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Brain and Cognition 53 (2003) 185–189
Brain and Cognition
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Acquired stuttering following right frontal and bilateral pontine lesion: A case study

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Accepted 7 May 2003

Abstract

We report neurological information and experimental data regarding acquired neurogenic stuttering in a 57-year-old male following ischemic lesion to the orbital surface of the right frontal lobe and the pons. The experimental data consist of stuttering frequency measures under various conditions that are well known to enhance fluency in most individuals with developmental stuttering. Specifically, we report data for adaptation, unison reading, delayed auditory feedback (DAF), and frequency altered feedback (FAF). This work is the first published report of such a comprehensive examination of a variety of fluency-enhancing conditions in acquired stuttering. The patient read six 200-word texts under different conditions: Six solo readings (Text 1), five unison readings followed by five solo readings (Text 2), five readings with non-altered auditory feedback (Text 3), five readings with 50 ms delayed auditory feedback (Text 4), five readings with increased FAF (Text 5), and five readings with decreased FAF (Text 6). Results indicate that, unlike the typical situation for developmental stuttering, this individual with acquired neurogenic stuttering did not show increased fluency during an adaptation paradigm or under unison, DAF, and FAF conditions. We discuss possible implications of these findings and emphasize the need for further research on acquired neurogenic stuttering.

1. Introduction

Recently, sophisticated experimental methods have been applied (e.g., Salmelin, Schnitzler, Schmitz, & Freund, 2000) to examine some hypotheses regarding the neural bases of developmental stuttering that had been formulated several decades ago (Orton, 1927; Travis, 1931). Unlike the situation for developmental stuttering, the neural bases of acquired neurogenic stuttering are matter of certainty. The literature contains several case reports on acquired neurogenic stuttering following heterogeneous lesion sites in the left hemisphere (Orton, 1927; Canter, 1971; Carluer, Lambert, Defer, Coskun, & Rossa, 2000), the right hemisphere (Ardila & Lopez, 1986; Fleet & Heilman, 1985; Horner & Massey, 1983; Lebrun & Leleux, 1985; Soroker, Bar-Israel, Schechter, & Solzi, 1990), or both hemispheres (Balasubramanian & Hayden, 1996; Helm-Estabrooks, 1993). Although lesions of the network of brain structures supporting motor speech can lead to a variety of speech disorders, including acquired neurogenic stuttering, cases have also been reported in which neurological lesions led to the disappearance or reduction of stuttering (Andy & Bhatnagar, 1992) or to the re-emergence of childhood stuttering from which the person had recovered in early years (Helm-Estabrooks, Yeo, Geschwind, Freedman, & Weinstein, 1986; Mouradian, Paslawski, & Shauib, 2000).

Several investigators have observed striking similarities in symptomatology between developmental and acquired neurogenic stuttering. Van Borsel and Taillieu (2001) reported that even professionals were unable to differentiate between these two disorders on the basis of conversational speech samples. Van Borsel (1997) further pointed out that, based on such similarities, one may be tempted to conclude that developmental stuttering is also due to neurogenic dysfunction. In addition,

0278-2626/$ - see front matter © 2003 Elsevier Inc. All rights reserved.
doi:10.1016/S0278-2626(03)00106-4

some have argued that acquired stuttering may be influenced by the same variables that have been documented to have an influence on the symptoms of developmental stuttering. However, studies examining the role of such variables in acquired neurogenic stuttering have yielded inconsistent results (Van Borsel & Taillieu, 2001; Ringo & Dietrich, 1995). For example, most studies have reported an absence of the adaptation effect (i.e., decreased dysfluency on successive readings of the same text) in acquired neurogenic stuttering. However, the method of measuring adaptation in these studies was often unclear (Ringo & Dietrich, 1995). Similarly, one cannot generalize the available data regarding the effects of delayed auditory feedback (DAF) on acquired stuttering. DAF appears to have been applied only to four cases associated with traumatic brain injury and degenerative diseases (Van Borsel, 1997). Replicating such work, and extending it to other fluency enhancing conditions such as frequency altered feedback (FAF), may provide additional information regarding differences and similarities between developmental and acquired stuttering. One group of investigators has reported FAF to be very effective in reducing stuttering for most cases of developmental stuttering (Stuart, Kalinowski, Armson, Stenstrom, & Jones, 1996) although others have obtained less consistent findings (Ingham, Moglia, Frank, Ingham, & Cordes, 1997). To the best of our knowledge, FAF has not been investigated with acquired neurogenic stuttering.

The present work reports a case of acquired neurogenic stuttering following ischemic lesion of the right orbital frontal surface and bilateral attenuation of the pons. In addition to the neurological background, we present stuttering frequency data collected in each of the aforementioned fluency-enhancing conditions (i.e., adaptation, unison reading, DAF, and FAF).

2. Method

2.1. Subject

CP, a 57-year-old male with a medical history of non-insulin dependent hyperglycemia, hypertension, and ischemic disease, had a cerebro-vascular accident on 12-4-2000. A CT scan evaluation on 12-13-2000 revealed a new subcortical ischemia involving the right hemisphere and bilateral hypodensity of the pons (see Fig. 1). The subject was evaluated by a speech–language pathologist and was found to have dysphagia and occasional deficits in naming. Since onset of the CVA, CP's speech had reportedly become non-fluent. Family members reported that the symptoms resembled those of the childhood stuttering that the patient had shown, but outgrown, at a young age. CP and his brother claimed

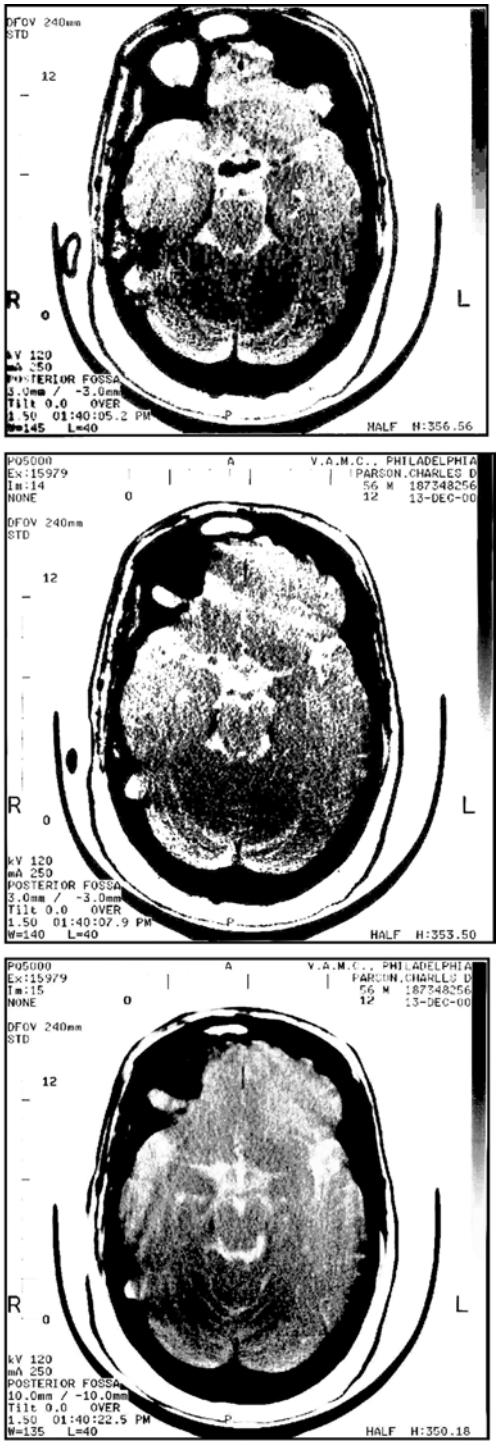

Fig. 1. CP's CT scans showing right hemisphere orbital surface lesion.

that CP had been free from symptoms of stuttering for well over four decades.

During oral reading of a 200-word passage, CP stuttered on 5% of the words. For this evaluation as well as all experimental conditions, stuttering was defined as part-word repetitions, audible and inaudible prolongations, broken words, and monosyllabic whole word repetitions.