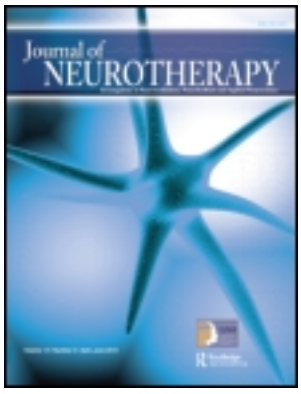

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LENS NEUROFEEDBACK TREATMENT WITH FETAL ALCOHOL SPECTRUM DISORDER AND NEGLECT

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Fetal alcohol spectrum disorders (FASD) stem from prenatal exposure of the fetus to alcohol. Resulting problems vary on a continuum of severity but commonly involve structural and functional brain abnormalities resulting in problems with academic performance, ADD/ADHD, information processing, math performance, memory, social skills, and increased rates of psychopathology, all of which generally persist into adulthood. This article presents the first uncontrolled case report of neurofeedback treatment of an FASD case on the milder end of this spectrum, which resulted in significant behavioral and academic improvements that were sustained on follow-up at 42 months. It is possible that neurofeedback may hold potential to improve functioning in persons with FASD.

INTRODUCTION

Since fetal alcohol syndrome (FAS) was first described (Jones & Smith, 1973), more than 3,500 papers have been published demonstrating that alcohol has devastating effects on the developing fetus, with the most profound effects being on brain development. In the past 40 years it has come to be recognized that there are varying degrees of severity of fetal alcohol effects depending on the dosage, resulting in the more recent term fetal alcohol spectrum disorders (FASD; Bertrand, Floyd, & Weber, 2005). FASD has been estimated to occur in between 0.2 to 1.5 cases per 1,000 live births (Centers for Disease Control and Prevention, 2002) and as many as 2% to 5% of school-aged children in the United States and Western Europe (May et al., 2009). Annual costs of FAS have been estimated to be from $3.6 billion (Lupton, Burd, & Harwood, 2004) to $5.3 billion, with an annual cost per person of $21.642 (Stade et al., 2009) and the cost of lifetime care estimated at $2 million (Lupton et al., 2004).

Event-related potential EEG studies (Burden et al., 2009; Burden et al., 2010; Burden, Jacobson, & Jacobson, 2005) have found somewhat similar deficits in persons with prenatal alcohol exposure, but with some differences, compared with persons with attention deficit hyperactivity disorder (ADHD). A review of studies of FASD (D'Angiulli, Grunau, Maggi, & Herdman, 2006) concluded that EEG provides evidence of neurophysiological abnormalities reflecting cognitive and sensory impairments. As may be expected, many children with FASD have comorbid ADHD (Fryer, McGee, Matt, Riley, & Mattson, 2007; Rasmussen et al., 2010), although careful analysis finds some differences from an ADHD population (Mattson, Crocker, & Nguyen, 2011). Prenatal alcohol exposure in the first trimester of pregnancy has been found to be significantly associated with an increased rate of conduct disorder (Disney, Iacono, McGue, Tully, & Legrand, 2008; D'Onofrio et al., 2007; Larkby, Goldschmidt, Hanusa, & Day, 2011), and behavior problems with FASD do persist into adulthood

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(Barr et al., 2006; Famy, Streissguth, & Unis, 1998; Spohr, Willms, & Steinhausen, 2007).

Diffusion Tensor imaging studies (Wozniak & Muetzel, 2011) have documented abnormalities in FASD children in the corpus callosum, major anterior-posterior fiber bundles, corticospinal tracts, and the cerebellum, which correlate with neurocognitive deficits, particularly in processing speed, nonverbal abilities, and executive functioning. Neuroimaging studies (Archibald et al., 2001; Lebel et al., 2008; Norman, Crocker, Mattson, & Riley, 2009; Sowell et al. 2001) have found global decreases in brain volume in FASD individuals, with particularly great reductions in the parietal area, but also in the caudate and cerebellum. A review by Coles and Zhihao (2011) concluded that ''recent fMRI studies have shown that besides functional activations, attention-demanding tasks usually also induce deactivation in several brain regions termed as the ''default mode network'' (p. 130).

Neuropsychological testing has been able to establish a profile associated with FASD (Mattson et al., 2011; Mattson et al., 2010). FASD children and adults have been found to commonly have deficits in IQ (Mattson & Riley, 1998), attention and executive control (e.g., planning and problem-solving ability, impulse control; Brown et al., 1991; Coles et al., 1997; Connor, Streissguth, Sampson, Bookstein, & Barr, 1999), speed of information processing (Burden et al., 2005; Jacobson, 1998), mathematics and number processing (Goldschmidt, Richardson, Stoffer, Geva, & Day, 1996; Mattson, Riley, Gramling, Delis, & Jones, 1998), memory (Coles, Lynch, Kable, Johnson, & Goldstein, 2010; Uecker & Nadel, 1996), and social information processing (McGee, Bjorkquist, Price, Mattson, & Riley, 2009).

Treatment interventions for FASD have focused on improving cognitive (e.g., memory, math) or adaptive skills (e.g., self-regulation, social skills), parenting training, or drug treatment (Kodituwakku & Kodituwakku, 2011). This article presents the first published case of the use of neurofeedback in the treatment of an FASD child.

CASE DESCRIPTION

Micah was a 10-year-old boy who was just entering fifth grade and who was born with FASD. His mother was described as ''brilliant'' and was a doctoral student at a prestigious Ivy League university, but she had used alcohol during her pregnancy. After giving birth his mother began more heavily using alcohol and began abusing drugs as well, and dropped out of the university, and at 10 months of age Micah was removed from the home for neglect and failure to thrive. He remained in a foster home until he was 22 months old, at which time because his mother had made no effort to deal with her substance abuse and had been living as a ''street person,'' he was given up for adoption.

Micah did not display any of the craniofacial abnormalities that characterize more serious cases of FAS, and he was obtaining C grades. The combination of these two factors suggests that Micah was on the milder end of the FASD continuum, although his mother's presumably high IQ may have modulated to some degree the fetal alcohol effects on intelligence level. Nonetheless, he displayed serious behavior problems and met nine of nine *Diagnostic and Statistical Manual of Mental Disorders* (4th ed.; American Psychiatric Association, 1994) criteria for a diagnosis of attention deficit disorder and eight of nine criteria for the diagnosis of ADHD. On a 0-to-10 scale, where 0 represents no problem and 10 the most extreme problem, his adopted mother initially rated his symptoms as follows: Impulsiveness 9, Hyperactivity 8, Forgetfulness 8, Anger/Irritability 7, Poor Concentration 7, Poor Handwriting 7, and Being Disorganized 6. These symptom ratings were when Micah was taking medication (Adderall). He also had speech problems and had been in speech therapy.

He was evaluated with a QEEG with recording electrodes placed according to the 19 standard regions defined by the International 10/20 System of electrode placement, referenced to linked ears. The QEEG was recorded when he had been off Adderall for at least 2 days. All electrode impedance levels were below 5 KOhms, with no interelectrode

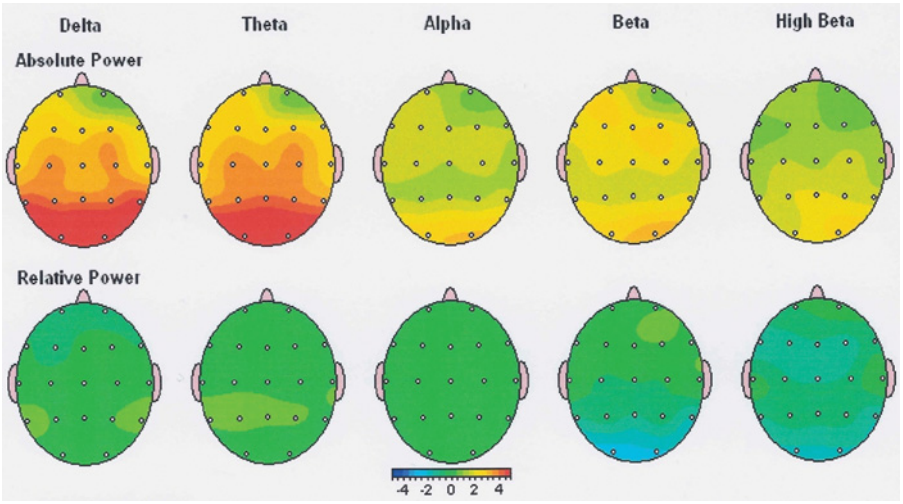

FIGURE 1. Pre-treatment quantitative EEG. (Color figure available online.)

differences of more than 500 ohms, and ear references that were balanced. The vigilance level was controlled by noting signs of drowsiness appearing in the EEG, and then pausing the recording and verbally interacting with the patient. A bipolar recording channel was used to monitor eye movement artifact. Fourteen minutes and 4 s of eyes-closed data were gathered, the recording was of adequate quality, and after editing to reduce artifact 3 min of data were analyzed using the NeuroGuide normative database. Split-half reliability was .98 and test–retest reliability was .97. Figure 1 displays a *Z*-scored FFT summary scaled to 5 *Z* scores. Deviations from norms were most excessive in posterior and central delta and theta. There was also hypocoherence present in these frequency bands in these areas, and hypercoherence in alpha throughout most of the head.

TREATMENT

The patient was treated using the Low Energy Neurofeedback System (LENS; Hammond, 2007; Ochs, 2006). This neurofeedback system provides feedback in the form of an exceptionally small electromagnetic field (field strength of 10^{-18} watts/cm^2), which was delivered in 1-s intervals down electrode wires while the patient sat with his eyes closed. The feedback signal was adjusted 16 times/second to remain five cycles/second faster than Micah's dominant brainwave frequency. After initially providing 1 s of feedback at all 19 standard electrode sites, treatment consisted of providing 1 s of feedback at six to seven electrode sites per session. The order in which sites were selected for feedback was based on a LENS map, proceeding systematically from sites where amplitude and variability was the least toward where they were greater.

Micah's initial mean symptom rating was 7.4. After seven LENS sessions the mean symptom rating from his mother had declined to 4. However, his mother did note one occasional side effect. After a session where feedback was given at parietal and occipital sites, where the amplitudes and variability was highest, he would become ''irritable and crabby'' for a couple of days, with more aggressiveness and impulsivity. After this had been observed on two occasions, we began reducing the number of seconds of feedback to 4 s when treatment included parietal or occipital electrode sites, and 5 to 6 s in other sessions. This resulted in less irritable reactivity. By the end of 19 sessions his dose of Adderall had been reduced by 50%, but nonetheless his mean symptom rating was 2.7.

After 26 LENS treatment sessions his mother began thinking about terminating treatment soon. Because of the author's observations that his raw EEG in posterior areas was still extremely

elevated, despite his symptomatic improvements, it was suggested that we consider adding some sessions of traditional neurofeedback in an effort to further reduce amplitudes. One session of traditional neurofeedback was done with electrodes at O1 and O2, inhibiting 3–9 Hz while mildly reinforcing 12–15 Hz. The same reactivity was observed for 3 days following that session, during which he was angry, would yell, and ''fly off the handle,'' and he was getting in trouble at school. His mean symptom rating on those 3 days was 4.1. Therefore, we returned to LENS training for five more sessions.

On 42-month follow-up Micah's mother gave a mean symptom rating of 2.9. She indicated that he had overcome his problems in expressing himself, and his previous year his grade point average was 3.86.

DISCUSSION

The FASD case that has been reported was one that was not on the severe end of the FASD continuum in that he did not display craniofacial deformities and he had average grades in school, perhaps because of genetic inheritance of a higher IQ from his mother. Nevertheless, a QEEG evaluation found extreme deviations from norms, and as Schonfeld, Mattson, and Riley's (2005) research has shown, half of nondysmorphic FASD children probably have conduct disorder. Clearly, Micah qualified for a diagnosis of ADHD.

This report is simply of an uncontrolled single case of FASD. Certainly there are much more severe cases of FASD. For example, fetal alcohol exposure has been considered an important cause of mental retardation (Abel & Sokol, 1987; Pulsifer, 1996), a condition once considered inappropriate for treatment with neurofeedback (Lubar, 1995) but where recent research (Surmeli & Ertem, 2007, 2010) has provided encouragement that neurofeedback can have a positive impact. The current case report holds out hope that FASD children, especially those who are nondysmorphic, may receive some benefit from neurofeedback. Controlled research will be essential.

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