Available online at www.sciencedirect.com
SCIENCE DIRECT®
Brain and Language 85 (2003) 185–189
Brain and Language
www.elsevier.com/locate/b&l

Thalamic stuttering: A distinct clinical entity?

John Van Borsel,* Sandie Van Der Made, and Patrick Santens

Ghent University Hospital, UZ Gent, 2PI, Spraakrevalidatie, De Pintelaan 185, Gent B-9000, Germany

Accepted 15 January 2003

Abstract

A 38-year-old right-handed male with no history of speech or language problems presented with neurogenic stuttering following an ischaemic lesion of the left thalamus. He stuttered severely in propositional speech (conversation, monologue, confrontation naming, and word retrieval) but only slightly in non-propositional speech (automatic speech, sound, word and sentence repetition, and reading aloud). It is suggested that thalamic stuttering may constitute a distinct clinical entity.

Keywords: Neurogenic stuttering; Acquired stuttering; Thalamic stuttering; Thalamus

1. Introduction

Although stuttering most often has its onset in childhood, usually somewhere between the ages of two and five (Johnson & Associates, 1959), dysfluent speech may appear also for the first time later in life, beyond the typical childhood period. A dysfluency that originates after the typical childhood period has been called acquired stuttering, or sometimes also late-onset stuttering or adult onset stuttering. Acquired stuttering may have various causes. It can be of psychogenic origin, drug-induced or malingered. Most frequently, however, acquired stuttering is of neurogenic origin, i.e., associated with damage to the central nervous system. In recent years, several cases of neurogenic stuttering have been reported (see Ringo & Dietrich, 1995 and Van Borsel, 1997, for a review).

At first sight, it would seem that neurogenic stuttering is not linked to any specific lesion site. The damage that lies at the origin of the dysfluencies in neurogenic stuttering may be bilateral or unilateral, focal or diffuse, cortical or subcortical, situated in the right hemisphere or the left hemisphere. Within one hemisphere the lesion may be localized in the frontal lobe, the temporal lobe, or the parietal lobe. Only in conjunction with damage to the occipital lobe neurogenic stuttering has not been observed. This should not mean, however, that neurogenic stuttering has no localizing significance at all. There is still the possibility that within neurogenic stuttering the symptoms vary according to the lesion site and that different types of neurogenic stuttering can be distinguished accordingly, just like is the case in other neurogenic disorders of speech and language, such as dysarthria and aphasia. A few proposals in that direction with regard to neurogenic stuttering have already been formulated (Ackermann, Hertrich, Ziegler, Bitzer, & Bien, 1996; Koller, 1983).

The present paper reports a case of neurogenic stuttering due to a thalamic stroke. An analysis of the dysfluency pattern prompted the suggestion that thalamic stuttering may be a distinct clinical entity.

2. Case history

The subject of this study was a right-handed male businessman who had no history of speech or language problems. At the age of 38 he suffered a stroke. An initial CT-scan on admission showed no abnormalities. Magnetic resonance imaging (see Fig. 1) 2 weeks later revealed an ischemic lesion in the left ventrolateral thalamus.

The initial neurological testing demonstrated aphasic symptomatology and a very mild right hemiparesis with a decreased sensation in the right hemisoma. Six months later these symptoms were still present, although less

* Corresponding author. Fax: +32-9-240-4993.
E-mail address: john.vanborsel@rug.ac.be (J. Van Borsel).

0093-934X/03/$ - see front matter © 2003 Elsevier Science (USA). All rights reserved.
doi:10.1016/S0093-934X(03)00061-0

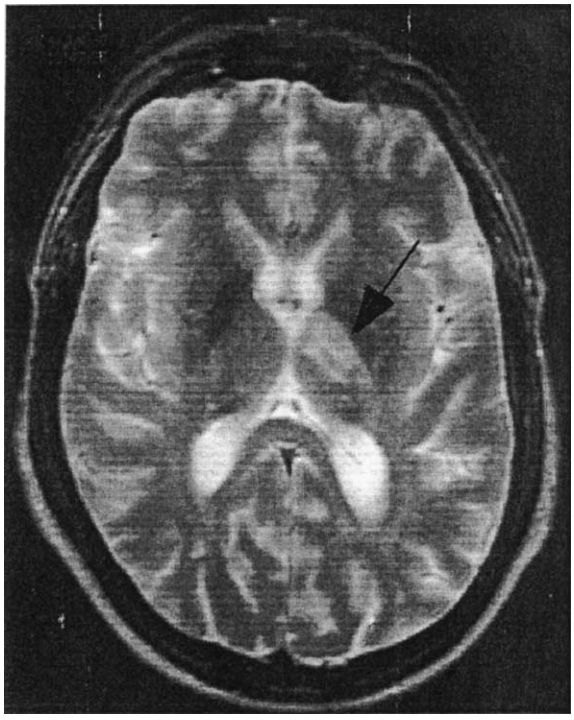

Fig. 1. MRI showing an infarct of the left lateral thalamus.

prominent. At that time a marked cognitive dysfunction was demonstrated by neuropsychological testing. Attention and episodic memory (Digit span from the Wechsler Memory Scale; Rey Auditory Verbal Memory Test) were clearly deficient. Mild executive dysfunction (Verbal Fluency; Stroop Color and Word Test; Wisconsin Card Sorting Test; Trail Making Test) and oral apraxia was found.

A neurolinguistic examination revealed aphasic symptoms consistent with a thalamic aphasia. The patient showed good auditory comprehension. He obtained a score of 55/61 on the Dutch version of the Token test (Van Dongen, Van Harskamp, & Luteijn, 1976) and a score of 45/45 on the sentence comprehension test of the S.A.N., a Dutch aphasia battery by Deelman, Koning-Haanstra, Liebrand, and Van De Burg (1981). Word and sentence repetition, as assessed by a subtest from the Aachener Aphasie Test (A.A.T.) (Graetz, De Bleser, & Willmes, 1992) appeared to be preserved too. When presented the subtest "benoemen" (naming of pictures of objects) from the A.A.T., however, the patient demonstrated manifest word finding problems and on the subtest "diernamen noemen" (naming animals) from the S.A.N., a word retrieval test, he scored on percentile 0. Moreover, there were frequently pauses during conversation.

In addition to language problems the patient also demonstrated fluency failures, which were further analysed from videotaped speech samples (Sony Video Hi 8 camera). Modalities included in the speech samples were automatic speech (days of the week, months of the year, counting from 1 to 20), repetition of sounds, words, and sentences (subtest from the A.A.T.), reading aloud (the IPA text "The north wind and the sun. . .," International Phonetic Association, 1974), conversation (on the patient's job and leisure activities), monologue (describing action and situation pictures from the AAT), confrontation naming (the subtest "benoemen" from the AAT) and word retrieval (subtest "diernamen noemen" from the S.A.N.). A possible adaptation effect was tested by having the patient read aloud the IPA text three times in a row. Finally, for three of the above speech types namely automatic speech, repeating words, and reading aloud, the patient was also tested under time pressure by having him perform the tasks again, at the end of the session, as fast as he could. The recorded speech samples were subjected to a consensus transcription (ordinary spelling) and a consensus analysis of the occurrence and nature of dysfluencies by two of us (JVB and SVDM).

Overall, across all speech samples gathered, a dysfluency index (number of stuttered syllables relative to the total number of syllables produced) of 12% was found. Interjections, word repetitions, and part-word repetitions were the most frequent type of dysfluency accounting for 35.5, 24.5, and 24.5%, respectively, of all dysfluencies. Less frequently the patient showed phrase repetitions (6.5%), revisions (5.5%), incomplete phrases (2%), sentence repetitions (1.5%), and prolongations (1%) (see Fig. 2).

A further analysis of the word level repetitions showed that whole-word repetitions consisted manly of a single repetition of a word (e.g., 36 out of 51 instances during conversation) and that part-word repetitions consisted most of the time of a single repetition of part of the word (e.g., 33 out of 51 instances during conversation). The most striking characteristic of the patient's dysfluency, however, was a discrepancy in

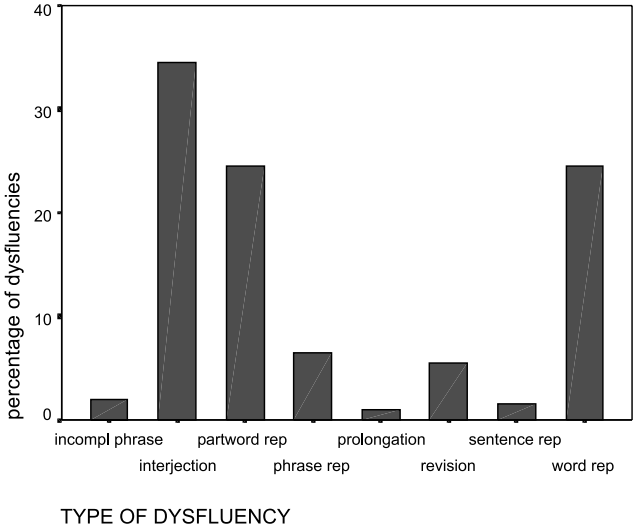

Fig. 2. Relative frequency of different types of dysfluency (across various speech tasks).