Clinical Addiction Psychiatry

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Part 3
Chapter
19

Praxis

EEG neurofeedback therapy

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Introduction and foundation

This chapter describes a novel approach to understanding and treating addictions. This approach involves the use of EEG biofeedback (neurofeedback) as a modality for treating addictions. In the case of EEG biofeedback, the data generated the model. It is preferable, however, for an explanation of the model to precede the data. Addictions can result from both psychological and physiological factors. Neurofeedback lies at the nexus between them.

Though it is tempting (and perhaps traditional) to leave the realm of brain behavior entirely to neurophysiology, doing so could omit or minimize some critical elements. Medical science has supplied a great deal of knowledge about structural deficits in brain function; additionally, however, the *functional* domain of brain behavior is crucially relevant. Cognitive neuroscience – which concerns itself with brain function – has only begun to engage the challenge of psychopathology.

Neuronal networks organize behavioral repertoires. These networks exhibit a high level of integration and hierarchical organization. On the operational level, as in all communication networks, timing is crucial. Whenever a system is compelled to operate under tight constraints, there is opportunity for departure from functional integrity.

A key assumption is that psychopathology in general, and addiction in particular, arises from deficits in *network relations*. Recent thinking posits "connectivity deficits" as central to disorders such as schizophrenia, autism, and Alzheimer's disease. These clinical syndromes aside, this model may also help explain less acute or more "characterological" problems.

Pharmacologic interventions may not exhaust all therapeutic options. Neurophysiologic interventions, based on EEG data, afford additional and complementary treatment. Operant conditioning of EEG parameters is known as EEG biofeedback – more recently termed neurofeedback to distinguish it from its predecessor, biofeedback applied to peripheral physiology. Though clinical applications of EEG feedback have been extensive with childhood behavioral disorders, numerous other applications have been reported in clinical research.

Origins of EEG biofeedback for addiction treatment

In 1989, Eugene Peniston published a controlled study using EEG feedback with treatment-resistant Vietnam veterans suffering from alcohol dependence and post-traumatic stress disorder (PTSD) (Peniston and Kulkosky, 1989). The EEG feedback was an adjunct to standard treatment at the time – individual and group psychotherapy. Ten subjects in the treatment group had good outcomes, while control subjects remained alcohol-dependent. Small replication studies appeared in subsequent years; most of these supported the original finding. Peniston also followed up with additional confirming studies (vide infra). One subsequent large-scale controlled study, undertaken at a residential treatment center in Los Angeles, replicated the previous Peniston findings; the neurofeedback intervention was also applied to patients with substance dependencies other than alcohol. Participant outcomes did not depend on the drug of choice; at 1-year follow-up, three out of four subjects maintained sobriety. At 3-year follow-up, most experimental subjects continued to maintain sobriety, while controls continued to revert to pre-treatment status.

Since that time, a small number of addiction treatment programs have incorporated neurofeedback. The lack of more robust growth of the technology in addictions treatment is puzzling. Perhaps this is a result of lack of familiarity with neurofeedback within the addictions treatment community.

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The larger context of addiction

The following comorbid conditions may accompany addictive diseases:

- Anxiety or depressive disorder
- Undetected learning disabilities that diminish self-esteem and coping capacities
- Personality disorder, including compulsive or antisocial behavior
- "Organic" personality disorder concurrent with addictive disease
- Unrecognized traumatic brain injury or psychological trauma that limits functionality.

EEG biofeedback training can influence and modify each of the potential accompanying dysfunctions (each is regulated by particular and accessible brain networks).

In neurofeedback, the key intervention is *self-regulation* of impaired, or disregulated, neuronal systems. Because the technique is non-verbal and targets brain behavior directly, it bypasses the usual (verbal) resistances often encountered in therapeutic practice. Since biofeedback aims for improvements in self-regulatory function, this technique could well be considered the *self-regulation remedy*.

The self-regulation approach is complementary to existing interventions. It is consistent with the recognized need for integrative modalities in addiction treatment. Neurofeedback itself is rarely employed as a stand-alone technique for addictions. Traditional addiction treatments feature gradual reduction of psychopharmacological agents along with psychotherapeutic support, including both psychodynamic and behavioral interventions. Successful neurofeedback may reduce or even eliminate the need for stimulants, antidepressants, mood stabilizers, antipsychotic medications, pain medications, and sleep aids. Neurofeedback training can also, over time, lead to greater insight, enhanced receptivity, and deeper engagement with the therapist. Throughout the process, a certain level of implicit trust is required for the trainee to "admit and permit" behavioral reinforcement; this makes the therapeutic context for this work critically important.

Model of brain function supporting EEG neurofeedback

The discovery during the "decade of the brain" that new neuronal growth does take place in the mature adult brain brings hope that even conditions previously considered intractable may be amenable to recovery. The discovery of neuronal growth in the adult human brain validated the notion of brain plasticity, even for those whose work was oriented entirely on structurally and neuroanatomically based models of chemical dependency. But brain plasticity does not depend upon new neuronal growth. It is an inherent property of neural systems.

Some reports document positive outcome rates of neurofeedback therapy that far exceed the typical 25–33% (1 year) sobriety rates achieved with other interventions. Addictive disease may be a constellation of learned behaviors that can be extinguished if and when the brain is given appropriate cues. Although addictive disease often has a heritable component, this should by no means serve as a justification for poor treatment response.

The following areas are critical to an understanding of neurofeedback's mechanism of action:

1. Architecture of neuronal networks
2. Organization of network functions
3. Dynamic management of states.

Architecture of neuronal networks

The human brain is perhaps one of the best examples of what has come to be known as a "small-world" model of networks. Both local and "global" connectivity characterize these networks. In the human brain, both local and global connectivity are quite extensive. Brain-based neurons that manage more distant communications are the pyramidal cells; these receive inputs locally through some 1000 to 10 000 dendritic connections. When dendritic inputs exceed threshold criteria, the neuron generates an action potential, a brief electrical transient capable of signal propagation. Action potentials progress the length of the axon; the axon itself ramifies many times and thus also distributes its signal to many nodes in the zone of the dendritic tree. In addition to this local network, each pyramidal cell also sends its axon to distal cortical regions via the white matter. Synaptic junctions can receive signals from any other synaptic junctions within typically as few as three synaptic links. We are wired in a way that marvelously maximizes and makes accessible all the global connectivity of which we are potentially capable. Cortical loci are "aware" of what is going on in any other part at any time.

Organization of network functions

A second key aspect of the "small-world" model of networks is that of hierarchical organization. In the brain, this hierarchy of control has several levels. At the top rung are the cortical neurons, numbering about ten billion. At the next level are the thalamus and the other subcortical nuclei (visible in medical brain imagery studies). The foundation or base of the hierarchy is the brainstem. Because the brainstem's role is so basic, its functions tend to be taken for granted, much like the heartbeat.

The implication for neurofeedback is this: any attempt to modify brain function communicates with the entire brain and with the entire regulatory hierarchy, all the way to the brainstem. *Neurofeedback reaches every brain level that is managed within this regulatory hierarchy.*

Central regulation of brain function is paramount here. EEG feedback accesses the organization of large-scale synaptic information transport. Fortunately, the rules are again very simple. A single excitatory post-synaptic potential is incapable of generating another action potential in the target neuron; there must be a co-conspirator that arrives within a very narrow time window. Therefore, the operation performed by the neuron, stated mathematically, is that of "coincidence detection." This process imposes very narrow timing constraints on all synaptic information transport since the "opportunity window" in this process is only about 10 milliseconds. The potential for functional deterioration of this transmission is obvious.

One role of the neuron is that of a correlation detector. Consider, however, that brain function cannot depend upon the functionality of each and every individual neuron. Information is instead encoded in neuronal assemblies. The brain gets the message through by utilizing many pathways, acting in unison. In other words, the brain is organized for massive parallel processing.

"Binding" – the necessity to discriminate which neuron is part of the dance and which is not – is therefore a major question in neuroscience. Time binding has been proposed as an explanation for how the brain elegantly solves this problem: simultaneity in firing defines the state of belonging to the ensemble. Firing coincidence at the individual neuronal level translates to simultaneity at the level of the neuronal ensemble.

When neurons fire in groups, the resulting signal is sufficiently large to be detectable at the scalp in the EEG. The EEG records continuing brain activity as it constructs and then deconstructs neuronal assemblies. The brain also arranges for repetition of *nearly* the same firing pattern at characteristic frequencies. The result is that the EEG depicts numerous packets of activity, each at its own characteristic frequency.

Neurologist Simon Farmer succinctly described this model (Farmer, 2002):

> We are beginning to understand that brain rhythms, their synchronization and de-synchronization, form an important and possibly fundamental part of the orchestration of perception, motor action and conscious experience and that disruption of oscillation and/or temporal synchronization may be a fundamental mechanism of neurological disease.

As a neurologist, Farmer restricts his discourse to neurological conditions, but these concepts clearly have wider application.

All these features of neuronal assemblies either are, or should be, under tight regulation by the brain. Gross malfunction shows up in cases of seizure disorder, dementia, and traumatic brain injury. Understanding EEG tracings that are characteristic of these disorders helps to illuminate EEG findings in subjects with addictive disorders.

Even the healthy brain provides electrophysiologic access to brain activity during operant conditioning or subtle stimulation. Neurofeedback works by a reciprocal process. On the one hand, neurofeedback "invites" the brain to alter its activity; on the other hand, the brain – an entity seeking to maintain its integrity and stability – acts to counter this interference. *The result of repeating this brain challenge is to strengthen the regulatory loops.*

Significantly, this process does not depend on the presence of EEG abnormalities. The process works to enhance regulatory control, not to bring it into existence de novo. Fortunately, this is the usual state of affairs – absent gross and incapacitating pathology. Thus, in treating addictions, the EEG "entering behavior" of almost all patients is sufficient to respond well to the treatment. This is because only minor increments in regulatory control are needed to make a life of abstinence a realistic possibility.

Dynamic management of states

In treating addiction, it is critical to take into account the importance of arousal states and affect regulation

in treatment. The dynamics of brain function and regulation underpin addictive dysfunction. The relevance of the principles of brain dynamics in both functional and dysfunctional patterns makes effective intervention possible. The neuroscientist Walter Freeman expressed the concept succinctly (Freeman, 2000):

> Every neuron and every patch participates in every experience and behavior, even if its contribution is to silence its pulse train or stay dark in a brain image . . .

And, along similar lines, the following (ibid, p. 134):

> The . . . communities of modules in the two hemispheres, cooperating through the brainstem, the corpus callosum, and the other inter-hemispheric commissures, express a single, global, dynamic framework.

These comments support the observations made by many neurofeedback clinicians that a very limited number of training protocols are sufficient to move the brain globally to better function. Still, it matters a great deal which part of the cortex is trained and which parts of the whole domain of EEG frequencies are targeted in training. Again, some very simple rules apply. The lower EEG frequencies tend to organize more persistent states, whereas the higher EEG frequencies tend to underpin transient activity such as specific cognitive events. As for location on the scalp, knowledge about localization of function is clearly of relevance here. Alternatively, treatment of addicted patients typically does not target localized function, but instead targets very basic regulatory mechanisms that are broadly distributed and not localized at all.

It is best to think of this work, first, as addressing particular regulatory systems. The ebb and flow of the EEG at any one locale reflects the activation–relaxation dynamics of specific brain networks, and these are constituents of what are called phasic and tonic arousal. Scalp EEG tracings profile levels of arousal. The alcoholic brain often records a tonic high-arousal condition – for which neurofeedback offers a remedy. In fact, arousal regulation/dysregulation is the main clinical issue in the neurofeedback treatment of substance abusers.

Second, affect regulation is another target of neurofeedback training in an addicted population, as so many exhibit concurrent mood instabilities or mood-unstable personality disorders. One school of thought correlates personality disorders with disorders of attachment during early childhood. Through neurofeedback, the underlying circuitry governing emotional attachment is directly trainable. Prevailing psychotherapies may address obstacles to emotional attachment, but they do not access the organic basis of these obstacles directly. Neurofeedback successfully targets both the symptoms and the root causes of persistent addiction because:

1. It trains the brain to modulate overarousal, a maladaptive dysphoric state that often triggers attempts to "self-medicate" the hyperarousal.
2. It addresses the underlying, unremediated disorders of attachment that often have developed into maladaptive behavior patterns, personalities, and lifestyles.
3. Neurofeedback training broadly targets executive function. Impulsivity and behavioral disinhibition are evidence of diminished executive function. Neurofeedback training moderates obsessive/compulsive tendencies and thrill-seeking behavior. This training also impacts satiety and reward systems, modulating drives ranging from appetite awareness to drug-seeking behavior. Concomitantly, neurofeedback leads to improvements in working memory and cognitive function. As the proper hierarchy of regulation is restored to neural networks, patients experience greater mastery across the neuropsychiatric spectrum, including gains in cognitive function.

Satiety, behavior, and the brain

Neurofeedback prepares the neural substrate for a drug-free existence. Drug-seeking behavior is often in response to a felt need. One possible remedy is to train the central nervous system (CNS) to function in the absence of that drug. Neurofeedback helps to alter brain reward circuitry so that the association between pleasure and satiety and the use of a specific drug is diminished. Neurofeedback does not provoke the extreme states of euphoria associated by users with many drugs. However, the enhanced capacity for feeling joy and pleasure (on a daily, consistent basis) that often results from effective neurofeedback training does allow the person to sustain and enjoy sobriety.

What about nicotine addiction? It is quite apparent to many smokers that their brains function better when they smoke. These people give up smoking at some considerable cost to themselves and perhaps to their relationships and work performance as well. The first intervention in smoking cessation is to train the brain that nicotine no longer offers performance

increments. Then the cessation of smoking can take center stage.

The success of this strategy is most evident in work with marijuana smokers. In many cases, clients gave up marijuana quite unintentionally once they began EEG training. People routinely report, at some point in neurofeedback treatment, "I forgot to use!"

Patients are usually more surprised by this phenomenon than clinicians, who have observed it in many cases of treatment. The same has been reported for nicotine addiction.

The role and treatment of trauma

The discussion above hinges on the clinical process of neurophysiological normalization. Neurophysiological normalization may be a required component of stable, sustained abstinence (e.g. where marijuana use is driven by a felt need).

Addictive behavior coupled with a history of trauma makes treatment more complex, more challenging. Some addictions are difficult to treat unless the underlying trauma is somehow addressed as well.

Traumatic episodes are events in which the individual's survival was suddenly threatened, or when life-threatening events were observed in those near and dear. Traumatic events also include episodes in which the patient "merely" interprets or perceives that a given event may be life-threatening. Individuals are also traumatized by life events that don't necessarily kill or maim so much as bleed the spirit or vitality of a person to a grossly debilitating extent. Such individuals may seek out and/or benefit from "soul-work" – therapy for a person whose life force has been crushed in some way so that it cannot seek and find positive expression. Neurofeedback can be helpful here as well. By positive reinforcement – rewarding low EEG frequencies – treatment helps the person to encounter his "existential self." Under these conditions, traumas can be resolved, and healing and reconstitution of the self often occurs. People report these therapeutic experiences (often in retrospect) as life-altering. The process is another aspect of "self-repair," in that the role of the therapist is to facilitate and assist the healing that the patient invokes, using his own resources. The use of neurofeedback technology to facilitate this process is known as "alpha-theta training."

Alpha-theta training has three goals. First, it physiologically, psychologically, and perceptually distances the environment, which allows the person to focus inward calmly. Second, it quiets cortical function in general and the verbal "self-censor" in particular. This allows the core self to emerge into awareness by means of imagery. Alpha-theta sessions are often suffused with imagery that is dream-like, that is hypnagogic in quality. Third, the EEG becomes more coherent (wave forms become more similar) over larger brain regions: this enlarges the subjective boundary of self and helps draw others into one's affective embrace. Key historical relationships that had been problematic in the past can be quite suddenly restructured and perceived differently, in a more positive frame. Early childhood trauma may be defused and integrated. In some instances, the greatest perceived benefit of this training is identified by the patient with a particular session within the sequence of what is now called "deep-state" training. Observers unfamiliar with the process and its theoretical basis can find much that seems mysterious and unsystematic here. Healing from within is not confined to linear or methodologically rigid parameters.

Recovery from treatment-resistant addiction can require improved cerebral regulation as well as a "deeper" healing of injuries to the self. Ideally, the result is transformational, such that addiction no longer fills a need or, for that matter, has any place in the evolved life. In practice, results often fall short of the ideal. Nonetheless, neurofeedback has proven to be a valuable tool that allows some to maintain abstinence, recover function, and make progress in their lives.

The historical development of neurofeedback

A review of neurofeedback history is essential for three reasons: 1) it documents the depth and breadth of prior research; 2) it yields understanding of why neurofeedback still attracts sceptics, and 3) it provides a basis for current treatment approaches. Deep-state training began with Joe Kamiya, who first demonstrated successful reinforcement of alpha rhythms in 1962 (Kamiya, 1969; Kamiya & Noles, 1970). Kamiya then published a study demonstrating that alpha training was successful in moderating trait anxiety (Hardt & Kamiya, 1978). It was not long, however, before the alpha work caught public attention. Reports of idyllic experiences with altered states brought the technique into favor with the psychedelic crowd who wished simply to avoid the downside of the LSD experience. This development in turn disqualified the technique for formal academic research, and whatever minimal

research that was done seemed destined to fail. Alpha training fell quite precipitously into disrepute, and this is largely attributed to one negative outcome study (Lynch *et al.*, 1974). Subsequently, only isolated clinicians and researchers carried the work forward, operating outside of the academic community and without benefit of grant funding.

M. Barry Sterman pioneered the movement involved in training physiological normalization. Sterman worked at the Sepulveda Veterans Administration Hospital in Los Angeles. Initially the work was thought to have limited application to seizure management, and later to the "neurobiological deficit" of attention deficit hyperactivity disorder (ADHD) in children. Proponents insisted at the time that they did not regard the technique as broadly applicable to psychological dysfunction. Even within the biofeedback community itself, the work did not gain much of a beachhead. The research was richly applauded, accepted, and, ironically, ignored. Who needed EEG feedback when there was peripheral biofeedback? In any event, biofeedback therapists weren't inclined to work with seizure disorders. The field of neurology, meanwhile, had no use for a behavioral technique for managing seizures. By 1985, funding for EEG feedback research at the NIH had run dry. Again, only isolated clinicians carried the torch.

Jim Hardt, a student of Joe Kamiya, was the first to work successfully with addictions using alpha training (Hardt, 2007). One of his early clinical successes is illustrative of the process. The case is described in Hardt's recent book, *The Art of Smart Thinking* (p.106):

> In 1979, I had the opportunity to provide alpha training to a woman who I later discovered to be a multiple drug-user and a drug dealer. I did not know that she and her husband (also a dealer) were consuming almost an ounce of cocaine a day between them. She was drinking a fifth of hard liquor to take the edge off the cocaine, and she smoked tobacco daily, and took LSD, psilocybin, mescaline, and marijuana on a regular basis. She also took tranquilizers and stimulants to change her mind state whenever she wanted and in whatever direction she wished. Her personal motto was, "Excess is not enough!"
>
> On the fifth day of her alpha training she described "falling into a pool of alpha," which forever changed her life. Although she had no intention of reducing or stopping her drug use when she started alpha training, and in spite of the fact that she liked her drug use lifestyle and thought her life was "working very well," her drug use began to fall away. Within six weeks of the end of her alpha training, she was not using any drugs. Even the tobacco smoking had stopped. And now, she found that she could no longer live with her husband, who had not done the alpha training, and who continued to use and to deal drugs.

Hardt had a 9-year follow-up with this person, who continued her drug-free life, even to the point of spurning caffeine. This case violates all the expectations. There was not even the desire to quit drugs, and yet the training had a health-promoting and constructive effect. There had been no "hitting bottom" or other crisis to launch this person on the path to recovery. Moreover, there was ongoing benefit of the training even after its cessation. In this case, the entire training sequence was transformational in effect. Yet, there was no single transformative moment (as is sometimes the case).

Work with the theta band found favor in the research by Tom Budzynski on twilight learning. By holding people in a theta-dominant state just short of stage-one sleep with reinforcement techniques, Budzynski found that while in that state they were highly suggestible. The boundary of the self was more "permeable" (Budzynski, 1972). Soon thereafter, however, Budzynski's interest diverged to the peripheral biofeedback modalities that were of interest and were receiving grant funding at that time.

Elmer Green and his colleagues (with the active support of Karl Menninger) conducted the principal work with alpha-theta training (Green *et al.*, 1970; 1974). This research used alpha training mainly as an entry portal to states of theta dominance, with which the transformative experience was more directly associated. In this model, activity in both theta and alpha bands was reinforced. However, this research did not focus on or purport to address psychopathology in general or addictions in particular.

Lester Fehmi propelled a third initiative in studying alpha. Fehmi pioneered multi-channel alpha training to promote large-scale alpha coherence over the posterior cortex to still the sensory cortices and promote the capacity for more global, diffuse, and immersed states of attention to counter the Western bias toward narrowly focused attention (Fehmi, 1978; Fehmi and Robbins, 2007).

Neurofeedback in addictions treatment: what the research shows

The pivotal research of the biofeedback group at the Menninger Clinic resulted in the large-scale application of alpha-theta training to veteran alcoholics at the nearby Veterans Administration Hospital in Topeka,

Kansas (Goslinga, 1975). Twemlow and Bowen reported on trends in self-actualization scores with EEG feedback on 76 subjects (Twemlow & Bowen, 1976; 1977). Significantly, the only predictor of self-actualization scores that emerged with EEG training was religiosity. Perhaps patients with a high baseline religiosity score were more likely to experience "imagery with high religious impact, including experiences of white light, crucifixion, and other metaphors of death and rebirth" while in the theta-dominant state.

In 1977, Watson, Herder, and Passini published a study on 25 alcoholics who were given 10 hours of alpha training and then compared to matched controls (Passini *et al.*, 1977; Watson *et al.*, 1978). The study yielded evidence of improvement on state and trait anxiety, and 18-month follow-up yielded evidence of reduced alcohol consumption (Watson *et al.*, 1978). A third study, in which alpha training was done with 66 psychiatric patients and compared with no-treatment controls, yielded no significant differential changes (on 54 measures) outside of chance expectation. This negative outcome is not at all surprising given current understanding (vide infra).

Additional early studies

The study that revived interest in alpha-theta neurofeedback applications for addiction was undertaken by Eugene Peniston, a psychologist then employed at the Fort Lyon Veterans Administration (VA) facility to work with Vietnam veteran alcoholics. He also adapted the Menninger protocol for his work, having personally had an experience with the protocol at Menninger in the course of a training program. Ironically, he did not react well to the feedback session personally, and had to be strongly encouraged to try it again. Subsequent experiences were also wrenching, but they led him to undertake this research with some of his patients. Peniston also had a personal motivation: his brother had succumbed to alcoholism.

Following the basic Menninger protocol, the EEG training was preceded by eight 30-minute sessions of temperature biofeedback, which serves to give people a first experience with the concept of self-regulation training. Further, it calms an overactive sympathetic nervous system and brings people down from overaroused states. The thermal biofeedback was augmented with exercises in paced, slow breathing to promote relaxation. The EEG training involved fifteen 30-minute sessions of alpha-theta training. The alpha band was set at 8–13 Hz, and the theta band at 4–8 Hz. Reward was a continuous tone for as long as threshold conditions were met. Guided imagery augmented the EEG training with verbal inductions given prior to entry into the training phase. The details of these aspects of the protocol are described in a later publication (Peniston & Kulkosky, 1999).

A total of 20 participants entered the program, with 10 receiving feedback in addition to the normal residential treatment program. Participants had to have a history of at least four prior treatment failures for alcoholism. The average was 5.4 treatment failures. They also had to have a confirmed history of alcoholism extending more than 20 years.

On follow-up, the control group stayed true to form. All had to be readmitted to another round of treatment within 18 months of completion. By contrast, all 10 of the experimental subjects were successful over the initial 18-month follow-up period, and, in fact, remained successful over the subsequent 10 years or more of informal follow-up. In this context, *successful* meant no more problem drinking. Interestingly, one of the 10 was able to return to social drinking without a problem. Two of the participants thought the EEG training was "for the birds," and immediately headed for the local bar upon release from the program. They both became ill at once and found that they had lost their tolerance for alcohol, a quite common outcome of alpha training. This alcohol-mediated illness has since become known as the "Peniston flu." Because of this early relapse, they were initially listed as treatment failures by Peniston. However, in view of their sustained sobriety after this initial episode, these two should be counted among the treatment successes. This meant 100% success where none would have been expected!

A firestorm met Peniston's work within the biofeedback community when it was presented at the 1990 annual meeting in Washington DC. The original Biofeedback Research Society had been organized in 1969 around the impetus of alpha training. When that became discredited in the mid-seventies, they sought refuge in biofeedback using peripheral modalities. Biofeedback therapists and researchers did not want to be reminded of the disgrace visited upon the organization by the early alpha training. They were convinced they had seen the last of alpha training.

Yet, Peniston had brought considerable supportive data to bear. First of all, there were clear changes in the percentage of time patients exceeded the threshold

in alpha-theta training. Second, the experimental group demonstrated significant recovery on the Beck Depression Scale. Third, the control group showed increases in beta-endorphin levels over the course of the study. These were attributed to the stress of the therapy. Such an increase was not found in the experimental group.

Additionally, Peniston showed substantial normalization in subtest scores on the Millon Clinical Multiaxial Inventory (MCMI) and the Sixteen Personality Factor (16-PF) Questionnaire among the experimentals (Peniston & Kulkosky, 1990). Significant decreases occurred in MCMI scales labeled schizoid, avoidant, passive aggression, schizotypal, borderline, paranoid, anxiety, somatoform, dysthymia, alcohol abuse, psychotic thinking, psychotic depression, and psychotic delusion. Alcoholics receiving the standard medical treatment showed significant decreases only in two MCMI scales – avoidant and psychotic thinking – and an increase in one scale, the compulsive. On the 16-PF Questionnaire (Cattell *et al.*, 1970), the EEG training led to significant increases in warmth, abstract thinking, stability, conscientiousness, boldness, imaginativeness, and self-control. The training appeared to produce fundamental changes in personality variables that supported the observation of sustained sobriety.

By 1991, Peniston had published yet another study on the application of alpha-theta training to PTSD (Peniston & Kulkosky, 1991). A criticism directed at his earlier study was that he had not so much demonstrated remediation of alcoholism per se, but rather of PTSD, to which the alcoholism was secondary. Although this was a criticism that Peniston could accept, his next study formally focused on PTSD rather than on alcohol dependence. Because of the intimate association of PTSD and substance abuse, and because it supports the case for alpha-theta training, the study is an important part of the story.

A total of 29 Vietnam veterans was recruited into a study similar to that described above. This study administered 30 sessions of alpha-theta training, and then used the Minnesota Multiphasic Personality Inventory (MMPI) to track changes in personality variables. Favorable change occurred among the experimentals on nearly all the clinical subscales of the MMPI. The most substantial changes were in the depression, psychopathic deviate, paranoia, psychaesthenia, and schizophrenia subscales. The PTSD subscale of the MMPI improved by a factor of two-thirds among the experimentals, while remaining unchanged among the controls.

On follow-up after 30 months, only three of the 15 experimentals reported having had any disturbing flashbacks. All three were given six booster sessions of neurofeedback, in the course of which one subject required rehospitalization. All of the 14 controls had to be admitted to Veteran's Administration treatment centers at least twice for additional PTSD-related treatment within the 30-month period.

Finally, in 1993, Peniston published yet another study in which recovery from PTSD was evaluated with four-channel EEG synchrony training to see if it offered any advantage over conventional single-channel alpha training (Peniston *et al.*, 1993). No comparison group was involved. The explicit synchrony training promotes a state of global coherence in the alpha and theta bands, which is more conducive to the often-sought disengaged, internally focused, state. However, outcomes were not dramatically different from prior feedback experience in such dual-diagnosis patients (PTSD and alcoholism). Follow-up among the 20 subjects at 26 months revealed four participants who showed recurrence of PTSD symptoms. Outcomes with respect to alcohol consumption were not reported.

The brusque rejection of Peniston's work upon first presentation to the biofeedback community relegated the continuation of research and replications to the select subset who were hardy and committed. Only a few of these studies have been published. In an outcome study, Saxby showed changes in personality variables similar to Peniston and documented sustained relapse prevention in 13 of 14 participants over a 21-month follow-up (Saxby & Peniston, 1995). Steve Fahrion, of the Menninger group, published a single case study that tracked EEG measures and personality factors (Fahrion *et al.*, 1992). Byers also documented normalization of personality measures (Byers, 1992). Kelley reported on a 3-year outcome study of alpha-theta neurofeedback training for problem-drinking among the Dine' (Navajo) people (Kelly, 1997). He took care to integrate the feedback work into the existing cultural paradigms. Kelley reported extinction of drinking behavior and reduced incidence of destructive behaviors in 16 of the 20 participants.

More recently, a European study applied a very different EEG training technique to the problem of alcohol dependency. The technique of transiently altering the slow cortical potential (low-frequency EEG), which was developed in application to seizure

management, was employed with seven patients hospitalized for their alcoholism. In follow-up at 4 months after release from the hospital, six of the seven had retained sobriety, and these six were the ones who had demonstrated mastery of the skill of controlling their slow cortical potential (Schneider *et al.*, 1993).

Bodenhamer-Davis published follow-up results for the Peniston protocol with juvenile offenders (Bodenhamer-Davis & Callaway, 2004). Follow-up after 7–9 years found 80% maintenance of abstinence, and a rearrest rate down by half with respect to the comparison group. Burkett reported results mirroring those of Peniston for a tough clinical population, homeless crack addicts (Burkett *et al.*, 2004). Alpha-theta neurofeedback was added to a faith-based mission for the homeless in Houston. Substantial success was achieved according to multiple criteria: that the homeless person be housed, maintain work, and remain drug-free.

In addition to the published reports, various conference presentations attested to the successful insertion of alpha-theta training into real-world clinical practice. Tom Allen reported that it had become a central component of his treatment of juvenile substance abusers. Dr. Nancy White tracked MMPI data for 41 successive alcoholics in her private psychology practice and found consistent remediation on the depression scale. Fahrion employed alpha-theta training with parolees from the Kansas prison system. Whereas the results were generally positive in this case, they also showed a disappointingly high dropout rate.

More recently, Smith and Sams (2004) reported on a pilot study of neurotherapy in application to youthful offenders who were also substance abusers. The study was carried out in a therapeutic community where the youngsters typically stayed for 12 months. The median number of arrests for the treatment group (30 in number) had been two. Nearly all were of upper middle class in socioeconomic status, and nearly all were Caucasian.

Neurofeedback was also added to a 12-step-based program. Training was at a rate of four sessions per week; this program involved heart rate variability (HRV) training, which is thought to impact emotional responsivity. Significantly, the neurofeedback protocol falls into the category of physiological normalization. No alpha-theta training was done with this group, although it is possible that the HRV training served as a surrogate.

The outcomes reported for this pilot study mirrored the Peniston and CRI-Help data in nearly every respect: length of stay improved with the number of sessions; those who received a minimum of 24 sessions graduated from the program at a 100% rate. Even those who had only 12 sessions doubled their length of stay over controls who did without neurofeedback. Mean Beck Depression Inventory scores ($n = 30$) declined from 25.5 to 11.3. Psychiatric medications decreased among 50% of trainees, and increased in only one trainee, who also happened to be the one person to suffer relapse later. All group continuous performance test scores improved to normative levels, with all effect sizes exceeding one standard deviation. Average MMPI scores ($n = 30$) improved from 64.5 before training to 55.6 after training. If one disregards the masculine/feminine category where change was neither seen nor expected, the average change in the remaining nine categories was 10 points. Average changes of 10 or more points were observed for: depression (17), hysteria (15), hypochondriasis (12), schizophrenia (12), and paranoia (10). More than 90% achieved abstinence, and those who experienced brief relapse returned to recovery activity quickly. Only one relapsed irretrievably.

Two studies failed to corroborate Peniston's work. The first was a comparison of electromyography (EMG) biofeedback, transcendental meditation, and alpha-theta feedback with standard treatment of alcoholics (Taub *et al.*, 1994). Whereas enhanced clinical success was achieved within the EMG and transcendental meditation arms of the study, this success was not demonstrated in the alpha-theta arm. EEG advocates met this report with some suspicion because Taub was viewed as part of the biofeedback contingent that would just as soon see alpha-theta training disappear.

The second study was a university-based project that attempted to evaluate *pure feedback*, stripped of all the personal touches and concerned support that were part of the service provided by Peniston in the original work (Graap & Freides, 1997). Unsurprisingly, the study failed to support Peniston's findings, since therapeutic caring cannot be subtracted from the mix in the interest of pure science without sacrificing some treatment outcome variance.

By this time, Peniston's work had taken root among many clinicians – who pointed out that the training cannot be separated from its supportive context to stand on its own. Purifying and distilling the technique

down to its essence for purposes of research was not a viable option. Some neurofeedback proponents insisted that narrow-minded academic researchers were once again impugning the work of sensitive and competent clinicians, just as had happened in the early days of the alpha work.

Somewhat piqued by criticism of their failed replication, Graap and Freides uncharitably pointed out that some of Peniston's data seemed to indicate that the populations in his two studies were not entirely independent. Peniston defended himself, but the controversy did not entirely subside. In any event, Graap and Freides never published the results of their study (Graap and Freides, 1998).

Another criticism directed at the Peniston study was that the numbers were too small to allow firm conclusions. This criticism was statistically unsound at best and, perhaps, even disingenuous. The number of subjects required is a function of effect size, and in this case the contrast between experimentals and controls was so radical that the number of subjects was quite adequate to rule out the null hypothesis.

Nevertheless, during the mid-90s a large-scale replication of Peniston's work was organized at EEG Spectrum, then headed by Siegfried Othmer, the co-author of this chapter. The principal modification was that EEG training would replace thermal training prior to the alpha-theta work. This research was done in collaboration with CRI-Help, a Residential Treatment Center in Los Angeles. The intent was to assess the role of neurofeedback in as life-like a situation as possible; hence staff at CRI-Help were trained to provide the service.

The study's intent was to focus on alcoholism, but there weren't enough cases to meet group size objectives in reasonable time. Subsequently, study subject criteria were expanded to include other drugs as well. Heroin, crack cocaine, and methamphetamine users represented 30% of the treatment population; 10% were alcoholics. Nearly all subjects were multi-drug users. Everyone received the standard Minnesota Model treatment, with the experimentals also receiving neurofeedback; controls received additional individual and group psychotherapy.

Executive function can be regarded as the highest echelon of motor planning. Training the circuitry of motor control at any point inevitably trains executive function as well. The placement at Fz (Fz and other sites refer to the standard International 1020 system of electrode placement) gets the frontal region explicitly involved. Right hemisphere training was done with a parietal bias to calm down phasic arousal. For more extensive details of this protocol and for the clinical results that were obtained, see Othmer *et al.* (1999). The alpha-theta training used 8–11 Hz for the detection of alpha and 5–8 Hz for theta, with electrode placement at Pz (International 10-20 System). The neurofeedback consisted of a combination of SMR-beta training and alpha-theta training. SMR-beta training refers to the original procedure developed by Barry Sterman for seizure management, with a slight modification: the beta training, at 15–18 Hz, was used with the left hemisphere and the SMR-beta training, at 12–15 Hz, was used on the right. SMR refers to the sensorimotor rhythm first identified by Sterman. Electrode placement was C3-Fz for left hemisphere training, and C4-Pz for right hemisphere training. The principal objective of this protocol was improved regulation of arousal and enhanced executive function. Training to elevate amplitudes of the resting rhythm of the sensorimotor system moves the person to lower and more controlled states of tonic arousal. The slightly higher frequency training on the left side was often helpful for depression.

With 121 participants entering the program, group size was now adequate to withstand any potential statistical criticism, provided one could aggregate the data for the different drugs. When the results were later analyzed separately for the different drugs, this assumption was supported. The two groups were matched on the Addiction Severity Index. Those with a diagnosed psychotic or personality disorder or a seizure disorder were excluded from participation.

Results surfaced almost as soon as the training began. Even within the first couple of weeks of training (rate of two sessions per day), a statistically significant group effect in retention rate was demonstrated. This is shown in Fig. 19.1. During this period of time, only the SMR-beta training was done; this argues for a significant role for SMR-beta training in the overall protocol. The proximate goal in this training was to normalize the measures on a continuous performance test – the TOVA (test of variables of attention). Participants were re-tested after ten sessions. If TOVA scores had not normalized by that point, the subject was given ten additional sessions. The average number of sessions to TOVA normalization was 13, showing that the majority of subjects were able to normalize their TOVA scores within ten sessions (the median was ten).

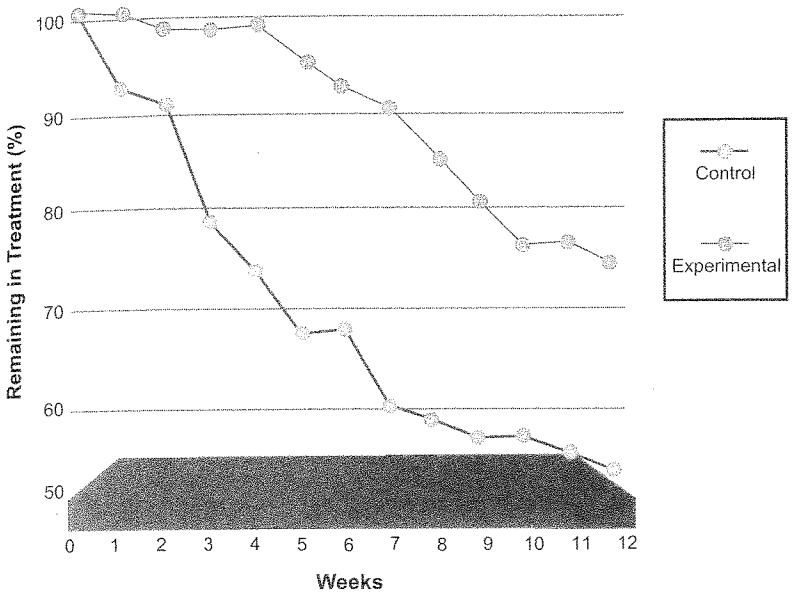

Figure 19.1 Retention in therapy for the experimental and control groups over the duration of the treatment program. Attrition rate for the controls was twice that of the experimentals. Significant differences appeared even during the SMR-beta phase of the EEG training.

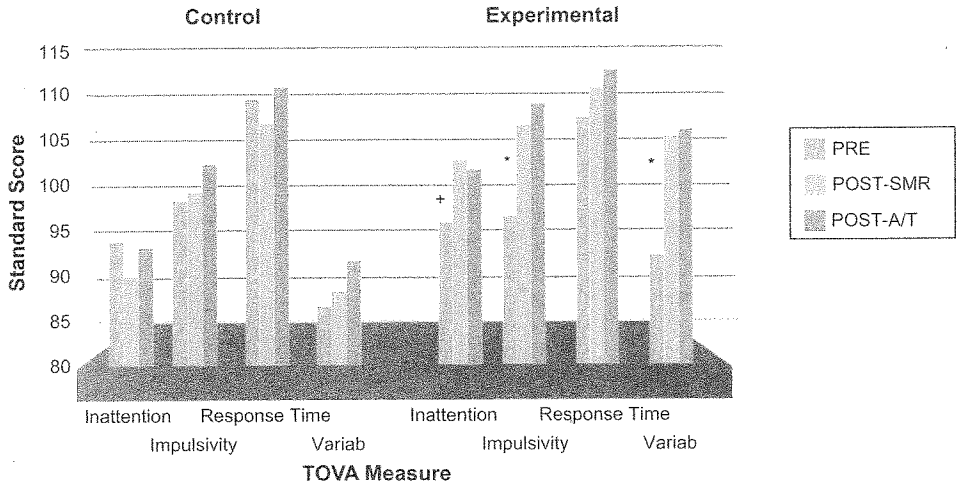

Figure 19.2 Pre-post standard scores are shown for the Test of Variables of Attention (the TOVA). Experimental subjects normalized all of their TOVA subtest scores with an average of 13 training sessions.

The average TOVA scores for the two cohorts are reproduced in Fig. 19.2. Remarkably, all post-training scores for the experimental group exceeded norms. This was somewhat surprising given the history of the participants. Many had been referred by Los Angeles County. Many were jobless. Some were homeless. About 30% had done prison time, and were likely to have had a history of psychological or physical trauma. CRI-Help estimates that their organization deals with the most challenging population that is engaged in regular addictions treatment anywhere.

A neurocognitive test battery was also given to all participants, but here the experimentals distinguished themselves from the controls significantly only on delayed recall testing. Though memory is one excellent measure of brain function, on the basis of the good continuous performance test data one might have expected a broader range of improvement. Gains were also observed on immediate recall and on the

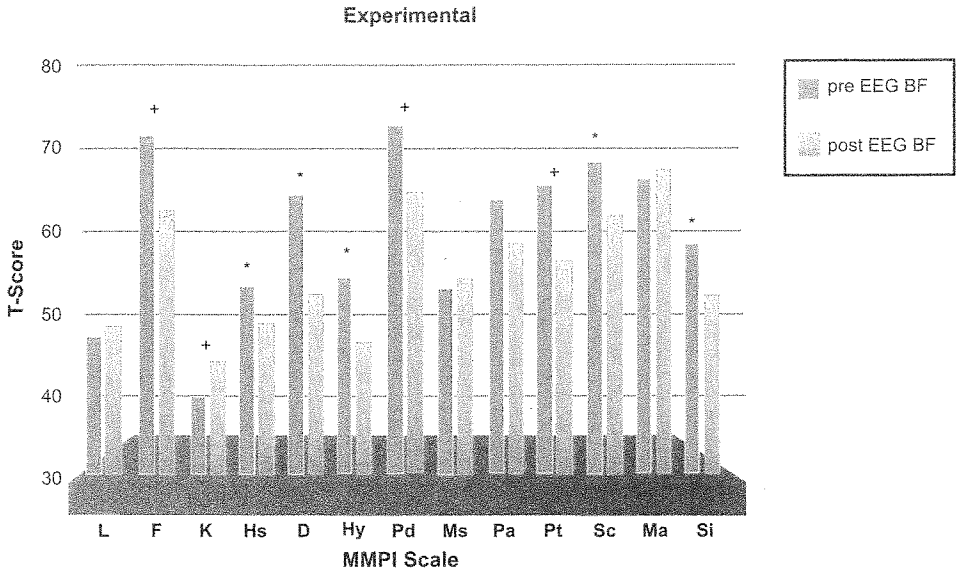

Figure 19.3 Pre-post MMPI data are shown for the experimentals. Substantial normalization of elevated scores is indicated. A star is used to indicate a significant treatment interaction, whereas a plus indicates significant change that nevertheless did not reach the level of significant treatment interaction.

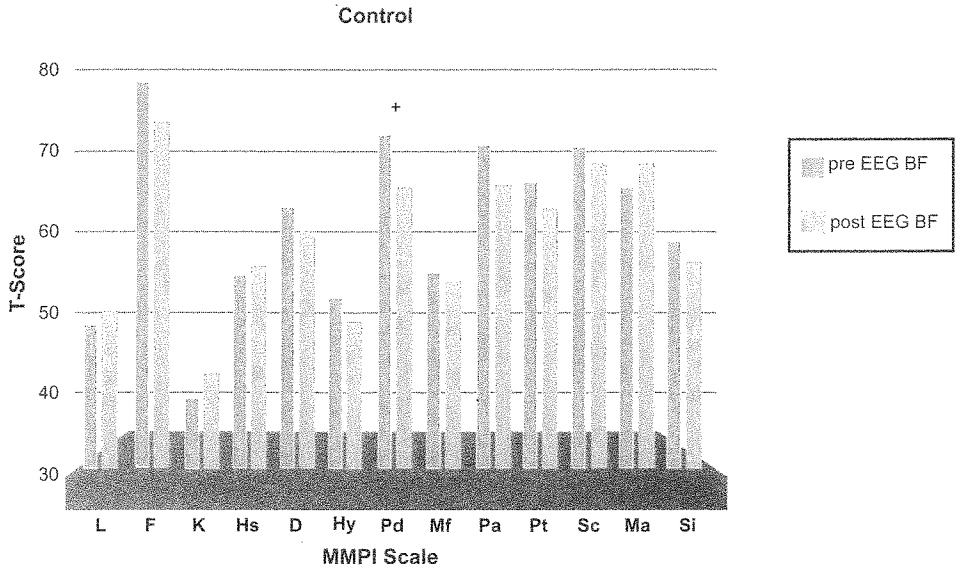

Figure 19.4 Comparative pre-post MMPI data are shown for the controls.

Tower Test, but there was no significant treatment interaction.

Following Peniston's method, the MMPI was also administered before and after the study, and the results mirrored his earlier data. The results are shown in Fig. 19.3 for the experimentals, and in Fig. 19.4 for the controls. A common thread running through all of these studies is the large change in the depression scale among experimental subjects. Additionally, the CRI-Help study confirmed Peniston's work with respect to significant changes in the hypochondriasis, conversion hysteria, and schizophrenia scales. The consistent direction of these changes over all studies indicates that this training clearly improved a

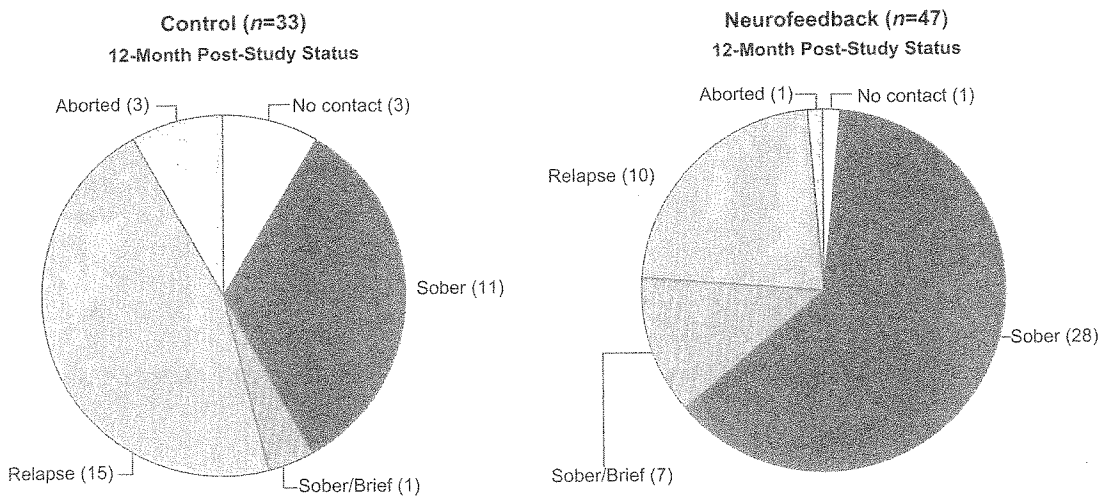

Figure 19.5 Relapse data are shown for the period 12 months post-graduation. Experimentals exhibit a three times higher success rate in maintaining abstinence if brief relapsers are included (35 versus 12).

number of aspects of the trainees' mental health status.

Relapse was tracked in these individuals over a 3-year period, but has been formally reported only for the first year. The data are shown in Fig. 19.5. In interpreting the data, one must consider that, at the end of the program, the graduating cohorts are no longer equivalent owing to the much higher attrition rate among controls.

A policy perspective would refer back to the point of entry into the program, where the groups are still matched. An arbitrary person inserted into the experimental program would have a three times higher likelihood of being relapse-free at the end of 1 year compared to someone who had received the standard treatment alone. This turns a success rate of nominally 20% into a success rate of more than 60%.

The current status

Given the novelty of the neurofeedback approach, it was difficult to get this work published. It finally appeared in the *American Journal of Drug and Alcohol Abuse* in August of 2005 (Scott *et al.*, 2005). The study had taken 4 years, and the subsequent path to publication had taken about 5 years. Since that time, numerous protocol enhancements have evolved through clinical application. Peniston reported a number of abreactions in the course of his training. The CRI-Help study had already changed to a reinforcement band of 5–8 Hz versus the prior 4–8 Hz, and the problem of abreactions essentially resolved. Apparently, this change could be made with no loss in overall clinical efficiency. Further positive changes have also occurred since.

In the CRI-Help study, Principal Investigator William Scott found that a number of people reacted badly to alpha reinforcement. Often, these people would show high alpha amplitudes at inception. In these cases, it was beneficial first to train alpha down before administering the standard protocol later. One may think of this modification as additional training toward functional normalization, distinct from the alpha-theta sessions, where the primary purpose is experiential. Hence, there is no conflict in training alpha first down and then up in the same individual. The first phase takes place as part of the alert-state training, and the second takes place in a state of low arousal, or deep-state, training.

Subsequent evolution of the technique has taken two forms. First, it was found that if the alpha reward frequency was individualized to the client, the response to the reinforcement became more consistently favorable. This adjustment mostly falls in a narrow range between 10 and 11 Hz, but the fine-tuning turns out to be quite effective.

A second change arose from the work of Les Fehmi and Jim Hardt, who consistently rewarded alpha in a multi-channel configuration. The multi-channel set-up favors the emergence of a spatially distributed state of alpha synchrony, rather than merely promoting higher alpha amplitude in the raw EEG. Rewarding for alpha synchrony explicitly accelerates achievement of the real objective of this training. Simply rewarding

higher alpha amplitudes could be contraindicated when the alpha band appears to be a zone of vulnerability, as it may be in cases of minor traumatic brain injury, fibromyalgia, and migraine. The more significant evolution occurred in the first stage of training, where the objective is enhanced physiological self-regulation. With the simple modification of allowing the reward frequency to be individually optimized, the training has become much more diverse, more effective, and also more targeted. Expanding the range of reward frequencies and shifting the reward frequency based on the person's response to the training has stretched the effectively reinforcible EEG spectrum up to 40 Hz and even down to 0.01 Hz (Othmer & Othmer, 2007).The traditional SMR-beta training most directly addresses activation of the motor system and what might be called cognitive arousal, the very quality that determines how well one handles a challenge like a continuous performance test. The expansion of the training window to both lower and higher frequencies showed that the vast majority of trainees have an even greater need for training at lower frequencies. The impact on affective states is much more immediate and compelling at low frequencies, with the additive benefit of greater impact on felt states, which in turn promotes engagement with the overall process. One might term this affective arousal, in that the training constructively addresses fear conditioning, trait anxiety, and affect disregulation.

Interconnection and normalization

With the option of training at low frequency along with the optimization strategy, trainees usually gravitate toward the training they most need at the particular time. This has produced a greater recognition of the role that psychological trauma, particularly early childhood trauma, plays in eliciting dysregulation, a condition that biofeedback strategies effectively remediates. A two-pronged strategy has evolved for addressing the trauma formations that underlie many treatment-resistant addictions. The first training, also referred to as "alert-state" training (formerly SMR-beta), addresses the physiological dysregulation, and the "deep-state" (alpha-theta) training tackles psychodynamic aspects.

The connections between treatment-resistant addictive disease and trauma, and their response to neurofeedback, offers the tantalizing possibility that neurofeedback may be applicable to other conditions as well. A history of trauma often accompanies severe chronic pain conditions, the major eating disorders, and intractable, severe, migraine. Such history is often found in treatment-resistant fibromyalgia, and chronic fatigue syndrome as well. Finally, there is a correlation of psychological trauma with disease mortality (Boscarino, 2008).

With addictive disease, the primary therapeutic effect of neurofeedback is its impact on the networks that perceive and process the trauma. Often the patient has experienced no physical injury. Psychological trauma starts as a "software" problem, which may then become progressively encoded in pathological central network relations. This paves the way, neuronally, for encoding and preferential firing of neuronal pathways resulting from subsequent trauma. In this fashion, the patient's vulnerability continues to increase. The accompanying syndromes then appear structurally based and even untreatable. The neurofeedback paradigm, however, approaches these clinical challenges as "software" problems, problems that are amenable to modification owing to the inherent plasticity of neural networks. The postulated pathological network relations have three principal features. First, the whole "system" becomes less stable; second, deviations appear in cortical–subcortical linkages; third, deviations appear in cortical connectivity. The instabilities are directly observable clinically in the variability of symptom severity over time, in the appearance of episodic conditions, and in fluctuating functionality. The subcortical dysfunctions are accessible by means of imaging technology such as SPECT and PET. From the standpoint of network theory, a hierarchical needs assessment shows that the first priority is to restore system stability. The second priority is to restore the (vertical) hierarchy of regulation, from brainstem to cortex; the third priority is to normalize the (horizontal) linkages between cortical regions where these may be anomalous.

Because neural networks are so tightly linked, any of the standard neurofeedback approaches will impact the system at all three levels. One can refine and optimize intervention, however, by taking advantage of *localization of function*. Executive function has a left frontal bias; emotional regulation has a right frontal bias; cerebral stability is optimized by targeting interhemispheric timing relationships, etc. There are also known frequency relationships. For example, cognitive function has a higher frequency bias; affect regulation has a lower frequency bias. The left hemisphere has a higher frequency bias than the right, and so on.

The two kinds of training that have emerged appeal to two fundamental aspects of the self: the first is the self in interaction with the outside world – this involves the usual gamut of concerns of the cognitive scientist from sensory processing to executive function to the organization of movement. The second involves the so-called *essential* self, the existential self that moves through time as a consistent psychic entity, maintaining a core character continuity throughout all the vicissitudes of life. In this self, the internal gyroscope is wound up, the moral compass is set, and the range and depth of emotional sentience are tuned. The addict requires attention and training in both domains, and these are directly accessible through interaction with the neuronal networks.

In addition to psychological trauma, there is also the problem of minor traumatic brain injury. These head injuries don't involve skull fracture or penetrating wounds, and, regrettably, may escape the careful attention of medical professionals. There is nothing minor, however, about the symptoms that are typically reported. Unfortunately, such symptom presentations are often unsupported by conventional CT imagery, and are thus largely discounted. Though dysfunction may last throughout life, the causal connection with the prior head injury is unlikely to be established. A recent evaluation of some 845 patients at New York addictions treatment centers found that 54% had suffered a minor traumatic brain injury (Gordon *et al.*, 2004). So, minor traumatic brain injury could be a significant etiological factor in intractable addiction. The lack of structural evidence of injury indicates that, as with psychological trauma, a major component of dysfunction lies within the domain of disturbed network relations. There is much evidence for this in functional imaging studies (Hoffman *et al.*, 1996; Thornton, 2005). Recoveries and other positive outcomes achieved through neurofeedback further support this functional model of minor traumatic brain injury (Walker *et al.*, 2002).

The stealth factor

One may consider both psychological trauma and minor brain injury as *stealth* conditions that may have gone largely unrecognized when patients present for addiction treatment. Each type of dysfunction is ultimately due to deficits in the brain's capacity to self-regulate. The remediation of these deficits is a necessary part of addiction treatment. Fortunately, neurofeedback training substantially addresses these disregulations, but they must also be recognized and tracked throughout adjunctive therapies as well.

At this point we are perhaps in a better position to understand the poor outcomes of some of the earlier studies. Alpha reinforcement training is not likely to help people with unremediated traumatic brain injury, and may even be counterproductive. For many substance abuse patients, a narrow focus on the addiction status in treatment is unlikely to yield success. It is vital to address the psychological, psychopharmacological, social, spiritual, and neurophysiologic bases of the condition.

Eugene Peniston brought all the necessary elements of the recovery process together systematically, perhaps for the first time. Subsequent attempts to distil from this work the single "active ingredient" failed from the start. Many ingredients are involved in the task of restoring wholeness. Restoration of adequate self-regulatory status is, however, indispensable as a preparatory step for the journey toward self-actualization. Succeeding developments have only built upon and refined the Menninger/Peniston approach.

The transformative event

There is evidence that addictive behavior can be extinguished by the use of specific biofeedback techniques. Success is now possible for patient populations previously considered untreatable. Neurofeedback kindles a recovery process that the brain continues on its own. The process appears to be increasingly self-reinforcing as treatment progresses. Neurofeedback can be considered a *healthy kindling* process, where success means a transformed life, a life in which addiction no longer fits. This extension of healthy adaptation is a benefit that seems to evolve beyond (time-limited) neurofeedback treatment protocols.

Sometimes, the process of change is sudden and is attributed to a single transformative event (which typically occurs in the alpha-theta phase of the training). This kind of result is described by one therapist as follows:

> A 45-year-old male came to me for practicum work after his initial training as a neurofeedback therapist. He had never done any alpha-theta training on himself. After 15 minutes of training at Pz, he gasped, grabbed the arms of the chair, and began sobbing. At the end of the 30-minute training period, he was initially unable to describe his experience except to say that he was "overwhelmed by

the beauty." After lunch, he came back to the office and described the following scene: "I felt an odd sensation of floating. I was startled at first but then relaxed into the experience. I then floated up out of my chair and accelerated rapidly to a point high above the Earth where I remained suspended, watching the Earth and the stars. The visual experience was incredibly beautiful. I then knew that I was connected to everything in the universe and that I was nurtured and loved by it all. There was no experience of time. Then, I was gently drawn back to my chair where I was immersed in waves of pleasant emotion." In a later recollection, he reported as follows: "For the next two days, I anguished over my inability to get back to that beauty during alpha-theta training. On the last day, I set the expectation that I would resolve the longing for the experience. After just a few minutes of training, I was back among the stars and all the beauty and sense of peace. After a while, I was drawn back to Earth."

The therapist commented that this man had worked for years in an adversarial environment involving oversight of government contracts. He had become, by his own admission, suspicious and guarded. He has since become warm, friendly, and engaging.

This type of experience is not uncommon in the alpha-theta training. Even more than with physiological normalization work, one has the sense that neurofeedback simply sets the stage for this kind of personal growth. Witnessing such healing in patients leaves the therapist more humble in regard to his own role in the process. With the client doing so much of the work, there is no need for the therapist to be intrusive. The *sine qua non* of this process is that there be a trusting relationship with the therapist. It is also critical that the therapist have his own traumas well resolved. Perhaps the circumstances for such a positive, supportive relationship were absent in the failed replication by Graap and Freides, where the objective was to test the intrinsic, uncontaminated alpha-theta procedure.

Early researchers trying to replicate the alpha training of Kamiya and Hardt made the mistake of thinking that alpha could be trained in isolation. In actual fact, the alpha amplitude remains intimately connected with and responsive to the state of the person. If conditions for the elevation in alpha amplitudes are lacking, no amount of reinforcement will make a difference. It would be akin to bird watching during a storm – the two activities are simply not compatible. This misunderstanding may also have contributed to the failed replications of the studies of Graap and Freides, and that of Taub.

In other words, treatment efficacy is complex. Indeed, all the early experiments by Les Fehmi in enhancing alpha in his own brain were complete failures. He could not get there by merely striving for it. Having discovered that a modest surrender to the process was necessary, he tried to practise that – but failed as well. It was necessary for him to *truly* surrender to the process, on every psychological level. So, merely subjecting someone to this procedure for a given length of time is not a predictor, let alone a guarantee, of a positive outcome. It is crucial to determine whether the given reinforcement results in state change. That in turn is likely to depend on variables such as trust, or sense of safety, or even, as was seen in Fehmi's own case, a complete absence of striving toward the goal. These uncodified yet nonetheless necessary contexts may not have been adequately provided in earlier research settings.

One of the present authors (S. O.) had an experience similar to Fehmi's – the alpha amplitudes would just not move! Yet, in a subsequent session involving verbal induction, the author yielded to the process readily. Fehmi utilizes verbal induction with all his clients along with (and independent of) his instrumental work. This availability of multiple pathways to success likewise puts the Taub study in a very different light. The success of the EMG and transcendental meditation arms in that study illustrates that different pathways may converge upon a common goal.

The transformative event is not unique to alpha-theta training (although, arguably, alpha-theta training is safer and more healthful than other paths). LSD, for example, was used in addiction treatment in the 1960s. One or two LSD sessions during an extended 6-month course of therapy made a large difference in outcomes that may only be attributed to these singular events. In three different universities, recovery at a 50% level was documented (Abrahamson, 1961). Recovery is sometimes achieved after the individual has a "peak experience." The affective component of the experience is what seems to matter. In one study, subjects were first given penicillamine; their LSD experiences were identical except for a dampening of affect. The opportunity for recovery, however, was thereby lost (Maslow, 1971).

Stanislov Grof routinely observed such transformative events in the course of his holotropic breathwork (Grof, 2000). In this approach, hyperventilation leads to a state of hypocapnia (reduced CO_2 levels in the brain), resulting in vasoconstriction that, in turn, alters

the EEG toward theta- and delta-dominant states. Such states favor access to traumatic memories as well as to altered states. The peak experience, or transformative event, thus provoked gave subjects a rare glimpse of the "actualized" self, which in turn became the subjects' map for further inner work and growth.

The technique of eye movement desensitization and reprocessing similarly works by stimulating the low EEG frequencies where traumas are preferentially accessed. Hypnosis too typically moves subjects into states where low EEG frequencies are dominant. Indeed, one of the present authors refers to[1] alpha-theta deep-state training as "EEG hypnosis."

What differentiates modern alpha-theta training from these other techniques is the precision afforded by the use of EEG. What distinguishes the neurofeedback approach in general is the fact that one technique can address multiple aspects of the dysregulated brain – up to and including the brain in a state of addiction.

The transformative event, when it occurs, leaves little doubt regarding its significance. But it is not essential to the therapeutic process, and it should not be an explicit objective in treatment. When we asked participants with good outcomes to explain their stable abstinence, all cited the twelve-step group experience in which they were still involved. None named the neurofeedback. This was disappointing to us, of course, but hardly surprising. The effect of biofeedback tends to be gradual, cumulative, and always ego-syntonic. Thus, it could conceivably hover somewhere beneath the person's radar. Because of the way this progress integrates with the developing self, accommodations are subtly made so that the biofeedback contribution is subsumed in the increasingly autonomous version of self that the person narrates.

Summary

This chapter provides evidence that addictive behaviors may be conceptualized as *disorders of dysregulation*. Such disorders may have both a structural and a functional basis. Addressing the functional aspect of these disorders results in a surprising degree of recovery. Observing and modifying the EEG using neurofeedback methodologies can change dysregulated brain networks to regulated ones; this operant reinforcement strategy works toward the recovery of numerous domains of brain function. Neurofeedback is an entirely new treatment resource for conditions such as addiction that currently do not readily respond to pharmacological intervention or to cognitively based therapies. This approach is particularly successful in resolving trauma formations – both psychological and physical – that can impede clinical success in many other clinical domains. A comprehensive approach that addresses multiple aspects of neuronal dysregulation manifestly enhances success in addiction treatment. The data compel consideration of EEG neurofeedback for inclusion in contemporary treatment programs. Additionally, the efficacy of neurofeedback and the mechanisms by which it works may argue for adopting a more inclusive "systems-level" perspective on the problem of addiction.

Conclusion

Stable recovery from addiction may be more likely when modern neurophysiological and behavioral technologies are combined with traditional medical and psychotherapeutic interventions. The model for these advances is the neural network encoding that makes the brain accessible to therapeutic intervention at the neuronal level. The technique of operant conditioning of EEG variables, combined with psychotherapy and pharmacological support, works to enhance clinical outcomes, even among the most challenging clinical populations.

Global remediation of mental functioning – rather than targeting addiction in isolation – is a compelling clinical strategy, given the high prevalence of comorbid conditions, including PTSD, minor traumatic brain injury, personality disorders, impulse control problems, behavioral disinhibition, anxiety, and mood disorders. These comorbidities, like addiction, feature deficits in neural network functioning, and these deficits may be treated by means of neurofeedback training.

Restoring the brain's regulatory and cognitive function by means of neurofeedback also heals psychological wounds by inducing and reinforcing particular network states that favor trauma resolution, and subsequent progress toward self-actualization.

[1] Dr. Steinberg has used alpha-theta in his clinical neuropsychology practice for nearly two decades. He has also used EMDR and hypnosis, but prefers the clinical successes attained through neurofeedback and alpha-theta.

References

Abrahamson, H. (ed.) (1961). *The Use of LSD in Psychotherapy*. New York: Josiah Macy Foundation.

Bodenhamer-Davis, E. & Callaway, T. (2004). Extended follow-up results of Peniston protocol results with chemical dependency. *Journal of Neurotherapy*, 8(2), 135.

Boscarino, J. A. (2008). Psychobiologic predictors of disease mortality after psychological trauma: implications for research and clinical surveillance. *Journal of Nervous & Mental Disease*, **196**(2), 100–7.

Budzynski, T. H. & Stoyva, J. M. (1972). Biofeedback techniques in behavior therapy. In *Behavior and Self-Control*, D. Shapiro, T. X. Barber, L. V. DiCara *et al.* (eds). Chicago: Aldine, pp. 437–59.

Burkett, V. S., Cummins, J. M., Dickson, R. M. & Skolnick, M. H. (2004). Treatment effects related to EEG biofeedback for crack cocaine dependency in a faith-based homeless mission. *Journal of Neurotherapy*, 8(2), 138–40.

Byers, A. P. (1992). The normalization of a personality through neurofeedback therapy. *Subtle Energies*, 3(1), 1–17.

Fahrion, S. L., Walters, E. D., Coyne, L. & Allen, T. (1992) Alterations in EEG amplitude, personality factors, and brain electrical mapping after alpha-theta brainwave training. *Alcoholism: Clinical and Experimental Research*, **16**, 547–52.

Farmer, S. F. (2002). Neural rhythms in Parkinson's disease. *Brain*, **125**, 1175–76.

Fehmi, L. G. (1978). EEG biofeedback, multi-channel synchrony training, and attention. In *Expanding Dimensions of Consciousness*, A. Sugarman & R. E. Tarter (eds). New York: Springer Publishing Company, pp. 152–82.

Gordon, W., Fenske, C., Perez, K. & Brandau, S. (2004). *Comorbidity between TBI and Substance Abuse*. Paper presented at: Strengthening Systems: Investing for Results. Alcoholism and Substance Abuse Providers of New York State 7th Annual Statewide Conference, New York, NY.

Goslinga, J. J. (1975). Biofeedback for chemical problem patients: a developmental process. *Journal of Biofeedback*, 2, 17–27.

Graap, K. & Freides, D. (1998). Regarding the database for the Peniston alpha-theta EEG biofeedback protocol. *Applied Psychophysiology and Biofeedback*, 23(4), 265–72.

Graap, K., Ready, D. J., Freides, D., Daniels, R., & Baltzell, D. (1997). EEG biofeedback treatment for Vietnam veterans suffering from post traumatic stress disorder. *Journal of Neurotherapy*, 2, 65–66

Green, E. E., Green, A. M. & Walters, E. D. (1970). Voluntary control of internal states: psychological and physiological. *Journal of Transpersonal Psychology*, 2, 1–26.

Green, E. E., Green, A. M. & Walters, E. D. (1974). Alpha-theta biofeedback training. *Journal of Biofeedback*, 2, 7–13.

Grof, S. (2000). *Psychology of the Future*. New York: SUNY Press.

Hardt, J. V. (2007). *The Art of Smart Thinking*. Santa Clara: BioCybernaut Press.

Hardt, J. V. & Kamiya, J. (1978). Anxiety change through encephalographic alpha feedback seen only in high anxiety subjects. *Science*, **201**(4350), 79–81.

Hoffman, D., Stockdale, S. & van Egeren, L. (1996). EEG neurofeedback in the treatment of mild traumatic brain injury. *Clinical EEG*, 27, 6.

Kamiya, J. (1969). Operant control of the EEG alpha rhythm and some of its reported effects on consciousness. In *Altered States of Consciousness*, C. T. Tart (ed.). New York: Wiley, pp. 519–29.

Kamiya, J. & Noles, D. (1970). The control of electroencephalographic alpha rhythms through auditory feedback and associated mental activity. *Psychophysiology*, 6, 76.

Kelly, M. J. (1997). Native Americans, neurofeedback, and substance abuse theory: three year outcome of alpha/theta neurofeedback training in the treatment of problem drinking among Dine' (Navajo) People. *Journal of Neurotherapy*, 2(3), 24–60.

Lynch, J. L., Paskewitz, D. & Orne, M. T. (1974). Some factors in the feedback control of the human alpha rhythm. *Psychosomatic Medicine*, **36**, 399–410.

Maslow, A. (1971). *The Farther Reaches of Human Nature*. New York: Viking Press, p. 108.

Othmer, S. F. & Othmer, S. (2007). Interhemispheric EEG training; clinical experience and conceptual models. In *Handbook of Neurofeedback: Dynamics and Clinical Applications*, J. R. Evans (ed.). New York: Haworth Medical Press, pp. 109–36.

Othmer, S., Othmer, S. F. & Kaiser, D. A. (1999). EEG biofeedback: an emerging model for its global efficacy. In *Introduction to Quantitative EEG and Neurofeedback*, J. R. Evans & A. Abarbanel (eds). San Diego: Academic Press, pp. 243–310.

Passini, F. T., Watson, C. B., Dehnel, L., Herder, J. & Watkins, B. (1977). Alpha wave biofeedback training therapy in alcoholics. *Journal of Clinical Psychology*, **33**, 292–9.

Peniston, E. G. & Kulkosky, P. J. (1989). Alpha-theta brainwave training and beta endorphin levels in alcoholics. *Alcoholism: Clinical and Experimental Results*, 13(2), 271–9.

Peniston, E. G. & Kulkosky, P. J. (1990). Alcoholic personality and alpha-theta brainwave training. *Medical Psychotherapy: An International Journal*, 3, 37–55.

Peniston, E. G. & Kulkosky, P. J. (1991). Alpha-theta brainwave neurofeedback therapy for Vietnam veterans with combat-related posttraumatic stress disorder. *Medical Psychotherapy: An International Journal*, **4**, 47–60.

Peniston, E. G., & Kulkosky, P. J. (1999). Neurofeedback in the treatment of addictive disorders. In *Introduction to Quantitative EEG and Neurofeedback*, J. R. Evans & A. Abarbanel (eds.). San Diego: Academic Press.

Peniston, E. G. Marrinan, D. A., Deming, W. A. & Kulkosky, P. J. (1993). EEG alpha-theta brainwave synchronization in Vietnam theater veteran with combat-related posttraumatic stress disorder and alcohol abuse. *Medical Psychotherapy: An International Journal*, **6**, 37–50.

Scott, W. C., Kaiser, D. A., Othmer, S. and Sideroff, S. I. (2005). Effects of an EEG biofeedback protocol on a mixed substance abusing population. *American Journal of Drug and Alcohol Abuse*, **31**(3), 455–69.

Schneider, F., Elbert, T., Heimann, H. *et al.* (1993). Self-regulation of slow cortical potentials in psychiatric patients: alcohol dependency. *Biofeedback Self Regulation*, **18**(1), 23–32.

Smith, P. N. & Sams, M. W. (2004). Neurofeedback with Youth Offenders. A pilot study in the use of analog/QEEG-based remedial neurofeedback training. Presentation at ISNR Annual Conference, August 8, 2004

Taub, E., Steiner, S. S., Smith, R. B., Weingarten, E. & Walton, K. G. (1994). Effectiveness of broad spectrum approaches to relapse prevention in severe alcoholism: a long-term, randomized, controlled trial of transcendental meditation, EMG biofeedback, and electronic neurotherapy. *Alcohol Treatment Quarterly*,

Thornton, K. (2005). EEG biofeedback for reading disabilities and traumatic brain injury. *Child and Adolescent Psychiatric Clinics of North America*, **14**(1), 137–62.

Twemlow, S. W. & Bowen, W. T. (1976). EEG biofeedback induced self-actualization in alcoholics. *Journal of Biofeedback*, **3**, 20–5.

Twemlow, S. W. & Bowen, W. T. (1977a). Sociocultural predictors of self actualization in EEG biofeedback treated alcoholics. *Psychological Reports*, **40**, 591–8.

Twemlow, S. W. & Bowen, W. T. (1977b). Biofeedback induced energy redistribution in the alcoholic EEG. *Journal of Biofeedback*, **3**, 14–19.

Walker, J. E., Norman, C. A. & Weber, R. (2002). Impact of QEEG-guided coherence training for patients with a mild traumatic brain injury. *Journal of Neurotherapy*, **6**, 31–43.

Watson, C. G., Herder, J. & Passini, E. T. (1978). Alpha biofeedback therapy in alcoholics. An 18-month follow-up. *Journal of Clinical Psychology*, **34**, 765–9.