

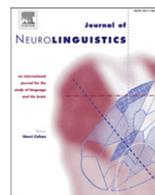


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Stuttering following acquired brain damage: A review of the literature

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ABSTRACT

Communication problems resulting from acquired brain damage are most frequently manifested as motor speech disorders such as dysarthria, syndromes of aphasia, and impairments of pragmatics. A much less common phenomenon is the onset of stuttering in adults who sustain a stroke, traumatic brain injury, or other neurologic events. When stuttering occurs in association with neuropathology, precise characterization and explanation of observed behaviors is often difficult. Among the clinical challenges presented by acquired stuttering are the problem of distinguishing this form of dysfluency from those associated with dysarthria and aphasia, and identifying the neuropathological condition(s) and brain lesion site(s) giving rise to this speech disorder. Another challenge to the precise characterization of acquired stuttering is the fact that some cases of acquired stuttering apparently have a psychological or neuropsychiatric genesis rather than a neuropathological one. In this paper we provide a review of the literature pertaining to the complicated phenomenon of acquired stuttering in adults and draw some tentative explanatory conclusions regarding this disorder.

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1. Introduction

Despite the fact that adult onset of stuttering behavior in previously fluent speakers has been discussed in the literature for more than 100 years, it remains unclear whether acquired stuttering is a distinct disorder or an epiphenomenon of other motor speech disorders such as apraxia of speech. Furthermore, conclusions regarding the nature of the underlying mechanisms of acquired stuttering remain speculative. A not insignificant factor contributing to the uncertainty surrounding acquired stuttering is the range of neurologic conditions that have been correlated with the syndrome: it has been reported in cases of single lesion strokes (e.g., Helm-Estabrooks, Yeo, Geschwind, Freedman, & Weinstein, 1986), multiple lesion strokes (e.g., Grant, Biousse, Cook, & Newman, 1999; Helm, Butler, & Benson, 1978), traumatic brain injury (TBI) (e.g., Helm-Estabrooks & Hotz, 1998; Lebrun, Bijleveld, & Rousseau, 1990; Ludlow, Rosenberg, Salazar, Grafman, & Smutok, 1987), seizure disorder (e.g., Lebrun, 1991; Sechi, Cocco, D'Onofrio, Deriu, & Rosati, 2006), Parkinson's syndrome (Canter, 1971; Koller, 1983), dialysis dementia (Rosenbek, McNeil, Lemme, Prescott, & Alfrey, 1978), and senile dementia (Quinn & Andrews, 1977). Additionally, various pharmacological agents have been reported to cause stuttering in individuals with no previous history of the disorder (see Brady, 1998 for review). In most of these cases, normal speech patterns returned after the drug was discontinued. A curious case of transient acquired stuttering in a young man with severe, chronic anorexia nervosa resulting in a significant hypoglycemic state was reported by Byrne, Byrne, & Zibin, 1993. All-in-all, given the many conditions that might give rise to acquired stuttering, one might well ask why it occurs so infrequently that it is deemed a phenomenon worthy of formal report. Indeed, the relatively rarity of acquired stuttering, itself, is in need of explanation.

Equally challenging with regard to acquired stuttering is the characterization of the speech output disorder. Few case reports provide comprehensive descriptions of the speech patterns that are labeled as "stuttering." An exception is the report by Jokel, De Nil, and Sharpe (2007) who systematically assessed the speech characteristics of 12 individuals with neurogenic stuttering secondary to either TBI or stroke. These investigators assessed their cases in relation to the six principal characteristics of neurogenic stuttering often referred to in the neurogenic stuttering literature (see for example, Helm-Estabrooks, 1999; Ringo & Dietrich, 1995; Rosenbek et al., 1978). These characteristics, which are generally understood as distinguishing acute onset, neurogenic stuttering from developmental stuttering, are as follows.

Six Features of Neurogenic Stuttering

1. Dysfluencies occur on grammatical words at a similar rate of occurrence as substantive words,
2. Repetitions, prolongations, and blocks occur in all positions of words,
3. There is a consistency in stuttering behavior across speech tasks.
4. The speaker does not appear overly anxious about the stuttering behavior,
5. Secondary symptoms such as facial grimacing, fist clenching, and eye blinking are rarely observed,
6. An adaptation effect is not observed,

After examining their 12 cases vis a vis the extent to which their behaviors did or did not conform to these six oft-cited features of acquired neurogenic stuttering, Jokel et al. (2007) concluded that their cases did not comprise a homogeneous group. Van Borsel and Taillieu (2001) presented taped speech samples of acquired and developmental stuttering to a panel of experienced professionals who regularly treated individuals with fluency disorders. The raters were asked to judge the severity of the dysfluency disorder, decide whether the disorder was consistent with the diagnosis of stuttering, and finally determine whether the disorder was neurogenic or developmental. Raters misidentified individuals with acquired stuttering as having developmental stuttering as often as they correctly identified individuals with developmental stuttering as having that form of dysfluency.

It would appear that sufficient level of uncertainty has been raised about the validity of the "six features of neurogenic stuttering" that they should be collectively regarded as a "rule of thumb" rather than pathognomonic indicators of neurogenic stuttering. In other words, if an individual with a history of a documented neurologic event shows all six behaviors associated with neurogenic, acquired

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