QEEG-Guided Neurofeedback for Children with Histories of Abuse and Neglect: Neurodevelopmental Rationale and Pilot Study

Lark Huang-Storms, MS
Eugenia Bodenhamer-Davis, PhD
Richard Davis, MS
Janice Dunn, MA

ABSTRACT. *Background.* Poor self-regulation of arousal is central to the behavioral difficulties experienced by children with traumatic caretaker attachment histories. EEG biofeedback teaches children to self-regulate brain rhythmicity, which may in turn affect global improvements in the areas of attention, aggression, impulse control, and trust formation. Research literature reports successful use of neurofeedback for children with ADHD, autism, asthma, stroke, and migraine. This study extends current research by investigating the effectiveness of neurofeedback in reducing behavioral problems commonly observed in abused/neglected children.

Methods. Treatment records of twenty adopted children with histories of removal from their biological home by Child Protective Services were obtained from a private neurofeedback practice. All of the children were assessed prior to treatment using the Child Behavior Checklist (CBCL) and the Test of Variables of Attention (TOVA) and again after 30 sessions of individualized, qEEG-guided neurofeedback.

Results. T-test analysis of pre- and post-scores on the CBCL showed significant changes in the areas of externalizing problems, internalizing problems, social problems, aggressive behavior, thought problems, delinquent behavior, anxiety/depression, and attention problems ($p < .05$). TOVA omission error, commission error, and variability scores also improved significantly following neurofeedback training ($p < .05$). Some pre-treatment qEEG patterns common to this group of children were identified.

Conclusions. The CBCL and TOVA score improvements observed in this study indicate that

Lark Huang-Storms and Janice Dunn are doctoral students in Clinical Health Psychology and Behavioral Medicine, University of North Texas, Denton, TX 76203.

Eugenia Bodenhamer-Davis is Director of the Neurotherapy Lab and Associate Professor in the Department of Rehabilitation, Social Work and Addictions, University of North Texas, Denton, TX 76203.

Richard Davis is in private practice in the Dallas-Fort Worth metroplex.

Address correspondence to: Lark Huang-Storms at the above address (E-Mail: larktim@teleport.com).

Journal of Neurotherapy, Vol. 10(4) 2006
Available online at http://jn.haworthpress.com

doi:10.1300/J184v10n04_02

neurofeedback is effective in reducing behavioral, emotional, social, and cognitive problems in children with histories of neglect and/or abuse. doi:10.1300/J184v10n04_02

KEYWORDS. qEEG, neurofeedback, reactive attachment disorder, child abuse, child neglect, behavior disorders, self-regulation

INTRODUCTION

Attachment Relationships and the Development of Self-Regulation

Over thirty years ago, Bowlby asserted the central importance of early caretaker relationships on the social and emotional development of children. His attachment theory suggested that an infant's ability to cope with stress is correlated with biologically driven mother-child behavior patterns that promote primary caretaker proximity (Bowlby, 1969). Ainsworth, Blehar, Waters and Wall (1978) expanded this theory by defining four infant attachment styles and contributing a greater understanding of the purpose behind the primary caregiver's role: to provide a secure base from which a child explores his/her surroundings and incorporates internal working models of trusting relationships. This seminal work was followed by a wealth of empirical research demonstrating a correlation between level of attachment security and the development of a wide range of psychopathology in children and adults, including mood, personality, conduct, and anxiety disorders (Crittenden, 1995; Schore, 1994).

Attachment behaviors serve important protective functions beginning at birth, and are believed to correspond with the onset of independent locomotion in vertebrates (Clutton-Brock, 1991). Increasing evidence suggests that a primary function of the attachment relationship is to develop a child's ability to self-monitor affect, self-regulate physiological arousal level, and self-organize coping functions for stress (Cassidy, 1994; Cicchetti & Tucker, 1994). Kopp (1989) asserts that the development of affect regulation proceeds from initial reliance on a caregiver, to self-soothing behaviors, and finally to language based cognitive coping strategies. The central role of attachment in the development of self-regulation may explain why relational trauma from the social environment has been shown to have more negative impact on the rapidly developing brains of infants and children than assaults from the inanimate physical environment (Schore, 2001; Sgoifo, Koolhaas & De Boor, 1999).

Because infants and young children are unable to effectively modulate affective and physiological arousal states independently, their developing capacity to cope with dysregulated states depends on the responses of caregivers who are psychobiologically attuned to their needs (Schore, 1994). Caretakers externally manage infants' psychophysiologic states by responding to them in consistently sensitive ways, for example through accurate mirroring of affect and sensitivity to gaze aversion as a signal of over-stimulation (Field, 1994). Through such processes, children learn to develop strategies to manage high arousal and regain a state of organization when homeostasis has been disrupted. Research by Goldberg, MacKay-Soroka, and Rochester (1994) showed that mothers in securely attached relationships with their infants responded equally to their babies' positive and negative affects, whereas mothers in poorly attached relationships responded predominantly to negative affects (therefore conditioning their children to increase negative affective behaviors). Thus, strategies developed early in life to manage arousal can be understood as forming the neurodevelopmental building blocks of lifelong personality structure and affective behavior patterns (Bradley, 2000).

Neurophysiological Impact of Early Relationship Trauma

When caregivers are neglectful, inconsistent, or abusive, infants and young children are

left vulnerable to psychophysiological distress states from which they cannot escape. Two interacting response patterns have thus far been identified in children. The first is a hyperarousal (fight or flight) response, which is mediated by sympathetic activation of the limbic-hypothalamic-pituitary-adrenal (HPA) axis, and results in increased levels of cortisol, acetylcholine, adrenaline, and noradrenaline within the developing brain. The second response, more common in girls and younger children/infants, is the "dissociative" continuum, which is mediated by the parasympathetic activation of dorsal vagal responses (i.e., reduced metabolism, heart rate, and respiration rate), endogenous opioids, and the dopaminergic system (Perry & Pate, 1994). The hyperarousal and dissociative responses to stress are not discreet; rather, when stressful situations are perceived to be hopeless or overwhelming, initial sympathetic arousal may be followed by disengagement from external stimuli via parasympathetic activation of the vagal and opioid systems (Perry, 1994).

Prolonged hyperarousal/dissociative states can chronically dysregulate a child's psychophysiological stress-response systems (e.g., the HPA axis) and patterns of coping behaviors (e.g., withdrawal or aggression; Manassis & Bradley, 1994; Post, Weiss, & Leverich, 1994). Chronic dysregulation is associated with sensitization of the brain stem and midbrain neurotransmitter systems, such that early adverse attachment experiences essentially "kindle" the limbic areas of the brain to be physiologically reactive (Post, Rubinow, & Ballenger, 1984). In particular, van der Kolk and Greenberg (1987) have suggested that the repeated trauma of child abuse may dispose the stress-sensitive amygdaloid nuclei to develop a kindling response, by which repeated intermittent stimulation produces increasingly greater alterations in neuronal excitability, potentially resulting in seizures. Othmer, Othmer, and Kaiser (1999) describe the kindling process as a "practice effect" of the brain's successive experiences of dysfunction or overarousal, and suggest that vulnerable physiologic feedback systems tend to become more dysregulated over time when left alone. Work by Adamec and Stark-Adamec (1989) demonstrates that kindling in the amygdala induces a "defensive personality" in domestic cats, the intensity of which is mediated by both experience and strength of neurotransmission between the basomedial nucleus of the amygdala and the ventromedial nucleus of the hypothalamus. Because of the dense interconnections amongst the prefrontal cortex, hypothalamus, amygdala, thalamas, cingulate gyrus, and hippocampus, it appears likely that various patterns established through early attachment experiences could set a precedent for the development of relatively automatic affect-regulating mechanisms within these feedback systems of the brain (Schore, 1994; Bradley, 2000).

In relational trauma, the developing limbic system is repeatedly exposed to high levels of excitotoxic neurotransmitters, such as glutamate, cortisol, and NMDA-sensitive glutamate receptor, all of which are associated with neurotoxicity and abnormal synapse elimination in early brain development (Choi, 1992; Moghaddam, Bolinao, Syein-Behrens, & Sapolsky, 1994). It is hypothesized that stress-induced increases in gluccocorticoids in postnatal periods selectively induce neuronal cell death in the limbic system and impact abnormal limbic circuitry. In particular, there is ample evidence that, in adults, the cellular organization of the hippocampus can be dramatically affected by levels of corticosteroids, which can exert deleterious effects on the hippocampal pyramidal cells (Sapolsky, 1993; Watanabe, Gould, & McEwen, 1992). In separate studies, Carrion et al. (2001) and De Bellis et al. (1999) found that children with histories of trauma and PTSD symptoms had significantly smaller total brain cerebral volume than matched control groups. In both studies, after statistically controlling for total brain volume, no significant decreases in hippocampal volumes were found in the PTSD child population, suggesting a more generalized effect of the early developmental neurotoxic effects of glucocorticoids.

According to Schore (2001), attachment experiences in infancy particularly influence the experience-dependant maturation of the right orbitofrontal cortex, which is dominant for the processing of affect-regulation, visual emotional information, and attachment experiences. During the first few months after birth, the right hemisphere develops more rapidly

than the left, which theoretically makes it more vulnerable to the consequences of extreme stress and neglect (Galaburda, 1984). Furthermore, Read, Perry, Moskowitz, & Connolly (2001) propose that abuse from six months until three to six years of age may have the greatest differential effect on the left hemisphere. Their findings are supported by the work of Teicher et al. (1997), which suggests that dendritic growth in the left hemisphere surpasses that of the right hemisphere at about six months of age.

Nonspecific EEG abnormalities have been found in populations of abused children, including psychologically abused children and physically abused children without head trauma (Teicher et al., 1997; Green, Voeller, & Gaines, 1981). Van Bloem's (2000) work with children and adolescents diagnosed with RAD revealed specific patterns of EEG slowing in the frontal lobes and right temporal lobe. Fisher, Turber, and Gunkelman (2005) also reported right temporal as well as vertex slowing in qEEG's of children with RAD. Ito, Teicher, Glod, & Ackerman (1998) observed childhood physical and sexual abuse to be associated with an increased prevalence of left-sided EEG abnormalities (particularly fronto-temporally). In terms of cortical EEG coherence, Teicher et al. (1997) found that a group of sexually and physically abused children had greater average left hemisphere hypercoherence than normal children, but comparable right hemisphere coherence patterns (indicating diminished left hemisphere differentiation in the abused group). Their work highlights the relationship between limbic system dysfunction and reversed left/right hemispheric asymmetry, asserting the possibility that early abuse may impede hemispheric integration and the establishment of normal left cortical dominance. In contrast, in a small clinical sample of women, Black, Hudspeth, Townsend and Bodenhamer-Davis (2002) found histories of childhood sexual abuse to be associated with hypocoherence in left frontal regions in the theta and beta bands and hypercoherence in posterior central regions across all bands, although these findings were not wholly replicated with a high functioning non-clinical college sample of women with sex abuse histories (Black, 2005).

The question naturally arises whether these brain abnormalities may be at least partially the result of genetic factors, intergenerational effects of parenting, or learned stress-coping behaviors. Indeed, in addition to evidence of intergenerational transmission of child abuse in humans (Kaufman & Zigler, 1989), there is evidence in group-living pigtail macaques of genealogical and demographic influences on maternal neglect and abuse of offspring (Dario, Wallen, & Carroll, 1997). Because it is ethically difficult to design studies to tease apart these hypotheses, there has not been definitive research to clarify these questions. However, there are a number of studies of neglected and abused children in orphanage settings that have found dramatically smaller frontal-occipital head circumferences (38% below the third percentile), as well as CT and MRI findings of enlarged ventricles and cortical atrophy in this population (Perry & Pollard, 1998; Rutter et al., 1998). Using functional magnetic resonance imaging (fMRI) with a population of Romanian orphans, Chungani et al. (2001) found these children had decreased metabolic activity in the orbital frontal gyrus, intralimbic prefrontal cortex, amygdala, hippocampus, lateral temporal cortex, and brainstem.

Animal research with rats provides further support for the neurodevelopmental impact of neglect/physical abuse, specifically on hemispheric laterality (Denenberg, 1983), hippocampal shrinkage (Meaney, Aitken, & van Berkel et al., 1988), and alterations of neuro-endocrine stress response systems (Fride, Dan, Feldon, Halevy, & Weinstock, 1986). Research conducted with chimpanzees and gorillas (Davenport & Rogers, 1970) and with rhesus monkeys (Harlow & Harlow, 1965), though not neurophysiological in focus, demonstrates the profoundly negative behavioral outcomes of severe social deprivation during the first year of life (similar to behaviors seen in neglected and abused children). Intriguingly, there is even evidence that maternal neglect in invertebrate wolf spiders results in decreased central nervous system (CNS) development, as observed in decreased brain weight and number of brain cells (Punzo & Ludwig, 2002). Spiderlings removed from their mothers also show less ability to hunt and learn maze navigation, as compared to spiderlings who remain with their mothers.

Behavioral Manifestations of Relationship Trauma

Neurodevelopmental research has established that, because of the brain's extreme malleability and sensitivity to experience in early childhood, traumatic events in the first few years of life can have long-term impacts on socio-emotional and cognitive functioning. This is particularly likely if events are severe, unpredictable, or ambient–all of which describe relational trauma in the form of neglect or abuse (Perry, 1994). Child neglect and abuse have been shown to have an etiological role in a remarkable range of behavioral disorders affecting children, including attention deficit disorders, mood and anxiety disorders, conduct and oppositional defiant disorders, reactive attachment disorder (RAD), learning disabilities, post-traumatic stress disorder (PTSD), eating disorders, substance abuse, and dissociative disorders (Beitchman et al., 1992; Boney-McCoy & Finkelhor, 1995). Although estimates vary widely, some researchers propose that up to 80% of abused children display symptoms of severe attachment disturbance or RAD (Hall & Geher, 2003).

Numerous studies demonstrate that children who have been mistreated or have had multiple, inconsistent caregivers are unlikely to develop secure attachment styles (Egeland & Sroufe, 1981). Insecure or "disorganized" attachment (George & Main, 1979) is suggestive of a lack of pattern/strategy for regulation of affect, thus it is not surprising that children with histories of disrupted attachment commonly experience externalizing problem behaviors, sleep and eating irregularities, and attentional difficulties indicative of poor self-regulation functions (DeGangi, 2000). Behaviorally, limbic dysregulation may inhibit a child's capacity to cope with stressors by maintaining heightened arousability (e.g., a child may rapidly escalate from feeling slight anxiety to terror) and supporting chronically heightened arousal states (e.g., a child may not maintain a focused state for academic learning due to hypervigilance to threat). Foster and adoptive parents and professionals at Child Protective Services (CPS) commonly voice concerns that these children demonstrate peer aggression, stealing, food hoarding/gorging, destruction of property, poor impulse control, limited cause-and-effect thinking, inappropriate sexual behavior, school failure, and hyperactivity (Iwaniec, 1995). Difficulty forming trusting relationships, indiscriminate affection with strangers concomitant with refusal to give affection to family members, limited ability to empathize, and poor social skills are also prevalent in this population of children, particularly those diagnosed with RAD (Hall & Geher, 2003).

Neurofeedback with Pediatric Populations

Neurofeedback, or electroencephalographic (EEG) biofeedback, is among the most promising modalities for the treatment of child and adolescent psychological disorders. Interest in neurofeedback's potential as a therapeutic intervention for pediatric populations is heightened by concerns regarding the uncertain long-term effects of psychiatric and stimulant medications on the developing brain (Wilens, 2004). Furthermore, for children with histories of relational trauma, attachment disorders, or RAD, conventional relationship-based therapies (including cognitive therapies and play-based therapies) have demonstrated limited clinical success (Chanitz, 1995; Wilson, 2001).

There has been increasing clinical and empirical support for the efficacy of neurofeedback interventions for adolescent and child applications (Hirshberg, Chui, & Frazier, 2005). The most substantial literature supports neurofeedback training to decrease slow wave activity while increasing the power of 12-15 Hz and 15-18 Hz activity for improvement of ADHD symptoms in children (Linden, Habib, & Radojevic, 1996; Lubar, Swartwood, Swartwood, & O'Donnell, 1995; Rossiter & La Vaque, 1995; Monastra, Monastra, & George, 2002; Fuchs, Birbaumer, Lutaenberger, Gruzelier, & Kaiser, 2003; Rossiter, 2004). In addition, the literature includes some clinical evidence that neurofeedback benefits children with autism spectrum disorder (Jarusiewicz, 2002; Sichel, Fehmi, & Goldstein, 1995), asthma (Tansey, 1992), pediatric stroke (Ayers, 1995), and pediatric migraine (Siniatchkin et al., 2000). As of yet, no study has investigated the effectiveness of neurofeedback with children and adolescents with histories of relational trauma, though there are a few unpublished clinical reports of

success with this pediatric population (e.g., Fisher et al., 2005).

Models Supporting Neurofeedback for Children with Histories of Relational Trauma

There is an increasing number of voices in the behavioral sciences asserting the prominent role of arousal dysregulation in the development of psychopathology (Grotstein, 1986; Le Doux, 1996). This has fostered a meaningful integration of biological and psychosocial approaches regarding the conceptualization and treatment of mental health and illness. Based on work by Gorman, Liebowitz, Fryer, and Stein (1989), Bradley (2000) proposes a general model for the development of affect regulation in which the pre-frontal cortex integrates experiential learning and cognitive schemas with the output of the limbic and reticular activating systems. Similarly, Schore (1994) describes a model of arousal regulation in which the orbitofrontal cortex sits at the apex of a fluctuating sympathetic-parasympathetic ANS balancing system. As outlined above, early experiences of relational trauma produce deficits in the ability to regulate affect and manage arousal through processes of limbic kindling, dysregulation of neurochemical ANS stress-response mechanisms, and maladaptive internalized coping strategies. In addition, the fact that relational trauma is a risk factor common to a wide array of psychological disorders points to the centrality of physiological and emotional self-regulation as a key variable in psychopathology.

Othmer et al. (1999) propose a dysregulation model of psychopathology based on a failure of control systems and feedback loops in the brain, specifically in terms of the regulatory EEG activity of the central nervous system (CNS). This model contributes the addition of the electrical frequency domain to the traditional neurochemical conceptualization of CNS functioning, and asserts that rhythmicity is the basis for the organization of brain function. Rhythmic activity of the brain is produced by complex interactions of feedback loops (or "servosystems") that are potentially vulnerable to "setpoint errors," "instabilities," and "over/under-shooting" in response to stimuli. Thompson and Thompson (2003) describe three basic resonant cortical loops that affect EEG rhythmicity (local, regional, and global), all of which can operate spontaneously or may be driven by subcortical thalamic pacemakers. As explained by Lubar (1997), changes in the patterned functioning of cortical loops (as a result of learning, for example) can affect the intrinsic firing rate of thalamic pacemakers, which is ultimately associated with changes in mental state.

As conceptualized by Sterman (1996) and by Othmer and colleagues (1999), EEG biofeedback is a means of operantly conditioning the rhythmic electrical manifestations of CNS regulatory function by challenging the brain to shift away from unstable firing patterns toward more homeostatic ones. The addition of an external feedback loop via biofeedback encourages the brain to alter its prevailing rhythmicity through a repetitive process of imposed disequilibrium and attempted return to baseline. This "regulatory challenge model" of neurofeedback facilitates improved ability to maintain homeostasis and improved stability of the regulatory CNS system itself. Although factors other than early relational trauma can disrupt normal brain development and regulation patterns, it would appear that children with histories of traumatic attachment struggle fundamentally with self-regulatory challenges. EEG biofeedback may teach children to self-regulate brain rhythmicity, which in turn may impact their ability to adaptively manage physiological and emotional arousal states. Because poor self-regulation of arousal is central to the vast majority of the behavioral difficulties experienced by children with traumatic attachment histories, neurofeedback has the potential to affect global improvements in the typical problem areas of attention, aggression, impulse control, hypervigilance, classroom learning, empathy and trust formation, sleep, hyperactivity, etc. The mechanisms and objectives of EEG biofeedback would seem to be expressly appropriate for the behavioral and neurophysiological needs of this population. The following pilot study using a clinical case series represents an initial step in the empirically-based investigation of neurofeedback treatment for children with RAD symptoms.

sessions, EEG was recorded from a referential montage, with a single-electrode placement referenced to the ipsilateral ear and ground on the opposite ear, using a sampling rate of 128 Hz.

The Test of Variables of Attention (TOVA; Greenberg, 1987) was used to assess self-regulatory changes in attention following neurofeedback training. The TOVA is a computerized visual continuous performance test in which two easily discriminated visual stimuli are presented for 100 milliseconds every 2 seconds for 22.5 minutes. The variables assessed by the TOVA (i.e., errors of omission, errors of commission, response time, and response time variability) have been shown to be significantly different between pre-treatment and methylphenidate treatment conditions, and are reported to be invulnerable to test-retest practice effects (Greenberg, 1987).

The Child Behavior Checklist (CBCL/4-18) is widely utilized in both clinical and research applications (Achenbach, 1991). It was used in the present study to assess behavioral and emotional changes following neurofeedback training, as reported by adoptive parents. The CBCL is a four-page form designed to obtain descriptions of the competencies and behavioral-emotional problems of children that impact successful adaptive development. Parents with a reading ability at the fifth grade level or greater typically complete the form in 10 to 15 minutes. The CBCL includes competence scales (i.e., activities, social, and school), as well as eight syndrome scales. The syndrome scales include an internalizing grouping (i.e., withdrawn, somatic complaints, and anxious/depressed), an externalizing grouping (i.e., delinquent behavior and aggressive behavior), as well as scales for social problems, thought problems, and attention problems.

Procedure

The pre-treatment CBCL was completed by an adoptive parent prior to or concurrent with the child's first intake session. Child clients completed the pre-treatment TOVA during the initial session. All but three participants in the study completed a post-treatment TOVA following the 30th neurofeedback training session. Several of the children continued for about 10 additional sessions, but only the post 30 session assessment data was used for treatment outcome analysis. The same adoptive parents who completed the pre-treatment CBCLs also completed the post-treatment CBCLs at 30 sessions of training. QEEG data collection and analysis occurred prior to neurofeedback training, so that individualized treatment protocols could be developed.

Neurofeedback training consisted of thirty minutes of auditory and visual feedback per session, while the child was seated comfortably in a quiet room. Although each child was treated with an individualized protocol, there was nevertheless much similarity among these protocols due to the common features seen in the group's qEEGs. The training site locations were derived from the individual NeuroRep weighted average qEEG topographies (not from the relative power Z scores reported earlier in this paper from the subsequent university study of the larger sample of children with relational trauma). The weighted average topographies showed excess slow wave amplitude in the frontal and/or central vertex locations for all subjects treated. This pattern was consistent with the RAD qEEG patterns reported by Van Bloem (2000) and by Fisher et al. (2005). In all cases, feedback initially was contingent on the reduction of 2-7 hertz (Hz) activity at CZ or FZ based on the International 10-20 electrode placement system (Jasper, 1958). Training at the frontal sites usually commenced by rewarding the reduction of 2-7 Hz activity. In a few cases, both the CZ and FZ sites were trained within the first 20 sessions, targeting FZ first for 10-15 sessions and then moving to CZ for 5-8 sessions. In cases where the qEEG showed excessive fast frequency activity at FZ or CZ as well, a second inhibit filter band of 20-32 Hz was also utilized in the initial protocol. No frequencies were enhanced during the initial frontal training period; all protocols were inhibiting only.

This protocol was followed until the individual subject's training records indicated consistent ability to maintain targeted thresholds, i.e., consistently reducing 2-7 and 20-32 Hz activity below baseline levels, and also when changes in behavioral symptoms were observed. In the majority of cases, guided by subjects' individual qEEGs and/or behavioral changes, the next

stage of training targeted right hemisphere protocols, enchancing 12-15 Hz and reducing 2-7 Hz at T4, P4 or C4. Often, this right side training was initiated following treatment at frontal and vertex sites if the clinician observed or parents reported increased agitation in the child. Right side training reliably reduced such responses. For those children not requiring this right side training focus, second stage protocols targeted various qEEG-determined sites and frequencies, most often reducing 8-12 Hz Alpha activity at C3, CZ, C4, P3, PZ, P4, T5, or T6. Again, no frequencies were enhanced in these alpha reduction protocols.

RESULTS

Differences in CBCL raw scores before and after neurofeedback training were analyzed using the Statistical Package for the Behavioral Sciences (SPSS). Eighteen of the 20 participants' parents completed valid pre- and post-CBCL parent forms, which were included in the analysis. Raw scores were used for analysis in accordance with CBCL manual recommendations for research (Achenbach, 1991). A two-tailed paired t-test analysis yielded meaningful statistical differences in 10 of the 14 assessed competence and syndrome scores, indicating that significant behavioral improvements were observed by adoptive parents following neurofeedback training. Total syndrome scale scores decreased an average of 23.05 points (SD = 21.44) with a 95% confidence interval of 12.73-33.39, and were significant at $t(18)$ = 4.69, $p < .001$. Externalizing scale scores decreased an average of 7.32 points (SD = 8.40) with a 95% confidence interval of 3.27-11.36, and were significant at $t(18) = 3.799$, $p = .001$. Both total and externalizing scale score changes represent large effect sizes ($d = .78$ and $d = .94$, respectively). Internalizing scale scores decreased an average of 4.74 points ($SD = 7.26$), with a 95% confidence interval of 1.24-8.24, and significance at $t(18) = 2.84$, $p < .01$. This represents a medium effect size ($d = .59$). Figure 1 presents these results graphically.

Within the CBCL syndrome scales, six of the eight scale scores significantly improved: social problems [$t(18) = 3.59$, $p = .002$], aggressive behaviors [$t(18) = 3.72$, $p = .002$], thought problems [$t(18) = 3.33$, $p = .004$], delinquent behavior [$t(18) = 2.82$, $p = .01$], attention [$t(18) = 2.82$, $p = .01$], and anxious/depressed [$t(18) = 2.80$, $p = .01$]. All of these changes represent medium effect sizes ($d > .55$). Somatic complaints and withdrawn scale scores also improved, but not to statistical significance. Figure 2 presents these results graphically. Of the three competence scales, there was a statistically significant improvement on the social scale [$t(12) = 2.67$, $p = .021$] with medium ef-

FIGURE 1. Averaged raw scores on the CBCL total, externalizing, and internalizing scales before and after neurofeedback treatment. All differences are statistically significant at $p < .05$.

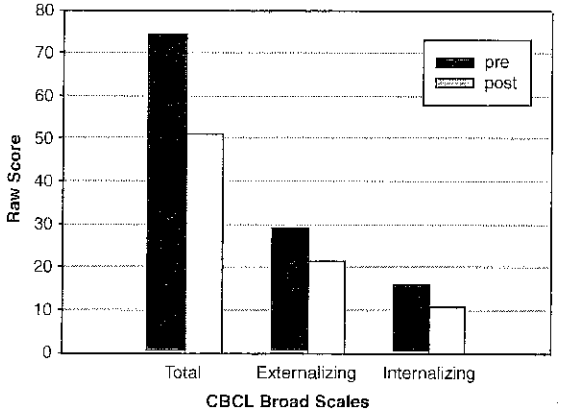
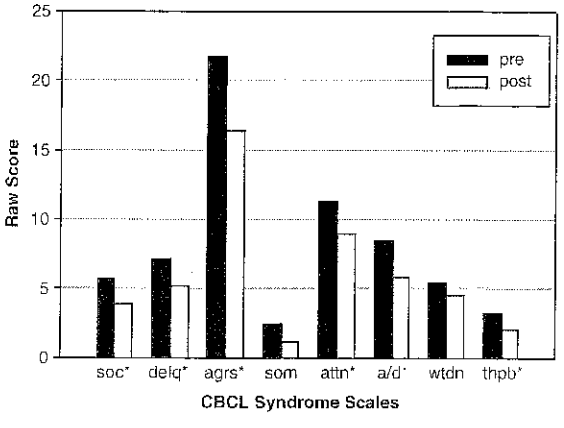


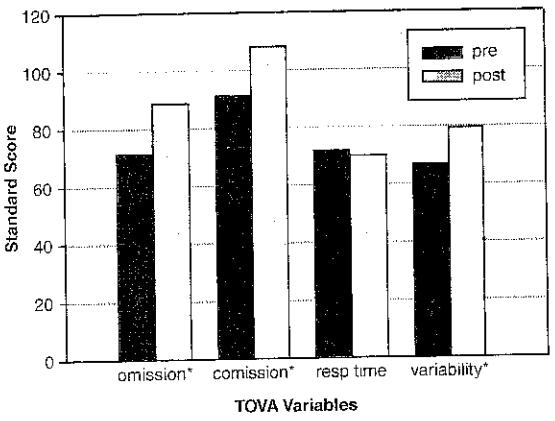
FIGURE 2. Averaged raw scores on the CBCL syndrome scales (social problems, delinquent behaviors, aggressive behaviors, somatic complaints, attention problems, anxiety/depression, withdrawn, and thought problems) before and after neurofeedback treatment. An asterisk indicates significance at $p < .05$.



fect size (d = .68). Activities and school scale scores also showed positive trends following treatment, but not to statistical significance.

Pre- and post-TOVA standard scores were also analyzed using SPSS. Ten children completed valid pre- and post-TOVAs, while the remaining 10 protocols were not included due to incompletion or one or more invalid quarter scores (e.g., due to >10% anticipatory errors). Two-tailed paired t-test analysis found significant differences post-treatment for three of the four TOVA variables: omission errors [$t(9) = -2.37$, $p = .042$], commission errors [$t(9) = -3.16$, $p = .011$] and total variability [$t(9) = -2.39$, $p = .04$]. On the TOVA, lower scores represent more problematic behavior, with average standard scores ranging from 85-115. Omission errors improved an average of 17.3 points (SD = 23.0) from an average pre-treatment score of 71.5 to an average post-treatment score of 88.8 (95% CI 0.8-33.8). Commission errors improved an average of 16.4 points (SD = 16.4) from an average pre-treatment score of 91.5 to an average post-treatment score of 107.9 (95% CI 4.7-28.1). Total variability scores increased an average of 12.3 points (SD = 16.3) from 67.1 at pre-test to 79.4 at post-test (95% CI 0.7-23.9). Omission, commission, and total variability scale score changes all represent medium effect sizes (d > .60), with average omission and total variability scores moving from abnormal to normal ranges following treatment. Total response time standard scores decreased slightly (from an average of 71.9 to 70.0). These results are depicted in Figure 3.

FIGURE 3. Averaged TOVA standard scores (omission errors, commission errors, response time, and response time variability) before and after neurofeedback treatment. An asterisk indicates significance at $p < .05$.



DISCUSSION

The CBCL and TOVA score improvements observed in this study suggest that neurofeedback is an effective treatment for children with behavioral problems associated with histories of neglect and/or abuse. Specifically, aggressive, delinquent, and socially problematic behaviors (e.g., lying, fighting, demanding attention, etc.) appear to be strongly impacted. Behaviors associated with attentional problems, anxiety/depression, and thought problems also appear to be significantly reduced. These data are encouraging because these problematic behaviors are not only common and disabling for children with traumatic attachment histories, but are also frequently resistant to change via traditional therapeutic interventions. On the other hand, because these behaviors are often the primary impetus for referral, they are also potentially vulnerable to overly-optimistic behavior ratings from parents seeking to justify treatment. It should be noted that the CBCL activities and school scales may not have shown significant changes because many children received treatment during summer vacation and, as several parents reported, it was difficult to assess these behaviors during summer months when school was not in session and social activities were less frequent.

Improvement of self-regulatory behaviors characterizes the changes in CBCL and TOVA Scale scores observed in this sample. For the majority of the children, CBCL and/or TOVA scores shifted from clinical/borderline ranges into normal ranges, indicating meaningful behavioral changes occurred. The statistical significance and medium to large effect sizes of score changes following treatment is notable, especially considering the small sample size of the study. If, as has been suggested, neurofeedback training teaches children to self-regulate brain rhythmicity, the broad changes observed in the CBCL Scale scores suggest a general decrease of physiological and emotional arousal

dysregulation. CBCL externalizing scale score changes, as well as decreases in thought problems (e.g., repetitive actions, distractibility), attention problems (e.g., poor concentration, impulsiveness), anxiety/depression (e.g., nervousness, crying), and somatic complaints (e.g., tiredness, headaches) may all be expressions of improved stability of the regulatory CNS system. Improvements on TOVA omission, commission, and total variability scores imply decreased impulsivity and improved attention modulation, and corroborate positive changes on the attention scale of the CBCL. (The slight decrease in response time scores on the TOVA is commonly seen immediately following neurofeedback, as individuals become more focused and deliberate in their responses.)

Although encouraging, this study clearly represents only a preliminary clinical investigation of the effectiveness of neurofeedback treatment for this population. Some of the major caveats of the study include the small and non-randomized sample, the lack of control group, and the failure to control for potential effects of medications and concurrent additional therapies. The use of an external behavioral rating (CBCL) and a standardized performance test (TOVA) provides a multidimensional method of evaluation, but with considerable limitations. In addition to the potential vulnerability of the CBCL to low reliability and validity due to parental investment in positive change, the CBCL is designed to be given at intervals of six months or more. In the present study, some children were re-evaluated as early as three months following the initial session. Standard deviations of certain CBCL and TOVA scores were also large, thus should be interpreted with some caution in spite of the robustness of *t*-tests to violations of normality. These data imply that there is a high degree of variability between children in terms of their initial presentation and their patterns of improvement following neurofeedback training.

It is interesting, both clinically and etiologically, that the majority of the children's initial qEEGs (specifically the weighted average amplitude topographies in NeuroRep) indicated use of similar treatment protocols (inhibiting 2-7 Hz activity at CZ or FZ followed by inhibiting 2-7 Hz and enhancing 12-15 Hz at T4, P4 or C4). Other clinicians have reported combining the slow wave inhibits at Fz and Cz with enhancement of 15-18 Hz (Fisher, Turber, & Gunkelman, 2005), which is apparently an effective alternative to the inhibit-only focus of the treatment reported in this study. Although T6 has been mentioned in the literature as a focal site in RAD (Schore, 1994), T6 was not found to have as much slow wave activity as the Cz and Fz sites in the pre-treatment qEEGs of the current sample. It should be noted that some of the children who comprised the sample had never received a formal diagnosis of RAD. Further analysis of qEEG patterns in this population may clarify areas of the developing brain that are particularly susceptible to early relationship trauma, as well as help establish more standardized neurofeedback protocols for this population. In the future, it would also be beneficial to analyze the actual power changes in the EEG frequency bands as a result of neurofeedback. Long-term follow-ups, additional assessment measures, larger, randomized samples, and control of additional therapies were regrettably not possible for the present study. Nonetheless, the findings clearly support the use of neurofeedback training for children with histories of relationship trauma and serve to clarify directions for further research. As a first study of this kind, it will hopefully stimulate interest in the potential of neurofeedback treatment for the many children and families struggling with the developmental effects of early abuse/neglect.

REFERENCES

Achenbach, T. M. (1991). *Integrative guide to the 1991 CBCL/4-18, YSR, and TRF profiles*. Burlington, VT: University of Vermont.

Adamec, R. E., & Stark-Adamec, C. (1989). Behavioral inhibition and anxiety: Dispositional, developmental, and neural aspects of the anxious personality of domestic cat. In J. S. Reznick (Ed.), *Perspectives on behavioral inhibition*. Chicago: University of Chicago Press.

Ainsworth, M. D., Blehar, M. C., Waters, E., & Wall, S. (1978). *Patterns of attachment*. Hillsdale, NJ: Erlbaum.

Ayers, M.E. (1995). A controlled study of EEG neurofeedback and physical therapy with pediatric stroke, age seven months to age fifteen, occurring prior to birth. *Biofeedback and Self-Regulation, 20* (3), 318.

Beitchman, J., Zucker, K., Hood, J., DaCosta, G., Ackman, D., & Cassavia, E. (1992). A review of the long-term

effects of child sexual abuse. *Child Abuse and Neglect, 16*, 101-118.

Black, L. (2005). *Effects of childhood sexual abuse on adult brain plasticity as measured by quantitative electroencephalogram, neuropsychological, and psychological tests*. Unpublished doctoral dissertation, University of North Texas, Denton.

Black, L., Hudspeth, W., Townsend, A., & Bodenhamer-Davis, E. (2002, October). *Effects of childhood sexual abuse on adult brain plasticity as measured by quantitative electroencephalogram.* Presentation at the Society for Neuronal Regulation annual conference, Scottsdale, AZ.

Boney-McCoy, S., & Finkelhor, D. (1995). Psychosocial sequelae of violent victimization in a national youth sample. *Journal of Consulting and Clinical Psychology, 63*, 726-736.

Bowlby, J. (1969). *Attachment and loss* (Vol. 1). New York: Basic Books.

Bradley, S. J. (2000). *Affect regulation and the development of psychopathology*. New York: Guilford Press.

Carrion, V., Weens, C., Eliez, S., Patwardhan, A, Brown, W., Ray, R., et al. (2001). Attenuation of frontal asymmetry in pediatric post-traumatic stress disorder. *Biological Psychiatry, 50* (12), 943-951.

Cassidy, J. (1994). Emotion regulation: Influences of attachment relationships. In N.A. Fox (Ed.), The development of emotion regulation: Biological and behavioral considerations. *Monographs of the Society for Research in Child Development, 59* (2-3), 228-248.

Chanitz, S. P. (1995). Interventions with children with developmental disabilities and attachment disorders. *Developmental and Behavioral Pediatrics, 16* (3), 17-20.

Choi, D. W. (1992). Excitotoxic cell death. *Journal of Neurobiology, 23*, 1261-1276.

Chugani, H. T., Behen, M. E., Muzik, O., Juhasz, C., Nagy, F., & Chugani, D. C. (2001). Local brain functional activity following early deprivation: A study of post-institutionalized Romanian orphans, *Neuroimage 14*, 1290-1301.

Cicchetti, D., & Tucker, D. (1994). Development and self-regulatory structures of the mind. *Development and Psychopathology, 6*, 533-549.

Crittenden, P. M. (1995). Attachment and psychopahology. In S. Goldberg, R. Muir & J. Kerr (Eds.), *Attachment theory: Social, developmental and clinical perspectives*. Hillsdale, NJ: Analytic Press.

Clutton-Brock, T. H. (1991). *The evolution of parental care*. Princeton, NJ: Princeton University Press.

Dario, M., Wallen, K., & Carroll, K. A. (1997). Infant abuse runs in families of group-living pigtail macaques. *Child Abuse and Neglect, 21* (5), 465-471.

Davenport, R. K. & Rogers, C. J. (1970). Differential rearing of the chimpanzee: A project survey. In G.H. Bourne (Ed.), *ThecChimpanzee*. Basel: Karger.

De Bellis, M., Keshavan, M., Clark, D., Casey, B., Giedd, J., Boring, A., et al. (1999). Developmental traumatology part II: Brain development. *Biological Psychiatry, 45*, 1271-1284.

DeGangi, G. (2000). *Pediatric disorders of regulation in affect and behavior: A therapist's guide to assessment and treatment*. San Diego, CA: Academic Press.

Denenberg, V. H. (1983). Lateralization of function in rats. *American Journal of Physiology, 245*, 505-509.

Egeland, B., & Sroufe, L. A. (1981). Attachment and early maltreatment. *Child Development, 52*, 44-52.

Field, T. (1994). The effects of mothers' physical and emotional unavailability on emotion regulation. In N.A. Fox (Ed.), The development of emotion regulation: Biological and behavioral considerations. *Monographs of the Society for Research in Child Development, 59*, 208-227.

Fisher, B. S, Turber, J., & Gunkelman, J. (2005, September). *Healing the attachment disorder before and after behavioral and neurological functioning*. Workshop presented at the annual meeting of the International Society for Neuronal Regulation, Denver, CO.

Fisher, S. F. (2002). Neurofeedback: A treatment for reactive attachment disorder. *EEG Spectrum*. [On-line]. Available: http://www. www.eegspectrum.com/

Fride, E., Dan, Y., Feldon, J., Halevy, G., & Weinstock, M. (1986). Effects of prenatal stress on vulnerability to stress in prepubertal and adult rats. *Physiology and Behavior, 37* (5), 681- 688.

Fuchs, T., Birbaumer, N., Lutenberger, W., Gruzelier, J. H., & Kaiser, J. (2003). Neurofeedback treatment for attention-deficit/hyperactivity disorder in children: A comparison with Methylphenidate. *Applied Psychophysiology and Biofeedback, 28* (1), 1-12.

Galaburda, A. M. (1984). Anatomical asymmetries in the human brain. In N. Geschwind & A.M. Galaburda (Eds.), *Biological foundations of cerebral dominance*. Cambridge, MA: Harvard University Press.

George, C., & Main, M. (1979). Social interactions of young abused children: Approach, avoidance and aggression. *Child Development, 50*, 306-318.

Goldberg, S., MacKay-Soroka, S., & Rochester, M. (1994). Affect, attachment and maternal responsiveness. *Infant Behavior and Development, 17*, 335-339.

Gorman, J. M., Liebowitz, M. R., Fryer, A. J., & Stein, J. (1989). A neuroanatomical hypothesis for panic disorder. *American Journal of Psychiatry, 146*, 148-161.

Green A., Voeller, K., & Gaines, R. (1981). Neurological impairment in maltreated children. *Child Abuse and Neglect, 5*, 121-134.

Greenberg, L. (1987). An objective measure of methylphenidate response: Clinical use of the MCA. *Psychopharmacology Bulletin, 23*, 279-282.

Grotstein, J. S. (1986). The psychology of powerlessness: Disorders of self-regulation and interactional regulation as a new paradigm for psychopathology. *Psychoanalytic Inquiry, 6*, 93-118.

Hall, S. E. K., & Geher, G. (2003). Behavioral and personality characteristics of children with reactive attachment disorder. *The Journal of Psychology, 137* (2), 145-162.

Harlow, H. F., & Harlow, M. K. (1965). The affectational systems. In A. M. Schrier, H. F. Harlow, & F. Stollnitz (Eds.), *Behavior of nonhuman primates, Volume II.* New York: Academic Press.

Hirshberg, L. M., Chiu, S., & Frazier, J. A. (2005). Emerging brain-based interventions for children and adolescents: Overview and clinical perspective. *Child and Adolescent Psychiatric Clinics of North America, 14*, 1-19.

Ito, Y., Teicher, M., Glod, C., & Ackerman, E. (1998). Preliminary evidence for aberrant cortical development in abused children: A quantitative EEG study. *Journal of Neuropsychiatry and Clinical Neurosciences, 10*, 298-307.

Iwaniec, D. (1995). *The emotionally abused and neglected child: Identification, assessment, and intervention.* West Sussex, England: John Wiley & Sons, Ltd.

Jarusiewics, B. (2002). Efficacy of neurofeedback for children in the autistic spectrum. *Journal of Neurotherapy, 6* (4), 39-49.

Jasper, H. (1958). The ten-twenty electrode system of the International Federation. *EEG and Clinical Neurophysiology, 10*, 371-375.

John, E. R., Prichep, L. S., & Easton, P. (1987). Normative data banks and neurometrics: Basic concepts, methods and results of norm constructions. In A. S. Gevins & A Remond (Eds.), *Handbook of electroencephalography and clinical neurophysiology* (Vol. 1). Amsterdam: Elsevier Publishing Co.

Kaufman, J., & Zigler, E. (1989). The intergenerational transmission of child abuse. In D. Cicchetti & V. Calson (Eds.), *Child maltreatment: Theory and research on the causes and consequences of child abuse and neglect.* New York: Cambridge University Press.

Kopp, C. B. (1989). Regulation of distress and negative emotions: A developmental view. *Developmental Psychology, 25*, 343-354.

LeDoux, J. (1996). *The emotional brain.* New York: Simon & Schuster.

Linden, M., Habib, T., & Radojevic, V. (1996). A controlled study of the effect of EEG biofeedback on cognition and behavior of children with attention deficit disorder and learning disabilities. *Biofeedback and Self-Regulation, 21* (1), 35-49.

Lubar, J. F. (1997). Neocortical dynamics: Implications for understanding the role of neurofeedback and related techniques for the enhancement of attention. *Applied Psychophysiology and Biofeedback, 22* (2), 111-126.

Lubar, J. F., Smartwood, M. O., Smartwood, J. N., & O'Donnell, P. H. (1995). Evaluation of the effectiveness of EEG neurofeedback training for ADHD in a clinical setting as measured by changes in T.O.V.A. scores, behavioral ratings, and WISC-R performance. *Biofeedback and Self-Regulation, 20* (1), 83-99.

Manassis, K., & Bradley, S. J. (1994). The development of childhood anxiety disorders: Toward and integrated model. *Journal of Applied Developmental Psychology, 15*, 345-356.

Meaney, M. J., Aitken, D. H., van Berkel, C., Bhatnagar, S., & Sapolsky, R. M. (1988). Effect of neonatal handling of age-related impairments associated with the hippocampus. *Science, 239*, 766-768.

Moghaddam, B., Bolinao, M. L., Syein-Behrens, B., & Sapolsky, R. (1994). Gluccocorticoids mediate the stress-induced extracellular accumulation of glutamate. *Brain Research, 655*, 251-254.

Monastra, V. J., Monastra, D. M., & George, S. (2002). The effects of stimulant therapy, EEG biofeedback, and parenting style on the primary symptoms of attention-deficit/hyperactivity disorder. *Applied Psychophysiology and Biofeedback, 27* (4), 231-249.

Othmer, S., Othmer, S. F., & Kaiser, D. A. (1999). EEG biofeedback: An emerging model for its global efficacy. In J.R. Evans & A. Abarbanel (Eds.), *Introduction to quantitative EEG and neurofeedback.* San Diego, CA: Academic Press.

Perry, B. D. (1994). Neurobiological sequelae of childhood trauma: PTSD in children. In M. Murberg (Ed.), *Catecholamines in post-traumatic stress disorder.* Washington, DC: American Psychiatric Press.

Perry, B. D., & Pate, J. (1994). Neurodevelopment and the psychobiological roots of post- traumatic stress disorder. In L. Koziol & C. Stout (Eds.), *The neuropsychology of mental illness: A practical guide.* Springfield, IL: Charles C. Thomas.

Perry, B., & Pollard, R. (1998). Homeostasis, stress, trauma, and adaptation: A neurodevelopmental view of childhood trauma. *Child and Adolescent Psychiatric Clinics of North America, 7* (3), 3-51.

Post, R. M., Rubinow, D. R., & Ballenger, J. C. (1984). Conditioning, sensitization and kindling: Implications for the course of affective illness. In R. M. Post & J. C. Ballenger (Eds.), *Neurobiology of mood disorders.* Baltimore, MD: Williams & Wilkins.

Post, R. M., Weiss, R. B., & Leverich, G. S. (1994). Recurrent affective disorder: Roots in developmental neurobiology and illness progression based on changes in gene expression. *Development and Psychopathology, 6*, 781-813.

Punzo, F., & Ludwig, L. (2002). Contact with maternal parent and siblings affects hunting behavior, learning, and central nervous system development in spiderlings of *Hogna carolinensis* (Araeneae: Lycosidae). *Animal Cognition, 5*, 63-70.

Read, J., Perry, B. D., Moskowitz, A., & Connolly, J. (2001). The contribution of early traumatic events to schizophrenia in some patients: A truamagenic neurodevelopmental model. *Psychiatry, 64* (4), 319-345.

Rossiter, T. (2004). The effectiveness of neurofeedback and stimulant drugs in treating AD/HD: Part II. Replication. *Applied Psychophysiology and Biofeedback, 29* (4), 233-243.

Rossiter, T. P. & La Vaque, T. J. (1995). A comparison of EEG biofeedback and psychostimulants in treat-

ing attention deficit/hyperactivity disorders. *Journal of Neurotherapy, 1* (1), 48-59.

Rutter, M., & English and Romanian adoptees study team (1998). Developmental catch-up, and deficit following adoption after severe global early privation. *Journal of Child Psychology and Psychiatry 39*, 465-476.

Sapolsky, R. M. (1993). Potential behavioral modification of glucocorticoid damage to the hippocampus. *Behavioral Brain Research, 57*, 175-182.

Schore, A. N. (1994). *Affect regulation and the origin of self: The neurobiology of emotional development.* Hillsdale, NJ: Lawrence Erlbaum Associates.

Schore, A. N. (2001). The effects of relational trauma on right brain development, affect regulation, and infant mental health. *Infant Mental Health Journal, 22*, 201-269.

Sgoifo, A., Koolhaas, J., & De Boor, S. (1999). Social stress, autonomic neural activation, and cardiac activity in rats. *Neuroscience and Biobehavioral Reviews, 23*, 915-923.

Sichel, A. G., Fehmi, L. G., & Goldstein, D. M. (1995). Positive outcome with neurofeedback treatment of a case of mild autism. *Journal of Neurotherapy, 1* (1), 60-64.

Siniatchkin, M., Hierundar, A., Kropp, P., Kuhnert, R., Gerber, W. D., & Stephani, U. (2000). Self-regulation of slow cortical potentials in children with migraine: An exploratory study. *Applied Psychophysiology und Biofeedback, 25* (1), 13-32.

Sterman, B. (1996). Physiological origins and functional correlates of EEG rhythmic activities: Implications for self-regulation. *Biofeedback and Self-Regulation, 21*, 3-33.

Tansey, M. A. (1992). EEG sensorimotor biofeedback training and the treatment of a six-year old asthmatic child. *American Journal of Clinical Biofeedback, 5* (2), 145-149.

Teicher, M. H., Ito, Y., Glod, C. A., Anderson, S. L., Dumont, N., & Ackerman, E. (1997). Preliminary evidence for abnormal cortical development in physically and sexually abused children using EEG coherence and MRI. *Annals of the New York Academy of Sciences, 821*, 160-175.

Thatcher, R. W. (1998). Normative EEG databases and EEG biofeedback. *Journal of Neurotherapy, 2* (4), 8-39.

Thompson, M., & Thompson, L. (2003). *The neurofeedback book: An introduction to basic concepts in applied psychophysiology.* Wheat Ridge, CO: Association for Applied Psychophysiology and Biofeedback.

Van Bloem, L. L. (2000). Quantitative encephalograms (QEEG) in children with reactive attachment disorder. *Journal of Neurotherapy, 4* (4), 106-107.

van der Kolk, B., & Greenberg, M. S. (1987). The psychobiology of the trauma response: Hyperarousal, constriction, and addiction to traumatic reexposure. In van der Kolk (Ed.), *Psychological trauma.* Washington DC: American Psychiatric Press.

Watanabe, Y., Gould, E., & McEwen, B.S. (1992). Stress induces atrophy of apical dendrites of hippocampal CA3 pyramidal neurons. *Brain Research, 588*, 341-345.

Wilens, T. E. (2004). *Straight talk about psychiatric medications for kids.* New York: Guilford Press.

Wilson, S. L. (2001). Attachment disorders: Review and current status. *The Journal of Psychology, 135* (1), 37-51.

RECEIVED: 01/26/06
REVISED: 05/22/06
ACCEPTED: 07/19/06

doi:10.1300/J184v10n04_02