

## CHAPTER 26

# Neurofeedback Therapy in Clinical Practice

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Neurofeedback is applied neuroscience—it is a new frontier in helping innumerable people who up until now have been condemned to just make the best of feeling chronically fearful, unfocused and disengaged.

*Bessel A. van der Kolk, M.D. (in Fisher 2014, p. xvii)*

**N**eurofeedback, also known as electroencephalogram (EEG) biofeedback or neurotherapy, is a brain wave-based emerging technology used to treat neurodevelopmental disorders, anxiety and mood disorders, addictions, posttraumatic stress disorder (PTSD), traumatic brain injury (TBI), and chronic pain. In addition, neurofeedback has applications for enhancing sports participation and peak performance. In this chapter, we provide historical and technical background, summarize key neurofeedback models, distill salient research findings, highlight selected evidence-based clinical applications, and offer a perspective on future directions. All of the techniques discussed invoke the notion of neuroplasticity, with the goal of improved self-regulation and cognitive function.

## A Brief History

The first description of the human EEG by Hans Berger in the early twentieth century was followed by investigations confirming that classical Pavlovian conditioning could be applied to the EEG in regard to the alpha brain wave band-blocking response. Patients could learn voluntary control over this response (Krodt and Henry 1941). Kamiya (1968) applied learning theory to demonstrate voluntary control via operant conditioning (reward or reinforcement) of the alpha frequency range, 8–12 Hz (Skinner 1953), and the ability of patients to voluntarily control the contingent negative variation or slow cortical potential was confirmed (McAdam et al. 1966). Sterman, applying operant conditioning of the EEG sensorimotor rhythm (12–15 Hz) to alter sleep spindle density and sleep quality, serendipitously discovered an anticonvulsant effect (Sterman et al. 1970; Wyrwicka and Sterman 1968). Sterman's largely unrecognized work eventually led to studies confirming the effectiveness of EEG biofeedback for treating seizure disorders and the recent surge of research interest.

## Models of Neurofeedback

### Traditional Frequency Band Training

Neurofeedback therapy is brain wave–based biofeedback that uses the EEG as the signal to control the feedback. The electrical activity of the brain is recorded via non-invasive sensors (electrodes) attached to the scalp as active, reference, and ground sites. This makes it possible for other sources of ambient stray signals to be separated from the EEG signal. The electrical potentials are digitized and further processed to reflect various characteristics (e.g., frequency, amplitude) that may be used to produce and present feedback information regarding brain activity to patients via auditory, visual, tactile, or other modalities. The objective is to change patterns of brain wave functioning, presumably through learning processes that influence brain activity associated with subjective states and observable symptoms or behaviors. In the earliest days of neurofeedback, this feedback was as simple as a single tone or flashing light that indicated change had occurred. More modern applications use tones, music, movies, other visual displays, and even video games to train people in more engaging and sophisticated ways. Targeted changes in the various displays correspond to targeted changes in aspects of the EEG. The goal is for the brain to learn more adaptive ways of responding.

Early neurofeedback interventions relied on single-channel or single active site recordings and focused primarily on changes in amplitudes of various frequencies. This is accomplished by setting thresholds for reinforcements, either for achieving a certain amplitude level or for remaining below a certain amplitude level. In this manner, increased production of the desired brain wave activity may be “rewarded” at a certain rate to increase further production. Alternatively, specified brain wave frequencies may be “inhibited” by withholding reward if the threshold is exceeded or providing reward when activity goes below threshold. Combinations of reward

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and inhibit thresholds may be used. The reward/inhibit paradigm remains in widespread use.

### Connectivity-Based Developments

The development of multiple-channel site recordings paved the way for brain region connectivity feedback measures. These measures incorporate aspects such as the amount of information shared between sites at a given time (coherence), speed of information transfer at a given time (phase), similarities in amplitudes across times (co-modulation), and extent to which phase and amplitude match (synchrony). These aspects are neither inherently desirable nor undesirable; their utility depends on the nature of the brain activity and the desired goals for brain functioning (Collura 2014).

### Quantitative Electroencephalography

The complexity and flexibility of neurofeedback have been enhanced by the quantitative EEG (qEEG), in which multiple simultaneous site recordings are made (using the entire standard international 10–20 system or more sites) and processed into highly diverse metrics for use in the feedback process. A qEEG typically generates a colored brain map of relative EEG activity; a normative database is used to compare practitioner uses this database to choose a protocol for training, which may be modified over time depending on subjective report and objective data obtained.

Various training paradigms using qEEG information have been devised; some rely on the static image and associated frequency and amplitude information of the EEG, and others are more interactive in real time, such as live z-score training (grounded in normative databases) of any number of EEG metrics in the feedback mechanism. This approach capitalizes on the inherent variability in EEG metrics, as represented in the standard deviation, with presumed greater flexibility for changing the particular patient's EEG metrics in the desired direction (Collura 2014). Another paradigm of particular interest is low-resolution electromagnetic tomographic analysis (LORETA; Pascual-Marqui et al. 2002). Using sophisticated mathematical operations, LORETA analyzes scalp recordings from a full-head EEG, transforms these into three-dimensional voxels, which combine into regions of interest; enables visualization of brain activity images over time that cannot be appreciated in static images; and allows more specific neurofeedback targeting of these regions of interest compared with single specific scalp sites. Advances in technology and software have made this approach more accessible to practitioners.

### Slow Cortical Potentials

Further developments influencing neurofeedback approaches involve slow cortical potentials. Traditional frequency-band training techniques focus largely on distinctions among the various frequency wave bands: delta, 0.5–4 cycles/oscillations/second or Hz; theta, 4–8 Hz; alpha, 8–12 Hz; sensorimotor rhythm, sometimes also referred to as low beta, 12–15 Hz; beta, 15–20 Hz; high beta, 20–30 Hz; and gamma, 35–45 Hz. The slow cortical potentials are generally within the 0.01- to 2-Hz range. They typically do

not demonstrate the kind of rhythmic oscillation seen in the more standard wave bands. Accordingly, they are not as amenable to training with standard reward and inhibit protocols.

Very slow activity involving a negative shift in the EEG in anticipation of an expected event, known as *contingent negative variation*, was first reported (Walter et al. 1964) and shown to be potentially under voluntary control (McAdam et al. 1966) in the 1960s. Variations in the slow cortical potential signal occur with activation or deactivation, thereby permitting training in a given direction. In the 1990s, clinical investigations identified the potential for treating a variety of challenging conditions, such as paralyzed or “locked-in” syndromes. This approach enabled patients with amyotrophic lateral sclerosis to express themselves with an electronic spelling device. Subsequently, benefits were shown in attention-deficit/hyperactivity disorder (ADHD), epilepsy, and migraine (Birbaumer 1999; Strchl 2009).

### Infralow-Frequency Training

Infralow-frequency (ILF) training is related to slow cortical potential training in that very slow frequency (below 0.1 Hz) monitoring is involved. At this level, amplitude training and setting thresholds within an operant conditioning (reward or reinforcement) paradigm are not possible. Information about higher frequencies is typically removed from the feedback, and only subtle shifts or fluctuations over time, not rhythmic oscillations per se, in the ultralow range are presented. With such feedback, once the connection between the external signal and the internal signal is established, “the brain takes responsibility for the signal and tries to steer it. This is a natural process, like the brain taking charge of the...steering wheel of the car even while the driver’s thoughts are directed elsewhere” (Othmer 2015).

Training entails recording from two channel sites, which are associated with electrode placements corresponding to presumed nodes of resting state networks—specifically, the default mode network, dorsal attention network, and central executive network. By targeting the nodes of these networks, it is conjectured that ILF training reorganizes brain network connectivity (Othmer et al. 2013). Treatment is highly individualized and less able to be captured in standard protocols. Consequently, the overall conceptualization and rapid positive responses reported, even with quite severe psychopathology, are not without controversy (Collura 2014). ILF training also has been advocated to address pediatric problems such as autism spectrum disorder, early childhood emotional and physical trauma, migraine, sleep disturbances, feeding and eating disorders, and drug dependencies before dysfunctional network connectivity has crystallized into more difficult and refractory conditions (Othmer et al. 2013).

### Real-Time Functional Magnetic Resonance

#### Imaging

Soon after the inception of functional magnetic resonance imaging (fMRI), it became apparent that the technology lent itself to applications involving real-time information about changes in blood oxygen level–dependent (BOLD) signal (Weiskopf 2012). Confidence in the BOLD response as an indirect measure of brain activity, the patient

tal for enhanced spatial resolution of specific regions of interest, and the strong association of the BOLD signal with brain electrical signals gave impetus for studying real-time fMRI responses with conditioning principles to change brain activation patterns. This provides another avenue for influencing the connectivity of brain networks (Ruiz et al. 2014). One major drawback is the expense associated with imaging equipment. ILF training has been offered as a more economical alternative, given that fluctuations posited in ILF training also correspond to activation of core networks.

### Standard Protocols

Collura (2014) described standard protocols that have been modified over three decades of clinical practice. Table 26-1 presents an example of the key components of protocols derived from Collura’s work and their clinical applications. Numerous other systems and protocols have been developed and are being used effectively (see Chapter 3, “Complementary and Integrative Medicine in Child and Adolescent Psychiatric Disorders”).

It is important to remember that standard protocols can have modifications with combinations of multiple inhibits, multiple thresholds, overlapping bands, or other features. Variations also occur with multiple channels and even multiple persons involved in the same training protocol. Furthermore, a myriad of applications use neurofeedback for optimal or peak performance, such as for the enhancement of athletic and other aspects of emotional control and stability under pressure. These protocols commonly use elements of sensorimotor rhythm, alpha, and/or Squash protocol training with other procedures. The potential for so many applications is accelerating neurofeedback research.

## Evidence Base and Clinical Applications

In order to understand the breadth of neurofeedback applications, it is important to keep in mind that the EEG is an epiphenomenon of neurochemical brain activity that correlates with (but does not necessarily cause) regulation and dysregulation of brain function. Consistent with the conceptualization in the proposed National Institute of Mental Health Research Domain Criteria for mental disorders, certain neurophysiological (and other, such as psychosocial) domains may cut across (and interact reciprocally with) current diagnostic categories and the broad range of human experience (Lilienfeld 2014). The fundamental principles of neuroplasticity support the potential for change in brain function. Increasingly sophisticated conceptualizations of the interrelations and mutual integration of brain regulatory networks (e.g., default mode, dorsal attention, central executive) are incorporated into the current zeitgeist for understanding the complexity of brain function (Othmer et al. 2013). The EEG provides a method to view this activity. Shaping the EEG into alternative patterns may be a vehicle through which change can occur. In the following subsections, we review evidence on neurofeedback for selected clinical conditions, some of which have been studied far more than others.

TABLE 26-1. Standard neurofeedback protocols and clinical applications

Protocol	Description	Clinical conditions and benefits	Risks
Alert	Specific activation paradigm: increasing beta is rewarded; theta and high beta are inhibited. Designed for beta deficits. Recorded from International 10-20 system left hemisphere C3 or central vertex Cz sites.	Counter hypoarousal states (e.g., sometimes seen in ADHD); reduced fatigue; improved mood, information-processing speed, and clarity.	Overactivation; may need to be countered by focus procedures.
Focus	Relaxed, but focused, internal sense; SMR is rewarded. Trained primarily at right hemisphere C4 or Cz sites.	Motoric and mental calming with focused attention; ADHD; anxiety disorders.	Excessive underactivation; may be countered with brief beta training.
Peak	Two-channel setup combining aspects of alert (left hemisphere C3 or Cz) and focus (right hemisphere C4 or Cz) protocols.	Optimal/peak performance (e.g., sports, music, business, or other decision making; enhanced emotional control; and stability under pressure).	Imbalance of overactivation and underactivation; may require relative titration.
Squash	"Family of designs" to reduce amplitude. Down-training via reward when specified band amplitude is below set value. "Bench press" workout analogy: repetitive process of modest effort exerted to make signal go down, which is rewarded, then followed by brief relaxation. Variable sensor placements depending on desired effect.	Optimal mental fitness/sharpness and improved mood.	Working too hard to achieve desired effect may backfire and require reorientation to enhance sense of flow with attentiveness or sense of release and letting go.
Relax	Specific alpha training with theta and high beta inhibition. Posterior sensor placements often preferred.	Lower hyperarousal, relaxation; improved mood; reduced headaches, insomnia, anxiety, and depression.	Adverse mood reactions (more common with frontal placements).

TABLE 26-1. Standard neurofeedback protocols and clinical applications (continued)

Protocol	Description	Clinical conditions and benefits	Risks
Deep	Specific kind of alpha/theta training. Alpha initially rewarded with transition to increased theta reward. Augmented with therapeutic suggestions, imagery, or visualization. "Crossover" eventually occurs with transition from alpha-dominant to theta-dominant; concomitant hypnagogic state facilitates "deep" processing of issues or other personal exploration. Strong therapeutic abreactions can occur. Posterior sensor placements are typical.	Relief from posttraumatic stress responses; reduction of substance abuse; personal exploration.	Abreactions require careful therapeutic monitoring.

Note. Protocols derived from work by Collura (2014). Many other neurofeedback protocols are effective. ADHD=attention-deficit/hyperactivity disorder; SMR=sensorimotor rhythm.

### Attention-Deficit/Hyperactivity Disorder

ADHD has been the condition for which the greatest amount of investigation in regard to neurofeedback has been conducted. Monastra et al. (2005) presented the first review of case and controlled group studies examining the effects of EEG biofeedback on ADHD symptom dimensions and related measures. They determined EEG biofeedback to be "probably efficacious" on the basis of guidelines jointly established by the Association for Applied Psychophysiology and Biofeedback (AAPB) and the Society for Neuronal Regulation (now the International Society for Neurofeedback and Research [ISNR]) and in effect at the time. Subsequently, Arns et al. (2009) provided meta-analytic data (10 prospective controlled studies, total  $N=476$ ; 5 studies with pretest and posttest designs,  $N=718$ ) that showed large effect sizes for neurofeedback on impulsivity and inattention and a medium effect size for hyperactivity. When Arns and colleagues used AAPB and ISNR guidelines for rating treatment efficacy, they considered neurofeedback for ADHD to be "efficacious and specific."

Sonuga-Barke et al. (2013), in their review of eight randomized controlled trials (RCTs; total  $N=273$ ), used criteria some believe to be more stringent and considered the findings to be less conclusive, despite evidence for some reductions in core ADHD symptoms. They made the case for more methodologically sound studies before assigning the highest level of evidence as a first-line treatment for ADHD. A recent meta-analytic update (Mikicoulau-Franchi et al. 2014) in which very stringent criteria were applied, including five randomized semiautic or sham-control neurofeedback studies (total  $N=263$ ), found the strength of effects likely to be dependent on the nature of the outcome rater (e.g., blinded or unblinded). They highlighted particularly notable improvements in the inattentive dimension of ADHD symptoms. Taken together, a consensus emerges that neurofeedback is efficacious to different degrees for dysregulation associated with ADHD, that neurofeedback fits well within an integrative model of treatment for ADHD, and that neurofeedback may enhance response to other concurrent interventions. The conclusion that for ADHD, neurofeedback is probably efficacious and appropriate for integration into more comprehensive treatment has been echoed by other reviews (Covensleben et al. 2012; Holmann et al. 2014; Hurt et al. 2014; Lofthouse et al. 2012; Moriyama et al. 2012).

### Substance Use Disorders

Substance use disorders (SUDs) are remarkably difficult to treat. Various substances of abuse affect the EEG and evoked response potentials differently. EEG alterations are further influenced by the extensive comorbidity (e.g., ADHD, depression, anxiety, neurotoxicity, posttraumatic stress, TB) associated with SUDs. Within the neurofeedback literature on SUDs, one particular protocol (and a specific modification of it) has garnered a notable degree of qualified support. This approach includes a key element of alpha/theta ("deep") training that was originally reported by Green et al. (1974). It was further developed and popularized by Peniston in a series of reports on incorporating alpha/theta training into more comprehensive treatment packages (Peniston and Kulakosky 1989, 1990).

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The basic "Peniston protocol" involves a preconditioning relaxation process comprising at least five initial sessions of thermal (autonomic) biofeedback training augmented with autogenic phrases and abdominal breathing to induce relaxation and quieting of the mind, and an induction script is read at the beginning of each session. After patients are able to consistently sustain hand warming to at least 94 degrees, they are instructed in the EEG biofeedback aspects of the intervention. EEG activity is recorded from occipital sites (typically O1), and with the patient's eyes closed, the therapist gives suggestions for relaxation and sometimes instructions to construct visualizations of specific personally relevant scenes and ultimately to "sink down" into a (theta) state of reverie as the mind is kept quiet and alert and the body calm. A quiet command, "Do it," initiates the process. As threshold criteria are met for alpha production, a tone sounds, and the patient becomes progressively more relaxed as he or she produces more of this brain wave activity. When threshold criteria for the theta lters a hypnagogic state of more freely accessible memories and reverie. The transition in reduction of alpha to greater preponderance of theta may signify "crossover" reactions, including strong therapeutic abstractions. Subsequently, in the relaxed and suggestible (or abreactive) state, the experience is discussed with the therapist. This can be even more beneficial when the clinical picture includes comorbid posttraumatic stress features.

Although several studies have supported the efficacy of this integrated approach with specific alpha/theta training, individuals addicted to certain substances, particularly the stimulants (e.g., methamphetamine, cocaine), may have neurophysiological effects that preclude effective use of the prototypical Peniston protocol. This may be exacerbated if comorbid ADHD features are present. Accordingly, Scott and Kaiser (1998) proposed a modification of the Peniston protocol with an initial course of attentional training—beta or sensorimotor rhythm augmentation with theta suppression—before proceeding to alpha/theta training. Their own research and subsequent research by others (Burkett et al. 2005) with homeless crack cocaine users have supported the utility of this modification.

Given the complex comorbidity associated with SUDs, the role of EEG in guiding more specifically targeted neurofeedback modifications also has been suggested (Sokhadze et al. 2008). Alpha/theta training is likely to be most effective when integrated into more comprehensive treatment programs that could include motivational interviewing, cognitive-behavioral therapy, medication management, and diet modifications. The most thorough review to date of published clinical studies evaluated with the efficacy criteria of the AAPB and ISNR concluded that "alpha theta training—either alone for alcoholism or in combination with residential treatment programs, and mixed substance abuse and combined with residential treatment programs, is probably efficacious" (Sokhadze et al. 2008, p. 1). Slow cortical potential neurofeedback also has been reported to be potentially as efficacious as alpha/theta training in individuals with alcoholism (Schneider et al. 1993). Simple reliance on a routine protocol is ill advised. As in all fields of medicine, clinical judgment is critical at each step along the treatment trajectory, and alterations in approach may be required to optimize outcomes.

### Posttraumatic Stress Disorder

The literature on neurofeedback treatment of posttraumatic stress has unfolded in tandem with that for SUDs. The pioneering work of Peniston with alpha/theta training for addictions was conducted with Vietnam War veterans who often presented with comorbid alcoholism and PTSD. The procedures adopted for these cases mirrored those described earlier for SUDs (Peniston et al. 1993). The crossover effect, associated with frequent abstractions experienced by veterans undergoing this neurofeedback training suggested that this kind of "deep" protocol could be potentially advantageous. It is important to keep in mind that the reexperiencing of posttraumatic memories and the associated intense affective-emotional responses and processing with the therapist are reminiscent of other evidence-based therapies such as cognitive processing therapies for PTSD. Accordingly, alpha/theta training may have a role as an adjunctive, if not always first-line, treatment. Additional comorbidities may complicate the picture and require clinical judgment regarding further modifications.

### Traumatic Brain Injury

The diverse sequelae of TBI, including attentional and memory difficulties, executive control dysfunctions, headaches, fatigue, sleep disturbances, and emotional overactivity, suggest the need for specifically targeted neurophysiological interventions. Various EEG-based approaches have been explored (Thornton and Carmody 2008), including a standard qEEG approach that incorporates aspects of protocols that have been used with ADHD and involve increasing beta and decreasing theta along the sensorimotor strip (central active sites of C3, Cz, and C4). Customized procedures also can be used to compare the patient's resting eyes-closed qEEG with a reference database. An activation database-guided qEEG biofeedback approach also may be used while patients actively engage in specific cognitive tasks. With an empirically derived normative database from individuals with no known history of TBI, a variety of metrics can be used, often including a combination of variables relevant to a specific cognitive task, and thereby can address the many bothersome symptoms within a broader rehabilitation program. For example, the most substantiated benefits were observed in tasks involving auditory memory. A review (May et al. 2013) of a broader range of neurofeedback approaches contained in 14 case reports (single or series) and 8 well-designed prospective cohort investigations found consistent evidence for statistically and clinically significant subjective and objective improvements, especially in attention, impulse control, executive functioning, processing speed, and general cognition. Hence, neurofeedback is a "promising treatment" that "warrants further investigation" as an adjunctive treatment for TBI (May et al. 2013, pp. 289, 295).

### Chronic Pain

Several studies have used neurofeedback for chronic pain syndromes; for example, Blanchard et al. (1997) found biofeedback to suppress EEG alpha activity to be as effective for significantly reducing headaches as temperature (hand-warming) biofeedback. Open-label studies in children and adults for treatment of migraine have been encouraging (Sinatchkin et al. 2000; Walker 2011).

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In an RCT of patients with fibromyalgia syndrome, Kayiran et al. (2010) found significant improvements in pain, psychological symptoms, and quality of life among the group receiving neurofeedback. Caro and Winter (2011) reported that among patients with fibromyalgia, those receiving neurofeedback showed improvements in visual attention parameters, tenderness, pain, and fatigue. Given that reviews have found inconsistent results, research is needed to identify the most beneficial neurofeedback protocols for treating fibromyalgia (Gliombiewski et al. 2013; Lauche et al. 2015).

Neurofeedback also has been explored for treatment of spinal cord injury-related and other neuropathic pain. Hassan et al. (2015) found immediate and longer-term reduction of central neuropathic pain corresponding to measurable short- and long-term modulation of cortical activity in a small case series of patients with paraplegia with central neuropathic pain. Jensen et al. (2013) have taken a systematic approach toward developing potential EEG biofeedback treatments for chronic pain. They used different neurofeedback protocols in a small series of individuals with spinal cord injury pain and found similar reductions in pain intensity that continued at 3-month follow-up. Further investigations of the efficacy and mechanisms of noninvasive neuromodulatory treatments suggest that neurofeedback can reduce the severity of chronic pain to a moderate extent but with uncertain durability and potential variability across heterogeneous pain conditions (Jensen et al. 2013). However, Jensen et al. (2013) suggested that neurofeedback may act synergistically by priming the central nervous system to be more responsive to other neuromodulatory or psychosocial treatments.

Further developments may be enhanced by capitalizing on evidence that neuropathic and musculoskeletal pain are associated with substantial reorganization of various brain regions, that this reorganization increases with chronicity, and that it is associated with the magnitude of pain experienced (Moseley and Flor 2012). Accordingly, cortical plasticity may be targeted for neurotherapeutic approaches. This may be even more effective when applied in new paradigms based on real-time MRI, which allows for more fine-grained effect on functional connectivity and spatial-temporal patterns of brain activity (Ruiz et al. 2014). Initial investigations have been promising but are not economically feasible at present in most clinical settings (Chapin et al. 2012). Alternatively, less expensive methods of brain imaging, such as near-infrared spectroscopy, could facilitate translation of these principles into practice (Chapin et al. 2012). Moreover, real-time fMRI and other brain imaging techniques are applicable to neuropsychiatric disorders and symptoms other than pain (Weiskopf 2012).

### Neurofeedback Treatments in Other Disorders or Conditions

Reviews of other clinical conditions suggest potential benefits with neurofeedback, but these are often amalgams of single case studies, case series, or comparison groups. Overall, the findings provide encouragement to adapt neurofeedback interventions to anxiety disorders, obsessive-compulsive phenomena, depressive disorder, learning disabilities, autism spectrum disorder, epilepsy or seizure disorders, and others (Hurt et al. 2014; Simkin et al. 2014; Tan et al. 2009). Therapeutic benefits



for some of the most challenging and severe presentations (e.g., bipolar disorder, nightmares, night terrors, sleepwalking, restless legs syndrome, pain medication dependency) are being reported for newly emerging LIF training, which, unfortunately, does not lend itself readily to investigation with standardized clinical trial protocols (Othmer et al. 2013).

### Voluntary Versus Involuntary or Passive Control

In addition to neurofeedback techniques in which the patient exerts volitional control while learning to change the EEG patterns, other approaches involve the patient remaining relatively passive while the EEG signal controls the feedback, such that the therapeutic effects occur without volitional effort. For example, studies of audio and visual (e.g., photic) stimulation of the EEG date back as far as the more operant-based learning procedures discussed earlier (Collura 2014). Involuntary or passive approaches use the properties of the EEG to determine the nature of the feedback signal on a continuously interactive basis as the EEG is sampled; essentially, then, these are EEG-driven procedures that include changes to aid in altering dysfunctions reflected in the EEG. Our own work (D. V.N. and M. L. E.) includes EEG-driven (photic and mildly pulsed, subliminal electromagnetic) stimulation to treat fibromyalgia symptoms, mixed TBI and PTSD, headaches, and attentional problems (Muehler et al. 2001; Nelson and Eszy 2011, 2012, 2015a, 2015b; Nelson et al. 2010; Schoenberger et al. 2001). Our impression is that these procedures sometimes work well as stand-alone interventions but may work even better when integrated into a more comprehensive treatment package. They also may work in conjunction with techniques that involve voluntary control. Moreover, consistent with some of the findings for operant-based procedures noted previously, these more passive or involuntary techniques prime the nervous system to be more responsive to other interventions, including those to which patients' symptoms were previously refractory, such as psychotherapy, cognitive-behavioral therapy, and cognitive rehabilitation. Also, our impression is that these techniques fit well within the concept of neurofeedback affecting connectivity of brain networks. Only more rigorous controlled clinical trials involving sequencing of multiple interventions will more definitively determine their relative utility and the validity of effects produced by underlying mechanisms.

### Clinical Case: Neurofeedback for Traumatic Brain Injury, Posttraumatic Stress Disorder, and Substance Abuse in an Afghanisthan/Iraq War Veteran

David served 6 years in the army, with three deployments in combat zones in Afghanistan and a fourth in Iraq. Three concussions plus numerous blast caused TBI, with frequent severe headaches and hypersensitivity to light and noise. Multiple strapped wounds and other injuries caused chronic pain. David developed PTSD with insomnia, flashbacks to scenes of his buddies bleeding to death, hypervigilance, and intense anxiety, which made it impossible for him to be in crowds, on busy streets, or on public transportation. Heavy drinking and prescription drug abuse contributed to explosive behavior and four assault charges. During a panic attack on the firing range, David

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could not pull the trigger. This led to admission to a medical center. After a suicide attempt there, he transferred to the inpatient psychiatric ward at a tertiary care center, where his diagnosis included mild TBI; adjustment disorder with mixed anxiety and depressed mood; PTSD; major depressive disorder, recurrent, with psychotic features; bipolar disorder, manic, with psychotic features; borderline personality disorder; alcohol illness prior to the second deployment; and prescription drug abuse. He had no history of men- abuse track and was given PTSD treatments. Medications generally made him feel worse. Trials of paroxetine, venlafaxine, trazodone, lorazepam, diphenhydramine, zolpidem, and quetiapine were ineffective. David was maintained on nortriptyline and hydroxyzine, even though he experienced only minimal benefit.

David recalled, "All traditional treatments failed. To call my prognosis unhelpful was an understatement. I was aggressive, paranoid, angry, depressed, and nihilistic, unable to focus enough to take part in conversations, unable to read more than a few sentences. Sleep was an hour or two in 48 hours. I left my room only when forced to for appointments, or (rarely) for food. Drug abuse and violent behavior continued, and I was still suicidal."

David was referred for neurofeedback. A modification of the Flexyx Neurotherapy System (FNS; Flexyx LLC, Walnut Creek, California) was used. FNS involves monitoring the EEG with a two-channel module that has on-board feedback-generating power; it uses proprietary software to link the digital brain wave recording module through the netic stimulation. The system returns a signal to the participant via conduction from the module, varying as a function of the detectable peak EEG frequency (but offset from it), thereby permitting strategic distortion of the EEG. The amount of electromagnetic stimulation was standardized, with the feedback frequency being offset from the dominant EEG frequency at +20 Hz. Pulses of electromagnetic energy operated at a duty cycle of 1% of the maximum permissible on-time for each pulse; they were powered no more than 1% of the time (e.g., the maximum on-time for 1% for 1-Hz pulse was 0.01 seconds). Testing indicated a power level of 100 pW through the sensor cable.

David attended approximately two or three neurofeedback sessions per week. He sat comfortably, eyes closed, engaged in no specific activity. Electrodes were placed over all areas of the cortex over the course of 25 half-hour sessions. Each session included a total of 4-second stimulation spaced over 4 minutes. Stimulation was not immediately discernible. No adverse reactions occurred. David was not asked to discuss past traumas as part of the procedure. After six sessions, his headaches, anxiety, and depressive behaviors subsided. Hydroxyzine was discontinued. Ongoing treatment led to substantial improvements in insomnia, nightmares, anger, suicidal thoughts, fatigue, hypersensitivities, flashbacks, and abuse of cigarettes, alcohol, and drugs. After 25 neurofeedback treatments, David said, "Now I remember so much. I'm not numb. I have a searing pain and anger. It feels good to talk." Before neurofeedback, he had no idea what he wanted to do after discharge. After neurofeedback, he wrote, "I want to help other veterans who find themselves in situations similar to mine." David enrolled in college, graduated with honors, and is now in a doctoral program.

### How to Find a Qualified Neurofeedback Therapist

Neurofeedback is not the province of any one discipline. Sophisticated practice of qEEG requires some specialist training. The practice of neurofeedback requires un-

derstanding of brain function and the autonomic nervous system and specific training in neurofeedback techniques. Basic understanding and training can be obtained by physicians, psychiatrists, psychologists, nurses, social workers, and other counselors or therapists who practice within the scope of their education and licensure. Neurofeedback may be the primary focus of a clinician's practice or an adjunct to it. Various certifications are available through professional organizations that provide information about training, literature, practitioner support, and professional meetings. Clinicians wishing to find local practitioners may consult organizations devoted to neurofeedback: the AAPB ([www.aapb.org](http://www.aapb.org)) and the Biofeedback Certification International Alliance (<http://bcia.org>), and ISNR ([www.isnr.org](http://www.isnr.org)).

## Summary and Conclusion

Dysfunction in attentional, affective, and executive control networks, and their integration, is reflected in the EEG. Such dysfunctions may be amenable to differentially targeted neurofeedback interventions that give the brain the opportunity to establish or reestablish more optimal levels of functioning. The potential of neurofeedback as a stand-alone or adjunctive treatment will develop through clinical trials, refinement of protocols, and other research.

## KEY POINTS

- Neurofeedback therapy is brain wave–based biofeedback that enables patients to alter their own electroencephalogram (EEG) signal on the basis of real-time feedback.
- Feedback about brain wave activity is typically provided via one or more sensory modalities and is based on operant and classical conditioning learning theories.
- The EEG signal can be used to set parameters for electromagnetic and other sources of stimulation to alter brain wave functioning while patients remain relatively passive.
- The current evidence base suggests broad transdiagnostic applicability of existing and emerging neurofeedback treatment paradigms and protocols.

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