

# EEG Neurofeedback for Treating Psychiatric Disorders

Alondra Oubré, Ph.D.

Neurofeedback, also called electroencephalogram (EEG) biofeedback or neurotherapy, is an adjunctive treatment used for psychiatric conditions such as attention-deficit/hyperactivity disorder, generalized anxiety disorder, posttraumatic stress disorder, phobic disorder, obsessive-compulsive disorder, bipolar disorder, depression and affective disorders, autism, and addictive disorders (Moore, 2000; Rosenfeld, 2000; Trudeau, 2000).

In an interview with *Psychiatric Times*, Siegfried Othmer, Ph.D., chief scientist at EEG Spectrum International Inc., described neurofeedback as neuroregulation in the time and frequency domains through the use of bioelectrical operant conditioning. Like repetitive transcranial magnetic stimulation (rTMS), neurofeedback is an innovative form of electrotherapeutics that complements neurochemical interventions for mood disorders. "With the use of anticonvulsants as mood stabilizers," Othmer said, "we have seen a

convergence of psychiatry and neurology in the field of pharmacology. Similarly, neurofeedback signals a convergence of psychiatry and neurology in bioelectrical approaches to treating affective disorders. By stabilizing the brain and rewarding it for holding particular states, neurofeedback acts as a natural anticonvulsant." The rationale for using neurofeedback therapeutically is that it corrects deficits in brain cerebral regulatory function related to arousal, attention, vigilance and affect (Othmer et al., 1999).

During neurofeedback sessions, patients learn to produce desirable brain wave patterns displayed on a computer screen by controlling the activity of a computerized game or task seen on a second screen. Increases in the amplitude of slow spindle activity are instantaneously rewarded. The reward corresponds to the earned score, similar to scores accumulated in a computer game (Othmer, 1999).

Neurofeedback represents a window of opportunity for assessing and shifting any given brain state (Manchester et al., 1998). The designated frequency band determines which brain state is rewarded (Othmer, 1999). Beta (15 Hz to 18 Hz) training usually produces a slightly upward shift in arousal levels, leading to increased wakefulness and attentiveness or to decreased depression. The sensorimotor rhythm (SMR) (12 Hz to 15 Hz) elicits a slightly downward shift in arousal. The SMR is associated with subjective feelings of relaxation, emotional calm and centeredness (Othmer, 1999). Combined left-side,  $\beta$ -SMR and right-side - neurofeedback is often used to treat brain wave dysregulation associated with traumatic memories. Right-side training is also employed for social and emotional deficits such as conduct disorder,

autism and reactive attachment disorder (Othmer, 2000; Othmer et al., 1999).

## **Assessment of Clinical Evidence**

The efficacy of neurofeedback in the treatment of seizure and pseudoseizure disorders has been well documented in peer-reviewed literature for over 25 years (Lubar, 1997; Swingle, 1998). On the whole, however, clinical support for the effects of neurotherapy is limited and based primarily on case studies, rather than randomized, controlled, blinded studies. While Joel Lubar, Ph.D., professor of psychology at University of Tennessee in Knoxville, recognizes the shortage of randomized trials on neurofeedback, he told *PT* that matched-group studies conducted in accordance with the Declaration of Helsinki are more appropriate than controlled trials for studying hyperactivity. He noted that 1,500 groups worldwide currently use neurofeedback for psychiatric applications, including attention-deficit/hyperactivity disorder (ADHD) and comorbidities. Since the 1970s, his team has investigated various interventions for treating hyperactivity in children and found EEG to be superior.

Lubar and his colleagues (1995) evaluated the effects of neurofeedback treatment on ADHD in 19 youth, ages 8 years to 19 years, under relatively controlled conditions. The subjects received one-hour sessions of  $\beta$  brain wave training daily for up to 40 hours over a two- to three-month period. The goal of the therapy was to increase 16 Hz to 20 Hz ( $\beta$ ) activity while reducing the amplitude of brain waves (4 Hz to 8 Hz). Compared to pre-training results, post-training changes showed improvements in Test of Variables of Attention (TOVA) scores, Attention Deficit Disorders Evaluation Scale (ADDES) behavior ratings and Weschler Intelligence Scale for

Children-Revised (WISC-R) performance. Twelve out of 18 subjects with pre-/post-TOVA scores had EEG-responsive improvements on an average of three of four possible scales. This change was comparable to pre-/post-medication differences in TOVA scores in youth with ADHD.

While TOVA scores typically return to baseline when the effects of pharmacotherapy wear off, the TOVA scores of the EEG-responsive subjects remained at the improved level. Significant post-test increases in IQ scores were observed in 10 EEG-responsive subjects who had been tested on the WISC-R two years earlier. Parental and teacher ratings of the children's behavior also improved following neurofeedback training. Thus, in the EEG-responsive youth, behavioral improvements corresponded with increased scores on TOVA and WISC-R. Lubar and his associates cautiously concluded that EEG neurofeedback training is a powerful adjunctive technique for treating ADHD when used as part of a multi-component therapeutic approach.

Additional research suggests that EEG neurofeedback may be an effective alternative to psychostimulants in the treatment of ADHD if medication is ineffective or has adverse effects or if patients are noncompliant (Rossiter and La Vaque, 1995). In one case study, a 36-year-old female diagnosed with ADHD, temporal seizure disorder and borderline personality disorder received 30 weekly sessions of SMR neurofeedback training and carbamazepine (Tegretol) (Hansen et al., 1996). The patient initially was reluctant to take carbamazepine but became compliant after starting neurofeedback training. However, because of the drug's side effects, she stopped, restarted and then again discontinued her medication. Following 17 sessions of neurofeedback, her quantitative EEG (QEEG) showed relative powers within normal ranges. Carbamazepine increased the favorable effect of neurofeedback on TOVA performance in the early phase of treatment. Although the subject's TOVA scores

fluctuated as she went on and off carbamazepine, all four scales were normal months after she ceased taking carbamazepine. At that time, her TOVA performance showed no evidence of attentional deficit.

In a survey, 36 children, ages 6 years to 17 years, receiving EEG neurofeedback as a treatment for attention-deficit disorder (ADD)/ADHD were evaluated for changes in both subjective and objective clinical parameters (Alhambra et al., 1995). After 20 sessions, subjective improvement based on parental observations was 86%. In objective assessments, the overall improvement was 74% for TOVA score and 78% for favorable changes in QEEG parameters. Over a 12-month period, neurofeedback was associated with either a decrease or termination of pharmacotherapy in 16 of 24 patients receiving medication for ADD/ADHD.

In a retrospective study, 11 females, ages 12 years to 21 years, diagnosed with dissociative identity disorder (DID) received 30 neurofeedback and 10 group sessions (Manchester et al., 1998). The treatment was designed to increase prefrontal  $\beta$  activity for alertness and simultaneously enhance activity associated with a reverie state. The combined increase of  $\beta$  and brain waves allowed patients to re-experience their traumatic memories while in a hypnagogic reverie state but free of the distortions that arise during dreaming or hypnosis. The ratio of  $\alpha$  to  $\beta$  activity is crucial in this type of training. If  $\alpha$  activity becomes too high, patients may sink into an unconscious state and not remember their past experiences. Three to 27 months following neurofeedback training, the post-treatment score for the DID group was 82, falling within the range of normal values. By bringing dissociated information, affect and sensation into

consciousness, neurofeedback training helped subjects to resolve conflicts that contributed to their dissociative defense symptoms. Neurofeedback resulted in favorable changes between pre- and post-treatment scores on the Minnesota Multiphasic Personality Inventory-2 (MMPI-2) in a 65-year-old woman diagnosed with a major depressive disorder and in a 42-year-old woman with chronic psychological maladjustment (Baehr et al., 1997). The researchers concluded that even though EEG asymmetry training is not an efficacious stand-alone therapy for depression, it is an effective adjunct to psychotherapy for treating certain mood disorders. Certain neurofeedback protocols may be beneficial for treating anxiety disorders (Moore, 2000), but the success of particular neurofeedback protocols for anxiety may depend on which diagnostic categories are used (Thomas and Sattlberger, 1997). Case studies on the effects of neurofeedback on bipolar disorder (BD) have produced mixed results. Although Rosenfeld (2000) was unsuccessful in treating two patients with BD using a neurofeedback protocol, Othmer (2001) found neurofeedback to be effective in managing mood swings in pediatric patients with BD when combined with pharmacotherapy and psychotherapy. In the Othmer case studies, neurofeedback protocols that directly affect inter-hemispheric communication were most efficacious for children diagnosed with BD.

In addition, EEG neurofeedback may have limited applicability for treating psychotic symptoms. Researchers successfully used neurofeedback to modulate slow potentials in schizophrenic and schizotypal subjects in the subacute phase (Gruzelier, 2000). And several studies show that neurofeedback is efficacious for long-term recovery in substance abusers (Kaiser et al., 1999; Trudeau, 2000).

## **Future Directions**

Despite positive evidence from case studies, Russell A. Barkley, Ph.D., professor of psychiatry and neurology at University of Massachusetts Medical School, disputes claims that EEG neurofeedback has an effect on ADHD. Barkley told *PT* that EEG neurofeedback is not supported by evidence-based medicine. "One chief problem," he warned, "is that pre- and post-changes occur in subjects with ADHD regardless of whether or not they receive neurofeedback." Barkley attributed reported improvements in objective measures of ADHD symptoms (such as parent and teacher rating scales of disruptive behavior) to the practice effect. "Because of the lack of adequately designed studies, any effects associated with EEG neurofeedback may be due to the placebo response," Barkley said.

However, Lubar et al's. 1995 study provided comparative pre- and post-treatment measurements of several parameters in subjects with ADHD who improved and in those who did not. As noted, the pre-/post-changes observed in the neurofeedback-responsive treatment group were nearly equivalent to changes reported for pre-/post-medication in subjects with ADHD. Other studies comparing the effects of EEG neurofeedback and psychostimulants reveal that neurofeedback produces post-treatment changes equal to those associated with pharmacotherapy (Nash, 2000). Based on these findings, supporters argue that neurofeedback achieves its therapeutic effects by acting on electrophysiological substrates of the brain and not via a placebo response (Othmer et al., 1999).