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Event-Related Potentials Distinguish Fluent and Stuttered Speech

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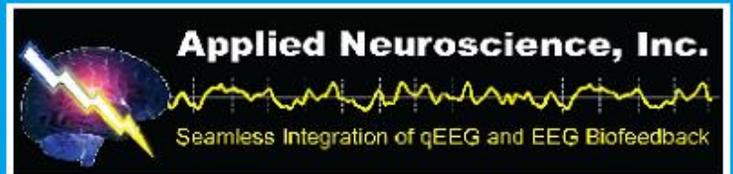
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ABSTRACT. *Background.* This study aimed to find the best possible approach to neurotherapy for stuttering.

Method. Five right-handed Dysfluent Speakers and five right-handed Fluent Speakers were compared before dysfluent and fluent speech onset with electrical event related potentials. EEG electrodes were at inferior frontal, precentral, and temporoparietal locations on each hemiscalp. Participants processed two words displayed consecutively at center screen, the first or second of which was to be uttered aloud according to the subsequent command digit being 1 or 2.

Results. The Fluent Speakers manifested significant left hemisphere contingent negative variations (CNVs) prior to the words and the contingency stimulus. The Dysfluent Speakers presented overall smaller CNVs that were more marked over the right hemisphere than left. This pattern was observed before both their stuttered and fluent words, with the former differing significantly from the fluent speakers hemispheric CNV pattern.

Conclusion. These results indicate that dysfluent speech is related to abnormal hemispheric asymmetry. Neurofeedback therapy for persons who stutter could aim at normalizing contingent negativity asymmetry seconds prior to speech.

KEYWORDS. CNV, contingent negative variation, EEG, EEG neurofeedback dysfluent speech, event related potentials, evoked potentials, stuttering,

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INTRODUCTION

EEG Investigations of Stuttering

Alpha suppression (an index of cortical activation) during speech in Dysfluent Speakers (DS) has been observed to be abnormally intense over the right hemisphere (Moore & Haynes, 1980; Moore & Lang, 1977; Moore & Lorendo, 1980). Moore (1984) observed that alpha suppression was right sided during dysfluent speech and left sided during fluent speech in a single patient. Moore (1986) found the degree of right sided alpha suppression was positively correlated with severity of dysfluency. Wells and Moore (1990) carried out a topographical EEG study of rest and speech in DS and Fluent Speakers (FS). During both rest and speech, the DS presented significantly more alpha power (i.e., deactivation) over left posterior areas, whereas the FS presented alpha suppression (i.e., activation) during speech at the same site. In anterior areas, the DS presented greater alpha suppression on the right, whereas the FS presented greater alpha suppression on the left with posttreatment fluent speech. Boberg, Yeudall, Svhopflocher, and Bo-Lassen (1983) compared fluent and dysfluent episodes before and after behavioral treatment for stuttering. They found pretreatment alpha suppression on the right during dysfluent speech and on the left after treatment. Ozge, Toros, and Comelekoglu (2004) found a similar significantly abnormal asymmetry in dysfluent speakers in the delta and alpha bands using quantitative EEG. Khedr, El-Nasser, Abdel Haleem, Bakr, and Trakhan (2000) obtained similar results.

Event-related potential investigations of stuttering. Speech-related potentials consist of sweeps time-locked to utterance onset. Early research aiming to identify speech-specific signals and topography, in normals, has been plagued by muscle artifacts (Brooker & Donald, 1980), a problem compounded in the investigation of DS who are known to occasionally present tremendous muscular struggling during dysfluent utterances. Zimmerman and Knott (1974) investigated the contingent negative variation (CNV) prior to S2 stimuli commanding

verbal and nonverbal responses in DS compared to FS. The DS presented a heterogeneous profile. Nevertheless, it was possible to note that whereas a left dominance of the CNV preceded speech in four of five FS, only 22% of DS presented that profile during fluent speech. A similar effect was observed for nonverbal (key press) responses as well. Prescott and Andrews (1984) claimed that prespeech CNVs were larger on the right side of the scalp in DS compared to FS. They did not present a test of the group by hemisphere interaction. Pinsky and McAdam (1980) obtained the opposite profile, also nonsignificant. Most evoked response potential (ERP) studies of other components in stuttering did not find, or did not look for, abnormal hemispheric asymmetry (Corbera, Corral, Escera, & Idiazabal, 2005; Cuadrado & Weber-Fox, 2003; Rosanowski et al., 1998; Vartanov, Glozman, Kisel'nikov & Karpova, 2005; Weber-Fox, Spencer, & Spruill, 2004). Morgan, Cranford, and Burk (1997) presented common (80% probability) versus rare (20% probability) auditory tones to people who stutter and to fluent speakers, requiring no response. The dysfluent group had greater late positive (P300) wave difference (between rare and common tones) over the left than right hemisphere, compared to the fluent speakers. Visual evoked potentials are usually normal in people who stutter, but weak amplitude is occasionally observed (Khedr et al., 2000).

Inconsistent results have occurred in the electrophysiological literature, as they also have in the brain imaging literature. The dominant trend of both literatures is that DS have underactivated left hemisphere circuits for linguistic stimuli, and this deficiency seems to be aggravated by an interference effect manifest in overactivation of the right hemisphere (Brown, Ingham, Ingham, Laird, & Fox, 2005; De Nil & Kroll, 2001). Despite the fact that electrophysiological techniques provide better temporal resolution than do metabolic imaging techniques, as far as we could determine, not a single electrophysiologic study has yet reported results significantly distinguishing signals related to stuttered versus fluent utterances in dysfluent speakers.

Motoric, cognitive, and perceptual anomalies potentially contributing to speech dysfluency. Stuttering manifests itself as a motor abnormality, but the problem is not limited to abnormal motricity. Many nonmotoric anomalies may contribute to dysfluent speech. For example, dysfluent speakers manifest abnormal attention (Bosshardt, 2006; Bosshardt, Ballmer, & de Nil, 2002; Heitmann, Asbjornsen, & Helland, 2004), abnormal short-term memory (Hakim & Ratner, 2004; Vartanov et al., 2005) and abnormal visual and auditory speech perception (Corbera et al., 2005; Cuadrado & Weber-Fox, 2003; Rosanowski et al., 1998; Weber-Fox et al., 2004). Thus, it is important to focus on all these aspects in the search for brain based direct contributions to stuttering.

The purpose of this study was to find EEG markers of dysfluent speech that could be used for EEG feedback therapy. The specific innovative purposes of the study to be reported next were (a) to subject dysfluent and fluent speakers to attentional and memory demands and collect resultant speech samples, (b) to collect speech-related electrical signals preceding dysfluent utterances distinctly from fluent utterances within the DS and the fluent utterances in FS, (c) to apply a richer EMG electrode array for artifact rejection than previous EEG studies of dysfluent speech, and (d) to analyze the raw digitized electrophysiological data with more sophisticated multivariate statistics than previously used.

METHOD

Participants

This project had received an ethics certificate from Université du Québec à Montréal. Exclusion criteria included an abnormal neurological history, substance abuse, or prior psychiatric consultation. Only right-handed male participants were included to reduce heterogeneity of electrophysiological profiles (Foundas, Corey, Hurley, & Heilman, 2006). Handedness was determined with a hand preference questionnaire (Collin & Braun, 1997), which included all the

unimanual items from Crowitz and Zenner (1962), Provins and Cunliffe (1972) and Fennell (1986). The FS group was recruited via a national newspaper and consisted of 18 Caucasian French-speaking right-handed male participants. Their first language was French, they volunteered to participate, they signed a consent form, and they received CDN\$30 for their effort. The DS group participants were recruited from a province-wide association for dysfluent speakers. They were diagnosed as severe developmental stutterers by speech therapists and received unsuccessful behavioral therapy. After application of experiment-related exclusion criteria (excessive muscular struggling, insufficient dysfluency on the experimental task), 8 of 13 dysfluent participants were excluded. The remaining 5 DS participants displayed stuttered speech (repeated, struggled, or delayed speech) during 1 hr interview conversation about the participants' dysfluency. The 5 participants chosen for the FS group (age = 21.2, education = 14.6 years) were younger ($p = .03$) but equivalent in education compared to participants in the DS group (age = 28.4, education = 14.2 years). Collegiate studies were exclusively in the liberal arts in both groups, and there are no known developmental changes of EEG or evoked potentials between the ages 21 and 28; as a result, age difference was given no further consideration.

Procedure

Twenty-one Ag/AgCl electrodes included three bilaterally symmetrical pairs for EEG: inferior frontal at iF3 (Broca's area: 11 cm lateral to vertex and 4 cm anterior; McAdam & Whitaker, 1971; Zimmerman, 1980) and iF4, precentral at PC3 (speech premotor area: 9 cm lateral to vertex and 2 cm anterior) and PC4 and temporoparietal at TP3 (Wernicke's area: 12.5 cm lateral to vertex and 5 cm posterior; Pinsky & McAdam, 1980) and TP4. There were nine sites for EMG: at the left temporalis muscle, the left masseter muscle, the left and right orbitalis muscles, the left and right cheeks, to the left and right of the lips, and over the larynx.

Electrodes for lateral eye movement recording were placed 1 cm above the left external canthus and 1 cm below the right external canthus, and for vertical eye movement recording under the supra and infraorbital crests. Linked reference electrodes were placed at the left and right mastoids. The ground was placed on the left forearm. Low pass filters were all set at 50 Hz and high pass filters at 0.1 Hz. Sampling rate was 250 Hz using a NeuroScan Synamps 2 system.

The stimuli were delivered on a PC computer screen by a custom program written in ASYST, which also sent controlled pulses to the Neuroscan electrophysiological system. Stimuli consisted of 214 French words beginning with a consonant and selected to elicit maximum dysfluency (complex consonants, difficult-to-pronounce phonemic sequences, high cognitive demands; Prescott, 1988). The DS were asked to avoid any fluency strategy previously learned. Participants were asked to avoid any prearticulatory movements and to fixate center-screen cross hairs, which were displayed whenever words were not displayed within each trial. The display sequence was as follows: A center screen cross hair was displayed for 750 msec. A word was then displayed at center screen for 1200 msec, followed by 1200 msec of center screen cross hairs, then a second word for 1200 msec, and 1200 msec of center screen cross hairs; finally, the digit 1 or 2 at center screen designated which word should be uttered out loud as quickly as possible and was displayed until a vocalization occurred. This task was designed to solicit attentional and memory resources prior to and during speech planning because these task demands are known to significantly and selectively challenge people who stutter (Bosshardt, 2006; Bosshardt et al., 2002). Vocalization was monitored by the digitized input from a microphone to the ASYST program and a specific code was immediately sent into the time line. When the participants took more than 800 msec to respond, a message "TOO LONG" was displayed (in French) to keep pressure on the participants to respond quickly (i.e., to favor stuttering). The experimenter terminated the

trial by pressing one of three buttons coding for DYSFLUENT UTTERANCE, FLUENT UTTERANCE, or REJECTED TRIAL (prearticulation movement or experimenter uncertainty). A dysfluent trial was recognized if the participant hesitated inordinately (>800 msec), audibly stuttered, repeated any part of the word (consonant, vowel, or phoneme), or pronounced the word *explosively*. This response code was also sent to the EEG recording system to allow sorting by utterance type. This experimenter classification of a trial launched the next trial starting with 750 msec of cross hairs followed by a stimulus sequence. Omissions (>4000 msec) and anticipations (<150 msec) were automatically coded in the time line and the trial was automatically rejected. Each participant was presented 214 word pairs altogether, in a fixed order.

Statistical analyses of the ERPs used the PC1 (Achim, 1995) and PC2 (Achim, 2001) tests respectively for within-group repeated measures comparisons and between-group comparisons. Both use principal component analysis to reduce the ERP waveform, or difference in ERP waveform in the case of PC1, to one or two factor scores, which are then analyzed by a regular F test (PC1) or Hotelling T^2 (PC2), reported as the equivalent *F* statistics. PC1 was also used within groups on ERPs, rather than on ERP differences, as a test of signal detection. Signal was detected when the average ERPs of the various participants look sufficiently alike for their projection on the dominant shape (first principal component) to differ significantly from zero. Only two-level factors can be analyzed at a time with PC1 or PC2, but differences of differences allow tests of any interaction of two-level factors.

Signal analysis spanned two intervals, namely -4.8 to -1.0 sec and -1.0 to 0 sec from the command digit onset (indicating which word to utter). These two intervals respectively correspond to active listening and maintaining the words in memory for an indication of which word to utter. The 1-sec interval prior to the command digit onset was analysed separately to simplify interpretation of the CNVs because a CNV prior to a

command stimulus could reflect a nonspecific preparation for speech, even though the specific word to utter is not yet designated. This interval is also distinct from the preceding one in that the participant is not seeing or reading words. Although the bulk of CNV studies produced this wave in preparation for a speeded motor response, CNVs can also be produced by expectancy of a stimulus laden with information critical for a decision (Tarkka & Basile, 1998). The CNV reflects expectancy, anticipation and focussing of attention (Tarkka & Basile, 1998). For each trial and each channel, the mean amplitude over the 0.5-sec interval preceding onset of the initial fixation cross was used as baseline level. All the channels and all the trials were scrutinized by the experimenters for ocular or muscular artifact affecting the EEG trials thus justifying exclusion of the trial. Trial rejection rate (whether because of experimenter uncertainty, experimenter-detected prearticulation movement, or postacquisition artifact rejection) was 24% for the FS and 40% for the DS (delta = *ns*). Percentages of dysfluent word utterances were zero in the normals, and 25, 19, 35, 42 and 60% in the five DS.

RESULTS

Reaction Times

Machine-detected vocal reaction times were normally distributed within group and condition, but the variances of the DS were significantly larger than those of the FS, $t(8) = 3.3$, $p = .03$, so the group difference was submitted to a t test for heterogenous variances. The DS were significantly slower than the FS, $t(8) = 2.8$, $p = .045$. A paired t test indicated that within the DS, the dysfluent utterances occurred later (1675 msec) than the fluent ones (1018 msec), $t(4) = 11.5$, $p = .03$. For comparison, the mean vocal reaction time in the FS was 499 msec. There was great variability of stimulus-to-utterance interval in the DS ($SD = 515$ msec for dysfluent speech, $SD = 136$ msec for fluent speech) compared to the FS ($SD = 89$ msec). Eighty percent of the dysfluent utterances were

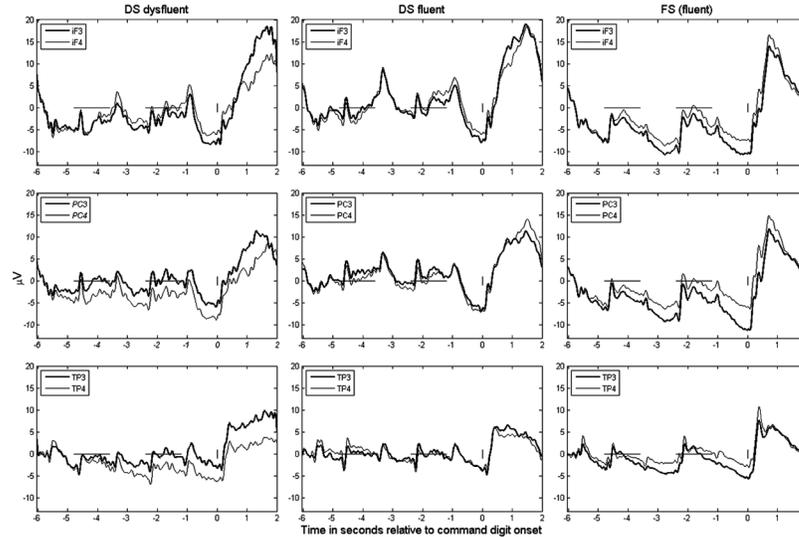
because of silent excessive delay (> 800 msec), detected and coded automatically. Thus, interjudge reliability of the judgements concerning presence of stuttering was not determined.

ERPs

The PC1 test for signal detection of repeated measures was used to test the hypothesis of a difference between the morphologies of the EEG related to fluent and dysfluent utterances (utterance type) of the DS as a function of hemisphere. Similar comparisons of the DS and FS groups were made using the PC2 test. More specifically, the analyses were carried on all three pairs of channels collectively (by appending the individual waveshapes of each hemisphere end to end). This was followed by a separate analysis for each channel pair to localise the effects among the three pairs of channels.

For the interval from -4.8 to -1.0 relative to digit onset, the comparisons of the FS and DS (dysfluent samples) revealed no main group or hemisphere effect but a significant Group \times Hemisphere interaction, $F(2, 7) = 25.98$, $p = .0006$. Breakdown of this effect by channel pair indicated that it was present only at the precentral, $F(2, 7) = 8.50$, $p = .013$, and temporoparietal, $F(2, 7) = 6.10$, $p = .029$, sites. For descriptive purposes, these two local Group \times Hemisphere interactions were further analyzed into simple effects of group at each electrode of the pair and of hemisphere within each group. No group effect was obtained at any electrode ($p > .1$). Significant hemisphere effects were obtained for the FS group for both the PC3-PC4 pair, $F(1, 4) = 8.90$, $p = .041$, and the TP3-TP4 pair, $F(1, 4) = 7.94$, $p = .048$, whereas the corresponding effects within the DS group seen in the opposite direction in Figure 1, respectively, fell short of significance ($p = .061$ and $p = .131$). Comparison of the FS and DS groups at the -4.8 to -1.0 intervals yielded no significant group effect. Comparison of the fluent and dysfluent trials within the DS

FIGURE 1. Means of left (thick line) and right (thin line) infero-frontal, precentral and temporo-parietal signals synchronized on command digit onset (time zero), with amplitude zero averaged using the interval between 1250 ms and 750 ms prior to onset of the first word. *Note.* The two horizontal bars at zero amplitude represent the time of exposure at center screen of the first and the second word. These signals are filtered at 7 Hz for purposes of visibility of illustration only.



group revealed only an Utterance Type \times Hemisphere interaction at the precentral, $F(1, 4) = 7.78$, $p = .049$, and temporoparietal, $F(1, 4) = 9.98$, $p = .034$, sites.

For the 1-sec interval immediately preceding the command digit onset, there was no significant hemispheric difference between utterance types within the DS group ($p = .06$ at PC3–PC4 and $p = .125$ at TP3–TP4), and there were no significant differences between the fluent utterances of the two groups, but the pattern of differences between DS and FS observed over the earlier interval still prevailed. The global Group \times Hemisphere effect, $F(2, 7) = 18.39$, $p = .0016$, was analyzed spatially into no effect at iF3–iF4 ($p = .84$) but significant Group \times Hemisphere effects at precentral, $F(2, 7) = 7.46$, $p = .018$, and temporo-parietal, $F(2, 7) = 5.91$, $p = .031$, sites. Descriptive simple effects still showed no group difference at PC3, PC4, TP3, or TP4 ($p > .15$), but hemisphere effects were obtained within the FS group at both PC3–PC4, $F(1, 4) = 11.89$, $p = .026$, and TP3–TP4, $F(1, 4) = 8.11$, $p = .046$, whereas the same comparisons within the DS (dysfluent samples), seen in the opposite

directions in Figure 1, respectively, attained only $p = .100$ and $p = .065$.

Analysis of electrical signal amplitudes for twenty-five 2 Hz-wide bands from 0 to 50 Hz (spectral and coherence analyses) on the -4.8 to -1.0 time period relative to command digit onset was completed. Analyses of the EMG signals during the 500 msec prior to speech onset and during the 500 msec prior to utterance (spatiotemporal, spectral, and coherence) were also completed. These measurements yielded a few significant but not particularly meaningful differences between groups (without adjustment for number of tests). Also, there was no difference between stuttered and non stuttered trials in the DS.

DISCUSSION

The results of this study are in harmony with the recent literature on central nervous system correlates of dysfluent speech. In addition, a new *electrophysiological* aspect of left hemisphere underactivation and right hemisphere overactivation has been shown to

relate *specifically* to dysfluent speech, namely, hemispheric asymmetry of CNVs. Additional EEG and EMG analysis did not yield any markers useful for neurotherapy for stutterers.

When visually processing verbal material (with contingent speech still several seconds off), prior to a dysfluent speech segment, the brain of the dysfluent participants appeared globally undermobilized, especially in left buccofacial motor cortex, an area that is *not* the most specialized for that particular cognitive activity in that time frame but that is nevertheless significantly involved, and in the left posterior speech cortex. At the same time, overactivation of these same areas occurred in the right hemisphere of the DS group several seconds prior to dysfluent speech but was not observed during the same interval prior to a normally spoken segment. This effect, CNVs with reversed asymmetry, occurred *before* specific response selection per se—and yet significantly and specifically predicted dysfluent speech. It occurred as the speaker was processing information later necessary, but not yet motorically relevant, to produce the designated utterance. Although the Utterance Type \times Hemisphere interaction on which the latter conclusion is based was only marginally significant ($p = .049$ with no correction for number of tests to balance the limited power afforded by the small group sizes), we believe that it deserves credit because the trials leading to fluent utterances in the DS produced ERPs in between those leading to dysfluent speech.

The electrophysiological-treatment approach most likely to produce significant clinical benefits, for DSs such as these, might consist of setting up a feedback signal based on (a) negativity during expectancy of an informative stimulus critical for a decision and (b) asymmetry of that signal at precentral and/or temporoparietal leads. Self-regulation of the CNV has been successfully obtained in depression (Schneider, Heimann, Mattes, Lutzenberger, & Bubaumer, 1992). Configuration of EEG feedback of hemispheric asymmetry has also been implemented successfully to alleviate symptoms of depression (Hammond, 2005). Hardman et al. (1997) studied whether they could train a large group of normal participants to

modify CNV laterality with neurofeedback. Using central sites (C3–C4: close to the one most asymmetric in the stutterers of our study), they were able to induce significant change in 50% of the sample ($N = 32$ participants). They also noted that this approach requires control of slow horizontal eye movements, which can contribute noise. They further noted that subjective ratings of more fatigue predicted less success at controlling DC asymmetry. A neurofeedback approach to stuttering should no doubt be accompanied by highly targeted training of focused attention related to processing of language in addition to various other techniques used by speech therapists.

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