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Stuttering treatment control using P300 event-related potentials

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ABSTRACT

Positron emission tomography studies during speech have indicated a failure to show the normal activation of auditory cortical areas in stuttering individuals. In the present study, P300 event-related potentials were used to investigate possible effects of behavioral treatment on the pattern of signal amplitude and latency between waves. In order to compare variations in P300 measurements, a control group paired by age and gender to the group of stutterers, was included in the study. Findings suggest that the group of stutterers presented a significant decrease in stuttering severity after the fluency treatment program. Regarding P300 measurements, stutterers and their controls presented results within normal limits in all testing situations and no significant statistical variations between pre and post treatment testing. When comparing individual results between the testing situations, stutterers presented a higher average decrease in wave latency for the right ear following treatment. The results are discussed in light of previous P300 event-related potentials and functional imaging studies with stuttering adults.

Educational objectives: The reader will learn about and be able to describe the: (1) use of P300 event-related potentials in the study of stuttering; (2) differences between stuttering and non-stuttering adults; and (3) effects of behavioral fluency treatment on cerebral activity in stuttering speakers.

1. Introduction

Although the etiology of stuttering remains obscure, hypothesis regarding the breakdown of motor speech control is central to current theories of the etiology of stuttering. Given this perspective, stuttering is characterized by disruptions in speech motor behavior (repeated or prolonged articulatory and phonatory actions) that result in the breakdown of speech fluency (sound and syllable repetitions, sound prolongations, blocks and broken words) (Max, 2007).

Ever since the studies of Orton (1928) and Travis (1931) several researches have as a recurring theme the neurophysiological bases for stuttering, trying to find possible differences in cerebral dominance between fluent and disfluent individuals. Researchers have speculated about potential involvement of aberrant neural processes in the onset and development of stuttering. Much of the earlier research into the nature of these hypothesized brain processes was based largely on the use of behavioral observations and electromyographical measures (Conture, Schwartz, & Brewer, 1985; De Nil & Abbs, 1991; Freeman & Ushijima, 1978; Kelly, Smith, & Goffman, 1995; MacFarlane & Prins, 1978; McClean, 1987; McClean, Goldsmith, &

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Cerf, 1984; Reich, Till, & Goldsmith, 1981; Smith, Denny, Shaffer, Kelly, & Hirano, 1996; Smith & Luschei, 1983; Zimmerman, 1980a, 1980b)

Functional brain imaging techniques such as positron emission tomography (PET) or functional magnetic resonance imaging (fMRI) provide a further approach to the investigation of the cerebral correlates of speech motor control. Brain imaging by PET or single-photon emission computed tomography (SPECT) studies of stutter probands have already been successful in identifying candidate regions of interest that distinguish affected from unaffected individuals. So far, these studies have indicated interhemispheric functional asymmetry, i.e. in fluent speakers the activation for speech and language is predominantly from the left hemisphere, whereas for stutterers activation is diffuse or predominantly to the right. Permanent hypometabolism of the left caudate has also been identified, and it has been observed that in the individual who stutters this basal ganglion is almost 50% less active than in fluent speakers. Apparently there is an alteration in the activity of the cerebellum circuit components in individuals who stutter, in comparison with fluent individuals. This is aggravated when using spontaneous speech, whereas this activity becomes normal in a situation of induced fluent speech (e.g. reading in chorus). When compared to fluent individuals, stutterers demonstrate cortical hypoactivity of the areas associated with language processing (primary auditory cortex and Wernick area) and hyperactivity of the areas associated with motor functions (e.g. Andrade, Meneghetti, Sassi, & Bertini, 2001; Foundas, 2001; Ingham et al., 1996; Kroll & De Nil, 2000; Sandak & Fiez, 2000; Van Borsel, Sunaert, & Engelen, 2005; Wu et al., 1995).

Studies with fMRI have indicated relative changes in activity or laterality between stutterers and fluent controls. The meta-analyses of these studies highlighted three neural signatures that seemed more or less specific to the stuttering group: overactivation of motor areas, including the primary motor cortex, pre-motor area and cingulum; predominantly to the right in the frontal operculum and anterior cingulum; absence of activation in auditory areas bilaterally; and overactivation in the vermal region of lobule III of the cerebellum (Brown, Ingham, Ingham, Laird, & Fox, 2005).

Auditory processing is a single factor among many potential contributing variables associated with stuttering (Rosenfield & Jerger, 1984). In stuttering research, altered auditory feedback (AAF) is a collective term for conditions that involve electronically altering the speech signal so speakers perceive their voice differently from normal. Numerous studies have demonstrated that stuttering frequency is often immediately reduced in response to AAF (e.g. Howell, Sackin, & Williams, 1999; Lincoln, Packman, & Onslow, 2006; Stuart, Kalinowski, Rastatter, Saltuklaroglu, & Dayaly, 2004; Van Borsel, Reunes, & Van den Bergh, 2003; Van Borsel, Sierens, & Pereira, 2007)

Given that PET studies during speech have indicated a failure to show the normal activation of auditory cortical areas in stuttering individuals, it is possible that fluency enhancing devices and techniques, i.e. delayed auditory feedback (DAF), frequency altered auditory feedback (FAF), masking and speaking in chorus, is not a result of those modifications by themselves, but of their common role in providing external auditory stimulus to facilitate activation of the auditory cortex (Max, 2007).

During the 90s, a later occurring component of the late cortical response, the P300 event-related potential, received considerable attention as a possible tool for investigating cortical mechanisms related to auditory processing (Suzanne, Kelly, & Thorne, 2001). Measurements of evoked electrical potentials at the scalp are a widely used approach to the study of speech sound perception.

The P300 is a complex event-related potential component occurring between 300 ms and 600 ms post-stimulus onset in response to a low probability stimulus, often elicited by a simple oddball principle (Polich & Kok, 1995). P300 amplitudes are related to factors such as stimulus probability, quality, and duration, and are associated with attention and task relevance (Hall, 2006). The amplitude of the P300 is affected by attention and memory load and is subject to habituation and reduction in repetitive tasks, indicating that the P300 may reflect neuronal activity related to focusing on new and/or novel information and updating working memory. The P300 is an endogenous potential, dependent on internal factors, and can provide insight into underlying cognitive processes. (Alonso-Prieto, Fernandez-Concepcion, Jimenez-Conde, & Machado, 2002; Alonso-Prieto, Alvarez, Reyes-Berazain, Barroso-Garcia, & Pendo, 2002; Hampton & Weber-Fox, 2008; Kibbe-Michal, Verkest, Gollegly, & Musiek, 1986).

In the early 90s evidence was presented, by using combined magnetoencephalography and magnetic resonance imaging, suggesting that the insula and auditory cortical areas of the superior temporal lobe are major sites of generation of the P300 response (Rogers et al., 1991). Therefore, P300, might provide valuable information related to hemispheric processing in stutterers (Peñaloza-López, Tellez, Pérez-Ruiz, Silva, & Garcia-Pedrosa, 2008). However, results have been contradictory.

Ferrand, Gilbert, and Blood (1991) found no differences between stutterers and nonstutterers in the latency of P300 response while Morgan, Cranford, and Burk (1997) found that P300 were higher in amplitude over the left hemisphere than the right hemisphere, suggesting auditory processing differences in stutterers for non-linguistic pure-tone stimuli. Hampton and Weber-Fox (2008) found that the P300 mean amplitudes elicited in adult stutterers tended to be reduced overall compared to fluent individuals, suggesting the possibility of weaker updates in working memory for representations of the target tone stimuli in stutterers.

Other studies, using brain-evoked response testing, revealed no relationship between brainstem-evoked response and severity of stuttering (Blood & Blood, 1984) and presented heterogeneous findings, regarding cortical hemispheric differences, for stutterers (Rosanowski et al., 1998).

The purpose of the present study was to investigate the relationship between stuttering amelioration and cerebral activity. P300 event-related potentials were obtained pre and post-treatment in order to investigate changes in signal amplitude and in the latency between waves.