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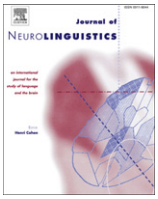

Dysfluency levels during repeated readings, choral readings, and readings with altered auditory feedback in two cases of acquired neurogenic stuttering

Venu Balasubramanian [a,b,*], Kristine L. Cronin [c], Ludo Max [d,e]

[a] Seton Hall University, South Orange, NJ, USA
[b] VA Medical Center, Philadelphia, PA, USA
[c] University of Connecticut, Storrs, CT, USA
[d] University of Washington, Seattle, WA, USA
[e] Haskins Laboratories, New Haven, CT, USA

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ABSTRACT

We investigated how conditions that are known to be fluency-enhancing for individuals with persistent developmental stuttering (PDS) affect the speech of individuals with acquired neurogenic stuttering (ANS). Based on others' claims that the overt speech dysfluencies of PDS and ANS are difficult to distinguish, as well as the derived hypothesis that similar neural mechanisms—or even similar neural deficits—may underlie both disorders, the purpose of the present work was to examine whether speakers with ANS experience the same improvements in speech fluency as do speakers with PDS during (a) repeated readings of the same material, (b) choral readings with a second speaker, and (c) readings with delayed or frequency-altered auditory feedback. Two individuals with ANS read passages out loud in each of these conditions. Results differed from those typically reported for individuals with PDS and, instead, showed (a) only a small amount of adaptation during repeated readings, (b) a small improvement for choral reading that never reached nearly fluent speech, and (c) an increase, rather than decrease, in dysfluency in the conditions with altered auditory feedback.

* Corresponding author. Department of Speech-Language Pathology, Seton Hall University, 400 South Orange Avenue, NJ, USA. Tel.: +1 973 275 2912 fax: +1 973 275 2370.
E-mail address: balasuve@shu.edu (V. Balasubramanian).

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These findings are consistent with those for another case of ANS previously reported by our own group. Possible reasons for the lack of facilitation of fluent speech under these experimental conditions in cases of ANS are discussed.

The cognitive and sensorimotor processes involved in speech production are under the control of an integrated, complex network of neural structures widely distributed in the central and peripheral nervous systems (Dhanjal, Handunnetthi, Patel, & Wise, 2008; Ghosh, Tourville, & Guenther, 2008; Guenther, 2006). Speech motor programming, a component process as conceptualized by various clinical models of motor speech production (Duffy, 2005; van der Merwe, 1997), is supported by the left-hemisphere perisylvian structures, prefrontal cortex, basal ganglia, cerebellum, and supplementary motor area (van der Merwe, 1997). Additionally, the crucial role of afferent systems, including those providing auditory and somatosensory feedback, has been widely recognized for both learning and maintaining high-precision speech motor skills (Dhanjal et al., 2008).

Specifically with regard to the role of the *auditory* system in speech production, studies suggest that (a) bilateral temporo-parietal regions function as a self-monitoring system (Hashimoto & Sakai, 2003), (b) auditory feedback plays a crucial role in the acquisition of speech motor skill before a shift takes place from a primarily feedback-based control strategy to a feedforward control strategy (Guenther, 2006; Guenther, Gosh, Nieto-Castanon, & Tourville, 2006; Harrington & Tabain, 2006; Max, 2004), and (c) both during and after speech development, information provided by the auditory feedback system is used to update adaptive internal representations (internal models) of the mapping between motor commands and vocal tract acoustic output (Houde & Jordan, 1998; Max, Wallace, & Vincent, 2003; Villacorta, Perkell, & Guenther, 2007).

Over the years, numerous authors have proposed that either deficiencies within the central auditory pathway itself or difficulties with the correct use of this auditory information for the control of speech movements may play an important role in the etiology of persistent developmental stuttering (PDS) (for a review, see Bloodstein & Bernstein Ratner, 2007). PDS is a disorder of speech fluency characterized by sound and syllable repetitions, audible and inaudible sound prolongations, and broken words (Conture, 1990, 2001; Wingate, 1964). Typically, the frequency of occurrence of these dysfluencies decreases in a variety of fluency-enhancing conditions. For example, when a person who stutters repeatedly reads the same passage out loud, stuttering frequency decreases on average by approximately 50% (Frick, 1955; Johnson & Knott, 1937; Van Riper & Hull, 1955)—possibly representing a form of speech motor learning (Max, Caruso, & Vandevenne, 1997). Similarly, if a person who stutters reads out loud while hearing auditory feedback that is either delayed (delayed auditory feedback, DAF) or frequency-shifted (frequency-altered auditory feedback, FAF), stuttering frequency also decreases (Macleod, Kalinowski, Stuart, & Armson, 1995; Martin & Haroldson, 1979; Soderberg, 1969; Stuart, Frazier, Kalinowski, & Vos, 2008; Stuart, Kalinowski, Armson, Stenstrom, & Jones, 1996). The most dramatic decrease in stuttering frequency (often to nearly fluent levels) is usually observed in choral readings (also called unison readings) in which a second individual reads out loud in synchrony with the individual who stutters (Ingham & Packman, 1979; Kiefte & Armson, 2008; Max et al., 1997).

Interestingly, some authors have suggested that the overt symptoms of acquired neurogenic stuttering (ANS)—that is, stuttering with an onset that occurs after speech development and that is a result of demonstrable neurological damage—are difficult to distinguish from those of PDS (Van Borsel & Taillieu, 2001). If so, one could speculate that similar neural mechanisms (e.g., processes of movement planning, sensorimotor integration, etc.), or even similar neurological deficits (e.g., disrupted neural connectivity in certain critical brain areas), underlie ANS and PDS. However, other researchers in the area of motor speech disorders have expressed reservation about the existence of such similarities (Canter, 1971; Duffy, 2005; Jokel, De Nil, & Sharpe, 2007). Not only does ANS occur as a result of lesions at any of a large number of very different neuroanatomical sites (e.g., Balasubramanian & Hayden, 1995; Balasubramanian, Max, Van Borsel, Rayca, & Richardson, 2003; Doi et al., 2003; Heuer, Sataloff, Mandel,