clinical decision-making.

#### CASE REPORT

A 48-year-old right-handed female patient, native European Portuguese speaker, with 12 years of formal education, was admitted to the emergency department. The patient was oriented, exhibiting an apparent stutter and marked changes in prosody. Moreover, she disclosed mild generalized dystonia as well as a postural and intentional tremor, a gait instability characterized by retropulsion (postural instability) and a widened base.

Brain magnetic resonance imaging (MRI) identified multiple supra- and infratentorial lesions in T2 FLAIR in cortico-pial topographies, predominantly in the posterior occipito-temporal lobes bilaterally and the frontal convexity bilaterally, insula and paramedian frontal cortex on the left, diencephalon, rhombencephalon, especially the thalamus, hippocampus, and internal capsule on the left (Fig. 1). A cerebrospinal fluid (CSF) analysis revealed 48 cel/mm<sup>3</sup> (< 5 cel/mm<sup>3</sup>) with normal glucose and protein levels. Multiplex polymerase chain reaction (PCR) for infectious agents was negative. Serum serologies, as well as anti-MOG and anti-AQP4 antibodies, were also negative.

On the first day, a speech and language assessment was conducted, with an acquired neurogenic stuttering considered

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as being the most probable syndromic diagnosis. Various tasks were performed, including automatic speech, spontaneous and descriptive discourse, reading a text aloud, singing, naming, auditory comprehension, word and phrase repetition.

In this first observation, the patient presented with disfluency (repetitions of syllables and initial sounds) in spontaneous speech, but not in narrative speech. Also, when asked to repeat words and phrases or even sing, the patient showed no dysfluency, a pattern that is not consistent with what is typically found in acquired stuttering.

Her speech was initially characterized by atypical features in articulation, phonation and prosody. Specifically, a clenched articulation in speech production was evident, marked by a low range of movement, a loss of articulatory precision and hypernasality, but again with an inconsistent behavioral pattern throughout the tasks. Phonation was characterized by fluctuations in pitch and intensity during a prolonged vowel production and prosody assessment revealed significant impairments: a monotonous speech and an inability to replicate various speech patterns or intonations.

The first language assessment revealed no difficulties in discourse, word finding, repetition, auditory comprehension, reading comprehension or writing abilities. All communicative behaviors were inconsistent, varying across tasks.

Given the clinical picture, the hypothesis of a FSD was considered by the multidisciplinary team (speech therapists, neurologists and psychiatrists). No correlations were found between the speech alterations and the neurological or imaging abnormalities. In addition, despite the higher prevalence of psychiatric comorbidities among patients with functional neurological disorders, no psychopathological findings were identified.

On the sixth day, the patient underwent a formal evaluation of language, speech and fluency, with an overall improvement in the clinical presentation. Regarding fluency, the disfluencies initially observed were no longer present upon reassessment. In terms of language, the patient produced grammatical errors, such as omission of constituents of the sentence, and also demonstrated difficulty identifying prosodic features and melodic contours associated with various phrases in a subtest of the Montreal Assessment Battery. Speech exhibited minimal impairments, such as fluctuations in articulatory performance, hypernasality, and prosody. As observed previously, there was no impairment in orofacial motor skills that could justify the noted defects. The patient used, predominantly, a low-intensity voice, but was able to increase loudness when asked. Occasionally, abrupt and exaggerated movements were observed during the assessment in performing rapid and alternating oral motor movement sequences.

Between the initial and second assessments, no treatment that could impact the clinical picture was administered. Although, given the possibility of inflammatory lesions on MRI and the presence of coordination and gait impairments, the patient was administered methylprednisolone 1 g IV for five days, resulting in a complete clinical, CSF and imaging resolution.

## DISCUSSION

The patient exhibited a complete resolution of the initial dysfluencies and an improvement in speech intelligibility without any medical treatment. This included the complete resolution of the initial dysfluencies and an improvement in speech intelligibility. Although this might be considered a case of spontaneous recovery, the observed characteristics cannot be explained solely by a speech-related condition, as there were inconsistent error patterns, including disfluencies that improved with melody or repetition, features that are not characteristic of acquired stuttering. Additionally, there was an absence of orofacial abnormalities that would explain the observed speech motor features, along with variable articulatory precision throughout tasks. These findings are not consistent with a typical motor speech disorder. Furthermore, the patient displayed specific language difficulties in identifying prosodic traits, in the absence of other linguistic impairments. This isolated language impairment is difficult to classify under any type of language or speech disorder.

The findings suggest the presence of a FSD, which is characterized by: a speech pattern that does not correspond to any type of known motor speech disorder and cannot be explained by a neurological lesion; a discrepancy between the orofacial motor assessment and the speech patterns; a variable speech rhythm; a reversible speech pattern within a brief time frame; presence of isolated grammatical errors without other linguistic impairments; an inconsistent hypernasality and a variation in prosody and/or phonation.<sup>3</sup> The clinical findings presented in this case are in line with the existing case descriptions in the literature, emphasizing an inconsistent speech pattern that is not attributable to any other impairment. The differences in the pattern features and severity of impairments are context-dependent, demonstrating variability across assessments.

The diagnosis of a FSD can be challenging, particularly when other neurological symptoms and structural brain lesions are present. Therefore, this case highlights the importance of multidisciplinary evaluations to establish an accurate diagnosis, as this may influence subsequent therapeutic decisions. This case contributes to the literature with information on the identification and assessment of FSDs.

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## AUTHOR CONTRIBUTIONS

BS: Conceptualization, data collection and analysis, writing and critical review of the manuscript.

FS, JF: Conceptualization; data collection, critical review of the manuscript.

All authors approved the final version to be published.

## PROTECTION OF HUMANS AND ANIMALS

The authors declare that the procedures were followed according to the regulations established by the Clinical Research and Ethics Committee and to the Helsinki Declaration of the World Medical Association updated in October 2024.

## DATA CONFIDENTIALITY

The authors declare having followed the protocols in use at their working center regarding patients' data publication.

## PATIENT CONSENT

Obtained.

## COMPETING INTERESTS

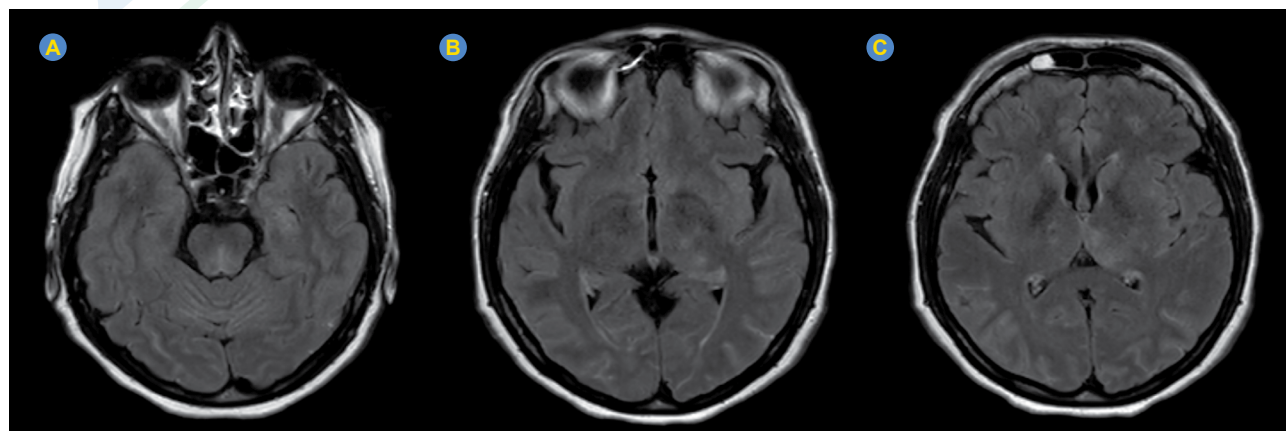
The authors have declared that no competing interests exist.

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**Figure 1** – Brain MRI (T2 FLAIR axial) showing multiple supra- and infratentorial lesions in posterior occipito-temporal lobes bilaterally and insula and paramedian frontal cortex on the left, along with hyperintensities on the left hippocampus and internal capsule. There are also hyperintensities in diencephalon and rhombencephalon, especially in the left thalamus.