ADHD and Stuttering: Similar EEG Profiles Suggest Neurotherapy as an Adjunct to Traditional Speech Therapies

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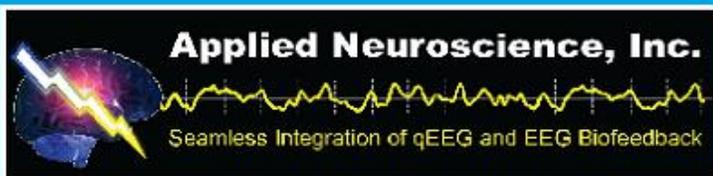Published online: 08 Sep 2008.

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SCIENTIFIC ARTICLES

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ABSTRACT. *Background*. This study investigated differences in theta and alpha activity measured by electroencephalography (EEG) at frontal sites between stutterers and nonstutterers during focused attention tasks.

Methods. EEG was recorded from 22 male, right-handed developmental stutterers and 22 male, age- and handedness-matched nonstutterers in six conditions: baseline resting-eyes-open; baseline resting-eyes-closed; eyes-open focused attention; eyes-closed focused attention; eyes-closed backwards-counting math task; and eyes-open auditory delayed non-match-to-sample task.

Results. Significantly more theta was recorded at frontal sites (FP1/2, F3/4, F7/8 and FZ) in each condition for stutterers than for nonstutterers. Significantly lower alpha (8-10 Hz) was recorded at these sites in stutterers than nonstutterers in all conditions. No hemisphere effects were found for either group.

Conclusion. The finding of more theta and low alpha activity in stutterers lends empirical support to an attentional component of stuttering. There are strong similarities in the EEG, morphology, and behavior of stutterers and individuals with attention-deficit/hyperactivity disorder (ADHD). These similarities suggest that neurofeedback, which has proven successful in the treatment of ADHD, may hold promise as a viable adjunct treatment to traditional speech therapies for stuttering.

KEYWORDS. Stuttering, attention deficit/hyperactivity disorder (ADHD), EEG, neurotherapy, neurofeedback, theta, frontal lobes

INTRODUCTION

Developmental stuttering is a heritable speech motor control disorder that affects over one percent of the population worldwide (MacFarlane, Hanson, Walton, & Mellon, 1991). Onset is in early childhood, typically with the onset of speech, and roughly 85 percent of developmental stutterers "outgrow" the disorder in late adolescence. Commonly held perceptions of stuttering often are limited to those portrayed in cartoon characters without regard to the havoc the disorder wreaks among those who are afflicted.

The physical discomforts alone can be painful and exhausting. In some instances, stutters' jaws come together with such excessive force and velocity that teeth are cracked, crowns are broken, and tongues and cheeks are bitten. Distorted tongue movements that extend outside the mouth may roughen and crack the lips. Some stutterers develop calluses, polyps and thickening of the vocal folds from years of straining to initiate voicing. Excessive air pressure and intense muscle spasms sometimes extend into the extremities.

The capacity for effective verbal communication is important for successful social development, interpersonal relationships and academic or occupational pursuits. Stutterers report difficulties in all the above, often related to the fact that they "look" normal and are therefore

expected to be able to communicate normally (Franken, Boves, Peters, & Webster, 1991). Severe stutterers (those ranging from 23% to as much as 80% disfluent; Webster, 1980b) can be nearly unintelligible, and those with severe silent blocks are often not able to initiate voicing at all. Many report being labeled retarded, learning disabled, uncooperative or antisocial. Stutterers score higher on measures of irrational attitudes and expectancies (Perceptions of Stuttering Inventory; Woolf, 1967). They often report interpersonal relationship problems, loss of jobs or promotions, and avoidance of higher education opportunities. It is an expensive problem, both in human and economic terms.

There is no cure for stuttering, and the disorder is notoriously resistant to treatment. Many speech-language pathologists report that stuttering is one of the most difficult speech disorders to treat and often goes unresolved (Andrews, Guitar, & Howie, 1980). Behavioral treatments have variable efficacy and serve to provide more or less reliable compensatory strategies that allow the stutterer to, in effect, "manually" produce fluent speech (Karniol, 1995; Webster, 1991). Physiological treatments are completely unreliable and in some instances dangerous. Peripheral equipment, which manipulates auditory feedback, is cumbersome and idiosyncratic (Webster, 1991). Traditional biofeedback applications for relaxation have had limited efficacy in that they serve to reduce the physical arousal associated with anxiety states, allowing for more range of muscle movement and state-dependent increases in fluency. However, a number of non-traditional biofeedback applications within the framework of specific behavioral therapies have been very useful in the acquisition of compensatory motor skills (Blood, 1995; Webster, 1980a). The greatest challenge to even the most successful stuttering therapy is the maintenance of the skills post-treatment. Stuttering is extremely susceptible both to behavioral drift toward pre-therapy levels of disfluency and to variability in cognitive or environmental load (Neilson & Neilson, 1991). The stutterer must work relentlessly to overlearn and maintain skills to achieve any degree of success.

To date, there appear to be no published accounts of applications of neurofeedback to stuttering. The present study serendipitously encountered evidence that suggests that neurofeedback may be useful as an adjunct to traditional speech therapies for stuttering.

The hypothesis tested in this study was that more theta activity (3-7.5 Hz) would be recorded at frontal sites (FP1/2, F3/4, F7/8 and FZ) in stutterers than nonstutterers during focused attention tasks. This conjecture was based upon the delayed auditory feedback hypothesis of stut-

tering (Stromsta, 1959, 1967, 1972, 1986), which has been the subject of intense investigation and which suggests that at least one etiology of stuttering may lie in the fact that two sources of auditory feedback which are received simultaneously in fluent speakers are separated in stutterers.

One of the key features that make developmental stuttering clinically distinguishable from acquired stuttering is its responsivity to manipulation of air-conducted and bone-conducted voice. When received simultaneously, these two sources of auditory feedback create a reliable feedback loop for the assembly and execution of fluent speech (Rosenfield & Jerger, 1984). Fluency can be induced in stutterers and disfluency in nonstutterers by altering either of those two sources, such as by whispering, singing, choral reading, or metronomic speech (Andrews et al., 1983). Fluency can also be induced by masking the stutterer's auditory feedback with white noise between 1000-3000 Hz at 50 decibels or above (Dewar, 1984).

Stutterers become more fluent when their returning auditory feedback is delayed by means of a recording and reproducing device (Webster, 1991, 1992). In nonstuttering adults, who normally receive both sources of voice simultaneously, repetition errors increase as a function of delay up to about 200 milliseconds (ms) and then decrease with longer delays but never disappear even with delays as long as 800 ms (MacKay, 1968; MacKay & MacDonald, 1984). In stutterers, delays as short as 10-20 ms to about 50 ms are optimal for inducing fluency (Webster, 1992). Separation of returning auditory feedback is presumably a factor in the breakdown of speech motor plan assembly in the stutterer, producing errors in the timing, sequencing, velocity, and trajectory of orofacial, laryngeal, and respiratory musculature (Kent, 1990).

Speech motor plan assembly occurs at the level of the syllable and requires successful attention only to aspects of feedback from an executed syllable that are relevant to the planning and construction of the imminent syllable (Levelt, 1983). Accurate plan assembly also requires successful inhibition of attention to irrelevant or erroneous competing feedback (Gracco & Abbs, 1986). As part of the supplemental motor area (SMA) basal ganglia/premotor-cerebellar system, the frontal cortex is involved in the temporal organization of behavior and the temporal control of movement. The prefrontal cortex is also implicated in the direction and maintenance of focused attention, which involves a strong inhibitory component (Fuster, 1989).

Based upon evidence of the separation of two auditory feedback sources in the stutterer, the present study hypothesized that stutterers

may be parsing potentially double the amount of auditory feedback during on-going speech production. Indeed, Fox et al. (1996), using positron emission tomography, found hyperactivation of the motor cortex (a system involving the superior lateral premotor cortex, SMA, insula and cerebellum) in stutterers during stuttered reading.

Carrying this idea one step further, potentially double the amount of feedback would require much more effort toward suppression or inhibition of attention to competing stimuli. Inhibition of attention has been associated with EEG theta activity (3-7.5 Hz), particularly in the frontal midline area (Bruneau, Roux, Guerin, Garreau, & Lelord, 1993; for reviews, see Crawford, 1994, and Crawford & Gruzelier, 1992). Pennekamp, Bosel, Mecklinger, and Ott (1994) hypothesized that the occurrence of theta is thought to reflect attentional processing responsible for correct detection of rare targets against standard targets in a sustained attention task. Since each speech syllable is unique in its plan assembly (Gracco & Abbs, 1986; Kent, 1990), each syllable represents a rare target in an overabundance of standard targets for the stutterer. Therefore, high levels of frontal slow wave (theta) activity associated with inhibitory processing may be a corollary to the hyperactivation of the fronto-cerebellar system found by Fox et al. (1996).

A second hypothesis in the present study was that stutterers would also exhibit more frontal alpha than nonstutterers in focused attention tasks. In 1977, Schacter reported that in contrast to classical alpha blocking response during sensory stimulation in alert wakefulness, sensory stimulation in the presence of low voltage theta induces alpha. Furthermore, Klimesch, Schimke, and Schwaiger (1994) investigated a relationship between theta and alpha activity based upon factor analytic variation of the upper and lower alpha bands in opposite directions. Klimesch and colleagues suspected that the lower alpha band showed more similarity to theta than to the upper alpha band. "Thalpha," 6-10 Hz activity, is targeted for decrease in neurofeedback treatment of attention deficit/hyperactivity disorder (ADD/ADHD) (Lubar & Lubar, 1999), another syndrome wherein the subject is presumed to ineffectively inhibit attention to competing stimuli.

ADHD children show neuropsychological deficits that suggest specific problems with inhibitory control of attentional selection (Williams, Stott, Goodyer, & Sahakian, 2000). They experience difficulty inhibiting motor behavior and inhibiting attentional focus on distracting or irrelevant stimuli (Barabasz & Barabasz, 1995), all executive function deficits which implicate the basal ganglia, ventro-lateral prefrontal cortical areas, frontostriatal functional loops and the premotor-cerebel-

lar system (Williams, Stott, Goodyer, & Sahakian, 2000; Castellanos, 1997). EEG measures reliably find significantly more slow wave (theta) activity in the frontal regions of those with ADHD (Barabasz & Barabasz, 1995), and some subtypes of ADHD show frontal hyperactivation (Lubar & Lubar, 1999; Chabot, Merkin, Wood, Davenport, & Serfontein, 1996), a similar EEG profile as that found in the following results among stutterers.

METHODS

Participants

Participants were 22 male, right-handed (as assessed by the Benton Handedness Questionnaire; Benton, 1959) developmental stutterers, mean age 28.2, and 22 age- and handedness-matched male nonstutterers. All participants were assessed by licensed, clinical speech-language pathologists and behavioral psychologists to determine the presence or absence of any speech disorders, to determine that all stuttering identified was developmental in origin, and to determine stuttering severity level. For purposes of this study, stuttering, or a disfluent utterance, is defined as any hesitation, prolongation or repetition occurring either at the beginning or within a syllable. Stuttering severity was determined on both syllable and word bases during both spontaneous conversation and reading. Severity ranged from 11% to 59% disfluent utterances (Webster, 1980a, 1980b), mean = 28.55%, median = 27.15%. All participants were screened for physical and psychiatric conditions, head injury, and medications. Participants were medication-free and had not ingested alcohol for 24 hours or caffeine for four hours prior to testing.

Experimental Procedures

All participants completed appropriate informed consent and screening forms. Sessions were counterbalanced for both groups across morning (10:00 a.m.-12:00 noon), afternoon (2:00 p.m.-4:00 p.m.), and evening (6:00 p.m.-8:00 p.m.) to control for diurnal effects. EEG recording was carried out in a radio-shielded, sound-attenuated room using a Lexicor Medical Technologies NeuroSearch-24 system. An ECI International Electro-cap was used to collect monopolar recordings from 19 sites in accordance with the international 10-20 system of electrode placement, referenced to linked earlobes and grounded just ante-

rior to FZ. Impedance readings were below 5 K ohms for each site for each participant before recording. Electromyographic (EMG) data from eye movements were monitored from electrodes placed inferior and lateral to the left outer canthus. EEG was monitored for movement and muscle artifact prior to each testing sequence so that participants could be assisted in relaxing their muscles and controlling eye movement prior to recording.

EEG was recorded in a baseline resting-eyes-open state and in a baseline resting-eyes-closed state for a minimum of three minutes or until at least 60 seconds of artifact-free data were obtained. Participants then completed an eyes-closed, backwards-counting math task based upon the "serial sevens" task in the Mini-Mental Status Examination (Folstein, Folstein, & McHugh, 1975). EEG was recorded in this condition for a minimum of five minutes or until 120 seconds of artifact-free data were obtained.

Participants then completed an eyes-open, auditory delayed-match to sample task (Milner, 1963; Milner, Petrides, & Smith, 1985). EEG was recorded for a minimum of six minutes or until 180 seconds of artifact-free data were obtained. The task consisted of a series of randomly generated syllables, each syllable approximately 25 ms in length, with a mean interstimulus interval of 75 ms. Syllables, presentation order of syllables, and interstimulus intervals were randomly generated by computer program. A randomly selected fifteen percent of generated syllables were repeated immediately after initial presentation (Sakurai, 1992; Lu, Williamson, & Kaufman, 1992). Participants were instructed to listen carefully to the sequence of syllables and press a computer key whenever they heard a syllable that was the same as the one before it. The program recorded the number of key presses made by each participant, both correct and incorrect, for later analysis.

EEG was then recorded during eyes-open and eyes-closed intervals in which participants were given focused attention instructions (Ishihara & Yoshii, 1973; Mizuki, Tanaka, Isogaki, Nishijima, & Inanaga, 1980). In the eyes-open condition, they were instructed to focus on one aspect of an abstract painting; in the eyes-closed condition, they were instructed to think about a pleasant trip they had taken. EEG was recorded in this condition for a minimum of three minutes or until 60 seconds of artifact-free data were obtained.

EEG was filtered by anti-aliasing filters (high pass 2 Hz, low pass 64 Hz), with a cut-off frequency of 256 samples per second (gain setting 32 K and a 60 Hz notch filter) and used an analog/digital converter coupled to a 486DX computer.

EEG analog records were visually reviewed in a systematic removal of artifact. One-second epochs containing eye blinks, movement or muscle artifacts were deleted from analysis. A fast Fourier transform (analysis mode window, Bechman Harris; .5 Hz resolution) was used to obtain peak-to-peak spectral magnitude plots for artifact-free data.

Dependent measures were mean spectral magnitude in microvolts for theta (3-7.5 Hz), low theta (3-5 Hz) and high theta (5.5-7.5 Hz) (Crawford & Barabasz, 1996; Vogel, Broverman, & Klaiber, 1968, cited in Schacter, 1977), as well as alpha (8-13 Hz), low alpha (8-10 Hz) and high alpha (10.5-13 Hz) (Coppola & Chassey, 1984). Pooled log mean spectral magnitudes (Sterman, Mann, Kaiser, & Suyenobu, 1994) from FP1/2, F3/4, F7/8 and FZ were subject to mixed design analysis of variance (ANOVA) conducted separately for each condition. Data from all nineteen sites were analyzed for post hoc analysis of thirteen frequency bands from 2 to 42 Hz.

RESULTS

Theta

EEG recorded significantly greater mean magnitude frontal theta (3-7.5 Hz recorded at FP1/2, F3/4, F7/8, FZ) in stutterers than in nonstutterers in all conditions: Baseline Resting Eyes Open, $F(1,42) = 9.14$, $p < .01$; Baseline Resting Eyes Closed, $F(1,42) = 4.87$, $p < .05$; Eyes Open Focused Attention, $F(1,42) = 9.21$, $p < .01$; Eyes Closed Focused Attention, $F(1,42) = 7.11$, $p = .01$; Backwards Counting, $F(1,42) = 6.8$, $p = .01$; Auditory Delayed Match to Sample, $F(1,42) = 7.58$, $p < .01$. No hemisphere effects were found (FZ was eliminated from analyses for hemisphere and site effects). A significant main effect for site was found in all conditions such that greater mean magnitude theta was recorded at F3/4 than at FP1/2 and F7/8, respectively. Significant main effects for group and area were observed in both the low theta (3.5 Hz) and high theta (5.5-7.5 Hz) sub-bands. Post hoc analysis of all 19 sites indicated significantly more theta in stutterers in temporal and central regions only in the resting eyes open condition ($F(1,42) = 6.88$, $p < .05$ and $F(1,42) = 5.35$, $p < .05$, respectively) (see Table 1).

Note that the variability among stutterers was greater than that among nonstutterers. There were no strong outliers to account for this variability, but several factors may be at work. The first is the complete idiosyncrasy of the disorder itself. The single consistency of stuttering, in this investigator's clinical observation of over 500 stut-

TABLE 1. Mean Magnitude in Microvolts of Theta (3-7.5 Hz) Recorded at Frontal Sites of Stutterers and Nonstutterers

	Group			
	Stutterers		Nonstutterers	
Condition	Mean	SD	Mean	SD
Resting Eyes Closed	3.62	1.50	2.80	.82
Resting Eyes Open	3.46	1.54	2.50	.68
Backwards Counting	3.77	1.38	2.91	.91
Delayed Auditory Match	3.65	1.14	2.88	.93
Eyes Closed Focused Attn.	3.65	1.41	2.81	.95
Eyes Open Focused Attn.	3.46	1.44	2.54	.71

terers, is its inconsistency both within and among individuals and its reliance upon minute changes in contextual variables. Secondly, three of the stutterers in this sample were clinically evaluated as severe stutterers (23 percent disfluent or above; Webster, 1980b), while the remaining 19 were evaluated as mild/moderate in severity. There is some discussion in the literature that very severe stuttering may be reflective of a different underlying etiology than mild/moderate stuttering (Boehmler & Boehmler, 1989). Finally, the stutterers in this sample, as is the case with most stutterers, had experienced different types and amounts of therapy in their lives, each of which, if even marginally successful in instigating behavioral change, can be presumed to contribute to neurological change as well.

Alpha

EEG recorded significantly greater mean magnitude frontal low alpha (8-10 Hz) in stutterers than in nonstutterers in all conditions: Baseline Resting Eyes Open, $F(1,42) = 9.81$, $p < .01$; Baseline Resting Eyes Closed, $F(1,42) = 4.07$, $p = .05$; Eyes Open Focused Attention, $F(1,42) = 10.45$, $p < .01$; Eyes Closed Focused Attention, $F(1,42) = 7.28$, $p = .01$; Backwards Counting, $F(1,42) = 7.86$, $p < .01$; Auditory Delayed Match to Sample, $F(1,42) = 6.61$, $p = .01$. As in the theta band, a significant main effect for site was found in all conditions such that greater mean magnitude low alpha was recorded at F3/4 than at FP1/2 and F7/8, respectively. No hemisphere effects were found (see Table 2).

TABLE 2. Mean Magnitude in Microvolts of Low Alpha (8-10 Hz) Recorded at Frontal Sites of Stutterers and Nonstutterers

| Condition | Group | | | |
| | Stutterers | | Nonstutterers | |
	Mean	SD	Mean	SD
Resting Eyes Closed	2.34	1.31	1.66	.95
Resting Eyes Open	1.95	.81	1.33	.76
Backwards Counting	2.31	1.04	1.55	.83
Delayed Auditory Match	2.26	.98	1.74	1.01
Eyes Closed Focused Attn.	2.25	1.05	1.59	.94
Eyes Open Focused Attn.	1.98	.83	1.30	.67

Significantly higher alpha (10.5-13 Hz) was recorded in stutterers only in the Eyes Open Focused Attention condition ($F(1,42) = 5.68$, $p < .05$). Significantly more total alpha (8-13 Hz) was recorded in stutterers only in the Eyes Closed Focused Attention condition ($F(1,42) = 6.13$, $p < .05$.

Post Hoc Analysis of Beta Bands

To summarize, significantly more beta activity between 16-24.5 Hz and at 40 Hz was recorded in stutterers in frontal, temporal, central and parietal regions in all conditions. These results are relevant to this discussion because they are consistent with Fox et al.'s (1996) PET findings. A caveat in discussing these results is the finding that stutterers have demonstrated higher EMG activity than nonstutterers (Caruso, 1991). While no conclusion can be drawn here, further investigation should be undertaken since activity in these frequency bands has been shown to increase in high-sustained attention subjects (Crawford, Clarke, & Kitner-Triolo, 1996) and during vigilant behavior in animals (Sterman & Bowersox, 1981) and humans (Makeig & Inlow, 1993).

DISCUSSION

The results of the present study support the hypothesis that more frontal slow wave activity (both theta and low alpha) can be seen in stut-

terers than nonstutterers during tasks that require selective inhibition of attention to competing stimuli. Further, the stutterers in the present study show hyperactivation of cortical areas akin to Fox et al.'s (1996) findings. By themselves, these data appear supportive of the contention that some pathology separates auditory information in the stutterer, contributing perhaps to overload or even interference effects during speech motor plan assembly. Extra auditory feedback may be reflected in greater output by thalamic alpha generators, forcing hippocampal theta "gating" mechanisms to work overtime to parse relevant inputs (O'Keefe, 1986).

Surprisingly, however, stutterers' EEG showed the same patterns of activation during baseline conditions as well. This finding suggests a pathological process at work well before plan assembly begins–one not simply instigated by an aberration in the plan assembly process itself. The practical implications of the findings in the present study lie in their similarity to EEG seen in the ADHD population.

An informal survey completed by this investigator of 700 stutterers' therapy applications to the Hollins Communications Research Institute revealed that seven percent of those applicants had an identified diagnosis of ADHD or ADHD/LD combined (only one of these was an adult). Clinical staff members' anecdotal impression is that the prevalence may be somewhat higher given what they suspect is a high number of undiagnosed cases, both in children and adults. ADHD is, like stuttering, a heterogeneous condition and is frequently comorbid with other conditions. However, estimates of the prevalence of ADHD in school age children ranges from two percent to 18 percent (Castellanos, 1997), so the prevalence of ADHD in the stuttering population does not appear to be different from the population at large.

Chabot, Merkin, Wood, Davenport, and Serfontein (1996) found that the quantitative EEG (QEEG) of ADD/ADHD children was distinguished by excess theta relative power, especially in frontal regions. They identified two neurophysiological subtypes of ADD/ADHD children, the second of which showed greater anterior QEEG abnormality and was characterized by increased theta and/or alpha with normal alpha mean frequency. The authors suggest this pattern is indicative of increased cortical metabolic activity (as in Fox et al., 1996) and/or either increased thalamic alpha generator output or a disinhibition of hippocampal theta generators (Steriade, Gloor, Llinas, Lopes da Silva, & Mesulam, 1990), a perspective similar to that discussed above in relation to stuttering.

Amen, Paldi, and Thisted (1993) identified five ADD/ADHD subtypes by neurological profile and behavior. Type 4 is similar to stutterers and to Chabot Type 2 and showed increased frontal activity, particularly in the gyrus rectus, and hypermetabolism in the anterior cingulate gyrus, which projects to orbital-frontal cortex. Lubar and Lubar (1999) describe these individuals' attention deficit as " . . . an inability to shift attention and excessive overattending to irrelevant details" (p. 109).

Mann, Lubar, Zimmerman, Miller, and Muenchen (1992) found results similar to the present study in ADHD boys during reading and drawing tasks. During reading, there was a significant increase in frontal absolute theta amplitude. During a drawing task, there were generalized frontal and central theta increases as well as increases in frontal alpha. Note that, as in the present study, excess theta was particularly evident at F3/F4.

In addition to QEEG, stutterers and ADD/ADHD individuals share other physiological characteristics. Lubar, Gross, Shively, and Mann (1990) found a decreased P3 component and poorly developed late components, particularly at FZ, in an ADHD-LD group as compared to controls and gifted children. Pribram (1991) describes late components as contingent upon both the physical characteristics of the stimulus and whether or not the subject is paying attention to the stimulus. Finitzo, Pool, Freeman, Devous, and Watson (1991) found differences in late components in stutterers as compared to nonstutterers, most notably in lower amplitudes of P2 over mesial frontal cortex (F3, FZ, F4). Newman, Bunderson, and Brey (1985) and Blood and Blood (1984) found longer latencies of the N1 deflection in stutterers. N1 is thought to be involved primarily in selective attention (Lubar, 1991).

Cerebral blood flow studies have differentiated stutterers from nonstutterers. Hypoperfusion has been found in stutterers in areas including the anterior cingulate and inferior frontal gyri (Finitzo, Pool, Freeman, Devous, & Watson, 1991). Decreased blood flow has also been found in ADHD subjects in the striatum, in prefrontal regions, and in the right caudate (Castellanos, 1997).

Stutterers and ADHD subjects share characteristics in cognitive/behavioral measures as well. Lubar, Gross, Shively, and Mann (1990) found that their ADHD-LD group made more errors of commission to a target stimulus in the auditory evoked potential paradigm than did controls or gifted children. The continuous performance test (CPT) has been used to demonstrate deficits in the ability of those with ADHD to respond appropriately to a rare stimulus (Castellanos, 1997). In the present study, stutterers made significantly more false positive errors to

an auditory stimulus during a delayed-match-to-sample task (F(1,42) = 30.83, p < .0001). ADHD adults can be differentiated from adults without ADHD on neuropsychological measures sensitive to frontal lobe executive function (Lovejoy et al., 1999) similar to those used in the present study (digit span, serial sevens, match to sample).

Finally, with relation to the increased alpha observed in stutterers, Lubar (1991) reports that he has found many ADHD children who show persistence of alpha during cognitive tasks as opposed to normal alpha suppression.

Based upon the findings presented above and those of the present study, there is a legitimate basis for comparing the neural activity of stutterers with that of ADD/ADHD individuals. If a treatment is demonstrated to alleviate the symptoms of one disorder, it is reasonable to predict that it may be effective in treating the other disorder.

Neurofeedback can be an effective treatment for individuals with ADHD. Kaiser and Othmer (2000) found that neurofeedback produced significant improvement in TOVA performance, a continuous Go/No-Go task much like the auditory match-to-sample in the present study. Children appear to particularly benefit from training to reduce excessive theta (Lubar, Swartwood, Swartwood, & O'Donnell, 1995). Adults and older teenagers appear to benefit from reduction of activity between 6 and 10 Hz (high theta/low alpha or "thalpha"), as well as increasing the amplitude and duration of beta activity (Lubar & Lubar, 1999; Lubar, Swartwood, Swartwood, & O'Donnell, 1995).

Reduction of 6-10 Hz activity may be the same protocol that could be effective for stutterers. Recall that roughly 85 percent of stutterers "outgrow" the disorder by mid- to late adolescence. The remaining 15 percent who do not develop a spontaneous recovery are the targets for long-term treatment and would be in the older teen/adult age group described above. In addition to reduction of theta and low alpha, increases in high alpha, which has been associated with efficient cognitive processing (Crawford, Knebel, Vendemia, Kaplan, & Ratcliff-Baird, 1995), may be appropriate. Increases in the amplitude and duration of beta, as described above (Lubar & Lubar, 1999; Lubar, Swartwood, Swartwood, & O'Donnell, 1995) may be appropriate in light of Crawford, Clarke, and Kitner-Triolo's (1996) findings of increased beta in high-sustained attention individuals during vigilant behavior. Care must be taken, however, with increasing beta activity in stutterers, particularly in the upper ranges of the frequency band. Beta can be associated with increases in EMG activity, and high levels of tension in the speech musculature plague stutterers.

The success of neurofeedback with the ADHD population should be encouraging to stutterers. As Lubar and Lubar (1999) state, "the point of view in working with this disorder [ADHD] using neurofeedback is that if we can change the underlying neurology, perhaps we can effect a more long-term change in this disorder" (p. 106). The same can be said for stuttering.

The most promising implication for the present findings is the combination of neurofeedback with behavioral, biofeedback-based therapies or traditional speech therapies for stuttering. The activation patterns that appear to be inherent in stutterers, whether cause or result, may be an obstacle to the success of the long-term behavioral changes that are necessary to create and maintain fluent speech. The neural physiology of the stutterer may be the culprit in the seemingly inevitable behavioral drift toward disfluency.

The question arises whether quantitative EEG may ever be used as a diagnosis for stuttering. This investigator considers that unlikely, in part because the disorder is most commonly defined by the presence of behavioral anomalies (e.g., disfluencies) and in part because, despite efforts to develop empirical measures of what constitutes a disfluency (Webster, 1991), the speech/language community continues its long-time struggle to reach agreement on exactly how the disorder should be defined and diagnosed (Van Riper & Erickson, 1996).

A follow-up study underway looks at frontal EEG activation in stutterers before and after an intensive, 19-day behavioral, biofeedback-based therapy program. Future directions will combine neurofeedback treatments with behavioral therapy to see if the clinical efficacy of the therapy is enhanced. Perhaps most significant to the stutterer, neurofeedback training will ultimately be administered longitudinally post-therapy and compared with controls to see if long-term maintenance of fluency skills is enhanced.

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