Speech motor programming in hypokinetic and ataxic dysarthria

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Abstract

It is widely accepted that the cerebellar and basal ganglia control circuits contribute to the programming of movement. Converging evidence from neuroimaging, limb control, and neuropsychological studies suggests that (1) people with cerebellar disease have reduced ability to program movement sequences in advance of movement onset and (2) people with Parkinson's disease are unable to maintain a programmed response or to rapidly switch between responses. Despite a substantial supporting literature, no studies have addressed these potential areas of speech programming disruption for speakers with ataxic and hypokinetic dysarthria. Control participants and adults with dysarthria completed speech reaction time protocols designed to capture these aspects of utterance preparation. Results provided initial support for processing deficits in speakers with ataxic and hypokinetic dysarthria that are separable from motor execution impairments.
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1. Introduction

The dysarthrias have traditionally been thought to result from a disruption of motor speech execution (Yorkston, Beukelman, Strand, & Bell, 1999) with little attention given to the preparatory processes of speech production. One particular aspect of speech preparation that merits consideration is motor programming, or the process of transforming linguistic–symbolic representations into a motor code. Despite divergent theoretical accounts of speech motor programming, it is generally accepted that neuromuscular representations of an utterance exist in some form prior to motor execution (Abbeduto, 1987; Gordon & Meyer, 1987; Roelofs, 2002; Schönle, Hong, Benecke, & Conrad, 1986; Sternberg, Knoll, Monsell, & Wright, 1988; Van der Merwe, 1997; Yaniv, Meyer, Gordon, Huff, & Sevald, 1990, but see Kelso, Tuller, & Harris, 1983). Though the term "motor programming" is most consistent with the nomenclature in extant research, it is understood that we are not yet able to delineate stages in the preparation of an utterance (Rogers & Storkel, 1998). Thus, motor programming is used in a general sense to refer to processes that occur prior to speech motor execution (but after word retrieval and sentence planning). It has not yet been shown to be separable from processes such as motor planning where phonemes are specified as general motor goals (Van der Merwe, 1997).

Multiple neural areas are thought to be involved in the motor programming of movement, including the basal ganglia, cerebellum, supplementary motor area, and frontal system (Cunnington, Windischberger, Deecke, & Moser, 2002; DeLong, 2000; Dirnberger et al., 2000; Kuriki, Mori, & Hirata, 1999; Leuthold & Jentzsch, 2002; Sakai et al., 2000). It is widely accepted that the basal ganglia and cerebellar circuits, in particular, have a fundamental role in the programming of movement. A wealth of neurophysiological research on non-human primates (e.g., Alexander & Crutcher, 1990; Bioulac, Burbaud, & Varoqueux, 1995; Chapman, Spidalieri, & Lamarre, 1986; Jaeger, Gilman, & Aldridge, 1993; Schultz & Romo, 1992) and neuroimaging studies

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of healthy adults (e.g., Cui et al., 2000; Deiber, Ibanez, Sadato, & Hallett, 1996; Dreher & Grafman, 2002; Horwitz, Deiber, Ibanez, Sadato, & Hallet, 2000; Jueptner & Weiller, 1998; Sakai et al., 2000) have substantiated this premise. The distinct roles of the basal ganglia and cerebellar circuits in motor programming have been illuminated by converging evidence from limb reaction time (RT) studies of adults with Parkinson's disease and cerebellar disease. The conclusions of these limb studies, highlighted below, are consistent with the speech sequelae of hypokinetic and ataxic dysarthria.

The extensive literature addressing limb motor programming abilities of adults with PD has consistently identified two areas of disruption. First, there is considerable agreement regarding the reduced ability of people with Parkinson's disease to rapidly switch from one movement (or motor program) to another (Benecke, Rothwell, Dick, Day, & Marsden, 1987; Contreras-Vidal & Stelmach, 1996; Delwaide & Gonce, 1993; Roy, Saint-Cyr, Taylor, & Lang, 1993; Rubchinsky, Kopell, & Sigvardt, 2003; Weiss, Stelmach, & Hefter, 1997). This impairment is thought to reflect difficulty with modifying or inhibiting an ongoing response (Inzelberg et al., 2001; Kropotov & Etlinger, 1999; Mink, 1996) or with activating new motor programs (Haaland & Harrington, 1990). Deficient transitioning to a new movement may become particularly pronounced when a rapid shift from a prepared response to a new response is required (Marsden, 1984). Second, limb RT studies have supported the premise that people with PD have difficulty maintaining programmed information prior to movement initiation (Berardelli, Rothwell, Thompson, & Hallett, 2001; Gueye, Viallet, Legallet, & Trouche, 1998). This hypothesis has gained empirical support from numerous kinematic investigations which have speculated that programmed representations of the movement decay prior to (and during) movement initiation (Agostino, Berardelli, Formica, Accornero, & Manfredi, 1992; Gentilucci & Negrotti, 1999a, 1999b; Romero, Van Gemmert, Adler, Bekkering, & Stelmach, 2003; Stelmach, Garcia-Colera, & Martin, 1989).

Symptoms of hypokinetic dysarthria are consistent with these two hypothesized deficits of motor programming. Speech behaviors, such as abnormally placed pauses, difficulty with progression through an utterance and difficulty initiating articulation, are characteristic of speakers with PD (Gurd, Bessel, Watson, & Coleman, 1998; Svensson, Henningson, & Karlsson, 1993) and could result from difficulty maintaining the speech motor program. Additionally, reduced ability to switch between speech motor programs would be consistent with speech behaviors such as difficulty stopping an ongoing response, marked hesitations between movement segments, and occasional inability to switch from one to another movement. These behaviors are indeed evident in the speech of individuals with PD (Adams, 1997; Duffy, 1995).

A few pioneering studies have been conducted of speech motor programming in speakers with PD. While findings from these studies did not support a disruption of speech motor planning or programming, there are methodological and theoretical considerations. The protocols employed to assess motor programming disruption did not typically separate speech programming from speech execution effects or failed to stress motor programming operations (e.g., Connor, Ludlow, & Schulz, 1989; Ho, Bradshaw, Cunnington, Phillips, & Iansek, 1998; Ludlow, Connor, & Bassich, 1987). For example, Connor et al. (1989) examined production of the isolated syllables /ba/, /da/, and /ga/ compared to repeated syllables (e.g., /papa/ or /pata/ or /paka/) produced as quickly as possible for seven seconds. The authors rightfully speculated that speech planning in Parkinson's disease should be more impaired for longer and more complex speech tasks. Though the authors found no significant acoustic differences between the group with PD and the controls, it may be that the complexity of the task was insufficient to adequately engage motor programming operations. Additionally, none of the investigations were tailored to manipulate specific aspects of speech motor programming, such as maintenance of a prepared utterance or transitioning between utterances. Instead, these studies focused on different potential manifestations of programming deficits, such as impaired relative timing (e.g., Ludlow et al., 1987).

In sum, the study of limb movements in people with PD has led to emerging consensus for two potential areas of programming disruption that may exacerbate motor execution deficits: (1) reduced ability to rapidly switch between movements, and (2) reduced ability to maintain programmed information. No investigations of speech motor programming in adults with PD have examined these possible areas of disruption.

Studies examining limb motor programming in individuals with cerebellar disease are relatively limited and inconclusive when compared to the vast literature on PD. Protocols often have involved simple, one-step tasks (e.g., Bonnefoi-Kyriacou, Trouche, Legallet, & Viallet, 1995; Jahanshahi, Brown, & Marsden, 1993), which may have obscured difficulties in movement programming as simpler movements are minimally affected by cerebellar lesions (Goodkin, Keating, Martin, & Thach, 1993). However, Inhoff, Diener, Rafal, and Ivry (1989) examined motor performance based on movement sequences. Thirteen participants with bilateral cerebellar disease, 12 participants with unilateral cerebellar disease, and eight healthy controls completed sequences that ranged from 1 to 3 keypress components in a RT task. Control participants, as well as those with mild cerebellar dysfunction, showed the expected "sequence length effect," that is, latencies to initiate the response increased as the sequence length increased. Conversely, no effects of sequence length were found for participants with moder-