

EEG Biofeedback (Neurofeedback) as a Modality for Treating Addictions

Siegfried Othmer, Ph.D. and Mark Steinberg, Ph.D.

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 viable modality for treating addictions. When surprising and relevant clinical data emerge in the course of developing a treatment model, the data can compel a reappraisal of assumptions. A conceptual framework is then called for to integrate and consolidate the disparate data. Without a sensible model, even compelling data may spend years in obscurity before clinicians generally and the scientific community at large will rely upon the data to support practical interventions. In the case of EEG biofeedback, the data provoked the generation of the model. Yet an explanation of the model preceding the presentation of data will lay the groundwork for understanding the methodology and the efficacy of neurofeedback for addictions.

Addictions accrue from both psychological and physiological factors, and the prevailing remedies predominantly appeal largely to one of these domains. Targeting either of these domains selectively has not had good clinical outcomes, especially over the longer term. The realm that connects the domain of physiology with that of psychodynamics has been oddly missing from theory and practice. This realm is *brain behavior*, or that which overtly organizes a person's behavior.

Though it is tempting (and perhaps traditional) to leave the realm of brain behavior entirely to neurophysiology, doing so could overlook or minimize some critical elements. Medical science has supplied a plethora of knowledge about structural deficits in brain function; additionally, however, the *functional* domain of brain behavior is crucially relevant. Cognitive neuroscience — which does concern itself with brain function — has only nascently engaged the challenge of psychopathology. Relieving the misery of addiction through understanding brain function and modifying it using its own mechanisms is truly new ground.

Neuronal networks organize behavioral repertoires in their entirety. These networks exhibit the essential feature of a high level of integration and of hierarchical organization. The brain's internal conversations that take place via these networks are both private and individual at each juncture, and yet are highly centrally regulated as well. On the operational level, as in all communication networks, timing is crucial. Unsurprisingly, wherever specifications are tight in a regulatory system, there is the opportunity for departures from functional integrity.

A key assumption is that psychopathology in general, and the problem of addiction in particular, is, in principle, a deficit in *network relations* (i.e., in the brain's internal communication). In accord with this model, more recent publications posit "connectivity deficits" as being central to disorders such as schizophrenia, autism, and Alzheimer's (Singer, 2007). These severe clinical syndromes represent the most obvious cases in point, but there may be more universal relevance for this model.

If such connectivity deficits lie in the functional domain, then they may be accessible for strategic use in remediation. If this is true, then relevant existing therapies must abide and incorporate these principles as well. For example, to benefit from extended psychotherapy (even though it is conducted primarily through language), the individual must also sustain altered functioning of cerebral regulatory networks, often achieved indirectly. Effective psychopharmacology must also subsume this model of shifts in the ambient operating level of neuromodulatory networks.

It is reasonable to view pharmacology as a direct appeal to biological regulatory apparatus, but pharmacology does not exhaust therapeutic options in that regard. It is also possible to interact with the bio-electrical domain of brain function; this approach encroaches upon the realm of brain dynamics and brain self-regulation at the speed of life and thought, thus affording a unique input to the decision-making habits that can make or break addictions. The electroencephalogram (EEG) gives efficient access to this aspect of brain behavior. It reflects the brain's continuous attempt to regulate its own affairs. Passive observation has not been successful in revealing many of its secrets. However, operant conditioning of EEG parameters has proven an effective "probe" into the regulatory role of different EEG features, often allowing scientists and clinicians to intervene productively to alter the state of the system in the moment and possibly to modify its long-term behavior as well.

Operant conditioning of EEG parameters is known as *EEG biofeedback* — more recently termed *neurofeedback* to distinguish it from its more pedestrian ancestry, that of biofeedback applied to peripheral physiology. Though clinical applications of EEG feedback have been extensive with childhood behavioral disorders, numerous other applications are reported in clinical research (Hammond, 2009) .

Origins of EEG Biofeedback for Addictions

In 1989, Eugene Peniston published a controlled study using EEG feedback with treatment-resistant Vietnam veterans suffering from alcohol dependency and PTSD. The feedback was adjunctive to standard treatment involving individual and group psychotherapy. The results were close to ideal in that the ten experimental subjects successfully shed their dependency, while the controls (who had received only the standard treatment) remained alcohol-dependent. This study drew attention to a method that had much earlier beginnings, but had since been eclipsed. It is described in more detail below. Strangely, even the biofeedback community was skeptical of this study, and it was ignored elsewhere. Nevertheless, a number of small replications appeared in subsequent years, all but two tending to support the original finding. Peniston also followed up with additional studies. Years later, a large-scale controlled study was done at a residential treatment center in Los Angeles, where the EEG feedback was again adjunctive to standard treatment. This study also replicated Peniston (albeit with modifications of the protocol), and it extended the claims to a number of other drugs beyond alcohol. Participant outcomes did not appear to depend on the drug of choice. At one-year follow-up, three out of four graduates had maintained sobriety. When outcomes were referenced back to initial intake, outcomes were three times better than for the controls! The study included a three-year follow-up period, over which time the experimental subjects continued to maintain their sobriety, whereas the controls continued to revert to pre-

treatment status.

Since that time, a small number of addiction treatment programs has incorporated neurofeedback. The lack of more robust growth of the technology in application to addictions treatment is puzzling. Perhaps its lack of growth and acceptance is attributable to the lack of familiarity with and education in the neurofeedback technique within the addictions treatment community. Occasional publications don't significantly shift the belief system within a community of professionals. Also, neurofeedback treatment does not conform neatly to models that most therapists know (which will hopefully change as more therapists become knowledgeable about the benefits of neurofeedback). Finally, institutional acceptance of new methods tend to be slower than for individual therapists, among whom this technique is already starting to thrive.

The Larger Context of Addiction

Compared with conventional treatment, neurofeedback is clearly faring better with the more intractable cases of addiction. More severe addictions involve more complex issues than just chemical dependency. The following features or comorbid conditions may accompany addictions:

- anxiety or depressive disorder
- undetected learning disabilities that diminish self-esteem and coping capacities
- deficits in executive function that predispose to behavioral disinhibition
- personality disorder, including propensities toward compulsive or antisocial behavior
- the addiction syndrome itself may have features of a personality disorder
- undisclosed or unrecognized minor traumatic brain injury that limits functionality
- an underlying reward deficiency syndrome

EEG biofeedback training can influence and modify each of the potential accompanying dysfunctions because each is governed by particular and accessible brain networks. Cases of dual diagnosis are thus broadly as addressable as straightforward cases of dependency. They may just call for tactical adaptations in applying the same basic technique and principles.

The common features observed in substance dependency share, as a unifying impairment, a disorder of *disregulation*, including, in particular, the state of addiction itself. So, the essential clinical challenge is to restore the cerebral networks to a better state of regulatory capacity. By virtue of the highly integrated and hierarchical organization of neural networks, this task is achievable with a very limited set of clinical protocols, as evidenced by the published studies. Importantly, because the technique is nonverbal and targets brain behavior directly, it bypasses the usual resistances 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 methods. Indeed, it falls in step with current

acknowledgment of the complexity of addictions and the need for integrative modalities to help patients recover and thrive. Neurofeedback itself is rarely employed as a stand-alone technique for addictions. With traditional treatments, there is a natural progression to reduce psychopharmacological agents and supplant them with psychotherapeutic support, both psychodynamic and behavioral. Successful neurofeedback should largely reduce or even displace the need for stimulants, anti-depressants, mood stabilizers, anti-psychotic medications, pain medications, and sleep aids. Neurofeedback training should also, over time, lead to greater insight, enhanced receptivity, and deeper engagement with a therapist. Throughout the process, a certain level of implicit trust is required for the trainee's brain to “admit and permit” reinforcement to do its work, which makes the therapeutic context for this work critically important.

Model of Brain Function Supporting EEG Neurofeedback

The continuing intractability of chemical dependency reinforces the view that addictive propensities and conditions are encoded in the physiology. Additionally, a strong predisposition to chemical dependency may be genetically encoded. 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 thought intractable might be amenable to recovery. The discovery legitimized the concept of brain plasticity, even for those researchers oriented entirely toward structurally-based models of chemical dependency.

The innovation of neurofeedback reaches well beyond a mere new wrinkle; By establishing successful outcomes in new proportions neurofeedback therapy has surpassed the meager success rate of nominally 25% for conventional treatment to attain a success rate of roughly 75%. This astonishing reversal in outcome probability compels a reappraisal of the mechanisms of addiction. Irrespective of genetic predisposition, recovery seems eminently and consistently achievable, using a neuro-behavioral technique. Given this success, we should consider addiction as a syndrome of learned behaviors that can manifestly be extinguished if the brain is given appropriate cues. The genetic heritage of an addict must simply be accepted as one constraint among many others that continue to make addiction treatment a challenge even under the best of circumstances. The bottom line, however, remains that the genetics of addiction are no longer immutable explanations, excuses for failures, or, thankfully, life sentences.

There are many possible points of departure for a discussion about brain behavior. This chapter addresses those features that are directly relevant to the modality of neurofeedback. For brevity's sake, consider three basic principles:

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

By appreciating how these principles underlie brain function, it is easier to understand the profound influence of EEG neurofeedback upon brain function and its frequent aberrations.

1. 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 connectivity and global interconnectivity characterize such networks and bring the whole entity into efficient global communication. In the human brain, both local connectivity and global interconnectivity are quite extensive. The neurons that manage more distant communications within the brain are the pyramidal cells, and these receive inputs locally through some 1,000 to 10,000 dendritic connections. When dendritic inputs happen to combine in such a way as to meet a threshold criterion, the neuron switches states and generates an action potential, a brief, large electrical transient that then becomes the bearer of information. Any action potential generated by the neuron progresses down its axon, which branches many times and thus also distributes its signal to many nodes in the same neighborhood as the dendritic tree. In addition to this local branching network, each pyramidal cell also sends its axon to connect to a distal cortical region via the white matter. Hence, global connectivity maximizes to a natural biological limit, with every relevant neuron participating. As a result, the human brain features synaptic junctions that are reachable from any other synaptic junction via only about three synaptic links. This biological intricacy (and intimacy!) bespeaks a staggering level of global integration in our brains resulting from the evolutionary process. We are wired in a way that marvelously maximizes and makes accessible all the global connectivity that is potentially available. Thus, every part of cortex can be made "aware" of what is going on in any other part at any time, yet this awareness is characterized by high selectivity.

2. 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 bottom rung are the cortical neurons, numbering about ten billion. At the next level are the thalamus and the other subcortical nuclei (the ones visible in medical brain imagery studies). At the top of the hierarchy is the brainstem. Because the brainstem's role is so endemic, its functions tend to be taken for granted, much like a heartbeat. At this top level, the integrity of our function is contingent on the behavior of mere thousands of neurons in the locus coeruleus. These neurons play a seminal role in the management of our nervous system. They are indeed the basis for psychopharmacological efficacy. All the medications that target neuromodulators target the brainstem that sources them.

The implications of these two basic organizing principles for neurofeedback are that any attempt to modify brain function communicates with the whole brain and with the entire regulatory hierarchy, all the way to the brainstem. Therefore, *neurofeedback reaches everything that is managed by this regulatory regime.*

In understanding how brain networks operate, central regulation of brain function is paramount. As to the basic rules of engagement that are implemented on this network, our interest at this moment lies with the matter of synaptic transport of information because it governs the central regulation of brain function. This represents only a minor portion of all the work done by neurons, but it is the part that EEG feedback accesses. Fortunately, the rules are again very simple. A single action potential can only propagate itself to a target neuron with the co-occurrence of another action potential at the same time. The basic operation performed by

the neuron, stated mathematically, is that of coincidence detection. This fact alone imposes highly specific timing constraints on all synaptic information transport because the timing uncertainty in this process is only about 10 milliseconds. The potential for functional deterioration of the transmission is obvious.

The role of the neuron is that of a correlation detector. Consider, however, that the brain could not function adequately where its integrity is dependent on the functionality of each individual neuron. Information is instead encoded in neuronal assemblies. The brain seeks precision through large numbers. These must necessarily act in unison. Hence, the brain must be organized for massive parallel processing. This gives rise to the problem of binding — the necessity to discriminate which neuron is part of the dance and which is not. Time binding has been proposed as an explanation for how the brain elegantly solves this problem. This is the principle that 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 impact is sufficiently large that this becomes observable at the scalp as the EEG. Through the EEG, therefore, we are in a position to witness the continuing activity of the brain as it constructs and then deconstructs neuronal assemblies. The brain also has to organize continuity of its own state. It does this by arranging for repetition of *nearly* the same firing pattern at a characteristic frequency. The result is that the whole EEG is comprised of numerous pockets of activity, each at its own frequency, and each rigorously separated from activity at neighboring frequencies.

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 more universal implications.

All these features of the neuronal assemblies either are, or should be, under tight constraints by the brain. Gross malfunctions show up in cases of seizure disorder, dementia, and traumatic brain injury. Though these are not the focus with addictions, observations of their correlations with the EEG give insight into the bigger picture of how the relationship between the EEG and brain self-regulation can also have relevance to addictions.

It is advantageous that, even when the EEG is well-behaved, it provides access to the brain's regulatory mechanisms through operant conditioning or even through subtle stimulation. Neurofeedback works by a reciprocal process: On one hand, neurofeedback “invites” the brain to alter its activity; on the other hand, the brain — as an entity seeking to maintain its integrity and stability — acts to counter this interference. This is

possible only because the EEG reflects tightly constrained brain activity. *The result of repeating this brain challenge is to strengthen the regulatory loops.*

Significantly, this process does not depend on deficiencies appearing in the EEG. Instead, this process draws upon the EEG being under reasonable control already. The process works to enhance regulatory control, not to bring it into existence in the first place. 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 in order to make a life of abstinence a realistic possibility!

3. Dynamic management of states

In treating addictions, it is critical to take into account the importance of arousal states and the management of affect in helping addicts break the addictive cycle. The dynamics of brain function and regulation underpin addictive dysfunction. However, it is the similarities in the *principles of brain dynamics* in both functional and dysfunctional patterns that provide the tools and capacities to intervene effectively. On the matter of brain dynamics, the famous neuroscientist Walter Freeman has expressed the key principle 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...”

Along the same lines, the following:

“The entire 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 insights 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 does matter a great deal which part of cortex is trained and which parts of the whole domain of EEG frequencies are targeted in the 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 of functional neuroanatomy and about localization of function is clearly of relevance here. Typically, the treatment of addicted patients does not target localized function at all, but rather very basic regulatory mechanisms that are most likely broadly distributed and de-localized.

It is best to think of this work, firstly, 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 collectively called *phasic and tonic arousal*. Engaging the EEG on the scalp necessarily impinges upon the regulation of arousal. The alcoholic brain is often contending with a tonic high-

arousal condition, to which neurofeedback offers a remedy. In fact, arousal regulation is the primary order of business for many substance abusers.

Secondly, affect regulation is a target of training in the addicted population, as so many of them exhibit concurrent mood instabilities or personality disorders. A growing school of thought ties personality disorders with disorders of attachment in early childhood. Through neurofeedback, the underlying circuitry governing emotional attachment is now 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 root causes of addictive persistence because:

1. It trains the brain to modulate overarousal, a maladaptive state that typically characterizes addicted populations and propels them to crave and indulge to mollify the overarousal.
2. It addresses the underlying, unremediated disorders of attachment that often have developed into maladaptive behavior patterns, personalities, and lifestyles.

Thirdly, the training broadly targets executive function. Impulsivity and behavioral disinhibition are indices of diminished executive function generally. Neurofeedback training moderates obsessive and compulsive tendencies and thrill-seeking behavior. Even more crucially, this training impinges upon issues of satiety and reward, modulating our drives ranging from appetite awareness to drug-seeking behavior. Concomitantly, neurofeedback leads to improvements in working memory and cognitive function. As functionality is restored to the neural networks in the proper hierarchy of regulation, patients experience greater mastery across the board, including cognitive function.

Satiety, Behavior, and the Brain

Neurofeedback essentially prepares the neural substrate for a drug-free existence. It is known that drug-seeking behavior is often the response to a felt need. One possible intervention is to train the nervous system to a level of functioning where that need no longer exists. This approach is illustrated by way of contradistinction to the use of naltrexone, which has the effect of preventing the person from enjoying the effects of alcohol.

Neurofeedback, on the other hand, can evoke the sense of equanimity, of euthymia, of safety, and even of pleasure that renders the resort to drugs superfluous. Importantly, neurofeedback does not replicate the euphoria associated with many drugs. Yet the enhanced capacity for feeling joy and pleasure throughout life does allow the person to sustain sobriety.

The need to train the brain in this fashion is particularly obvious in the case of 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 as well. The first obligation, then, of a comprehensive therapeutic remedy is to train the brain to the point where nicotine no longer presents the smoker with a performance benefit. Then, and only then, should the cessation of smoking take center stage.

The success of this strategy is illustrated most dramatically with marijuana. In many cases, clients gave up marijuana quite unintentionally once they undertook EEG training. People will routinely report at some point, “I forgot to use.” They are more surprised by this phenomenon than clinicians, who have observed it in many cases of treatment. This has also been reported for nicotine addiction.

The Role and Treatment of Trauma

All of the above falls in the domain of neurophysiological normalization. This may be a required element to sustain abstinence (cf., marijuana use that is driven by a felt need). But, it is only part of the story. A history of trauma makes addiction more complex, and the addiction is difficult to shed without healing the underlying trauma. Traumas are those events in which the individual's survival was suddenly threatened, or when life-threatening events were observed in those near and dear. Saddling even those without recognized overt traumas are the needs and yearnings for a type of healing known sometimes as “soul-work” — where the self has been crushed in some way and not allowed to find positive expression.

Neurofeedback can be helpful here as well. Through a process of rewarding low EEG frequencies under appropriate circumstances, treatment helps the person to encounter his “existential self.” Under these conditions, traumas are resolved, and a healing and reconstitution of the self often occurs. Routinely, people report these 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 does with his own resources. Using neurofeedback technology to facilitate this process is known as *Alpha-theta training*.

The role of Alpha-theta training is three-fold. Firstly, it physiologically, psychologically and perceptually distances the environment and allows the person to calmly focus inward. Secondly, it quiets cortical function in general and the verbal self-censor in particular. This allows the core self to emerge into awareness through imagery. Alpha-theta sessions are often suffused with a variety of imagery that is dream-like and hypnagogic in quality. Thirdly, the EEG becomes more coherent over larger brain regions, which enlarges the subjective boundary of the self and draws others into one's affective embrace. Key historical relationships that were problematic in the person's life can be quite suddenly restructured in a more positive frame. Early childhood traumas may be defused and integrated. In some instances, much of the benefit of this training can be traced to a particular session within the sequence of what is now called *deep-state* training. To observers unfamiliar with the results and its theoretical bases, this process can seem mysterious and unsystematic. Ironically, what may seem lacking in chemical recombinant specificity is, in actuality, a quintessential healing from within.

Recovery from treatment-resistant addiction typically requires attention to both aspects of training, improved cerebral regulation in first instance, followed by deeper healing of the injuries to selfhood. Ideally, the result can be transformational, and addiction will no longer fill a need or even have a place in the transformed life. In practice, the results often fall short of this ideal. However, neurofeedback has proven valuable in allowing people to maintain abstinence, recover function, and progress in their lives.

The Historical Development of Neurofeedback

A review of the history of neurofeedback is in order, both to establish its validity by conventional research standards and to render comprehensible its relative obscurity in light of its apparent efficacy. This also leads to the contemporary research on addictions. The deep-state training had its beginnings with Joe Kamiya, who first demonstrated successful reinforcement on alpha rhythms in 1962 (Kamiya and Noles, 1970). Then, Kamiya published a study demonstrating that alpha training was successful in moderating trait anxiety (Hardt and Kamiya, 1978). It was not long after the initial reports, however, before the alpha work caught the popular fancy. Reports of idyllic experiences with altered states brought the technique into favor with the psychedelic crowd who wished simply to avoid the downsides of the LSD experience. This quickly spoiled the technique for formal academic research, and the bit of research that was done seemed almost destined to fail. Alpha training fell quite precipitously into disrepute, and this is largely attributed to one negative outcome study (Lynch, 1974). Subsequently, only isolated clinicians and researchers carried the work forward, operating outside of the academic environment and without benefit of grant funding.

M. Barry Sterman pioneered the move in training toward physiological normalization. Sterman worked at the Sepulveda Veterans Administration Hospital in Los Angeles. Initially the work was thought to have limited applications to seizure management, and later to the “neurobiological deficit” of 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 continued carrying 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 nine-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 salubrious and constructive effect. There had been no “hitting bottom” or other crisis to launch the 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 its 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 they were highly suggestible in that state. The boundary of the self was more permeable (Budzynski 1972). Soon thereafter, however, Budzynski's interest diverted to the peripheral biofeedback modalities that were then becoming of interest, as well as being fertilized with funding.

Elmer Green and his colleagues (with the active support of Karl Menninger) conducted the principal thrust of work with Alpha-theta training (Green, 1970; Green, 1974). This research group considered alpha reinforcement 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).

Neurofeedback in Addictions Treatment: What the Research Shows

1. The pioneering studies

The foundational research of the biofeedback group at Menninger 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 some 76 subjects (Twemlow and Bowen, 1977). Significantly, the only predictor of self-actualization scores that emerged with EEG training was religiosity. This could well be traceable to the considerable likelihood that patients would 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 ten hours of alpha-training, and then compared to matched controls (Passini, 1977). The study yielded evidence of improvement on state and trait anxiety, and 18-month follow-up yielded evidence of reduced alcohol consumption (Watson, 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 some 54 measures) outside of chance expectation. This negative outcome is not at all surprising, given our modern understandings, as is argued below.

2. The Peniston studies

The study that revived interest in Alpha-theta neurofeedback for application to addictions was undertaken by Eugene Peniston, a psychologist then employed at the Fort Lyon Veterans Administration facility to work with Vietnam veteran alcoholics. He also adopted 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 in that his own 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 over-aroused states. The thermal biofeedback was augmented with autogenic phrases promoting the induction of calm states, as well as 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 particulars on these aspects of the protocol are described in a later publication (Peniston 1999).

Twenty participants were entered into the program, with ten of these getting the feedback in addition to the normal residential treatment program. Participants had to have a history of at least four prior treatment failures for their alcoholism. The average was 5.4. 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 experimentals were successful over the initial 18-month follow-up period, and, in fact, remained successful over the subsequent 10 years of more informal follow-up. In this context, *successful* means 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 the 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, these scientist-practitioners sought refuge in biofeedback using peripheral modalities. By 1990, biofeedback therapists and researchers did not want to be reminded of the trauma visited upon the organization by the rejection of the early alpha training. They were convinced they had seen the last of alpha band reinforcement, and good riddance.

Yet Peniston had brought considerable supportive data to bear. First of all, there were clear changes in the percentage of time patients exceeded threshold in the Alpha-theta training. Secondly, the experimental group demonstrated significant recovery on the Beck Depression Scale. Thirdly, 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 did not manifest in the experimentals.

Additionally, Peniston showed substantial normalization in subtest scores on the Millon Clinical Multiaxial Inventory (MCMI) and the Sixteen Personality Factor Questionnaire (16PF) among the experimentals (Peniston, 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, 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 support the observation of sustained sobriety.

By 1991, Peniston had published yet another study on the application of Alpha-theta training to PTSD (Peniston, 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 abide, 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 part of the story.

Twenty-nine Vietnam veterans were recruited into a study similar to the first. 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, psychasthenia, 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 re-hospitalization. All of the 14 controls had to be admitted to VA treatment centers at least two times 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, 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 disengaged, internally focused state that appears to be so therapeutically productive. However, outcomes were substantially consistent with the prior studies in these dual-diagnosis patients (PTSD and alcoholism). Follow-up among the twenty 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 of clinicians who were hardy and committed. Only a few of their findings have been published. In an outcome study, Saxby showed changes in personality variables similar to Peniston and documented sustained relapse prevention in 13 out of 14 participants over a 21-month follow-up (Saxby and 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 three-year outcome study of Alpha-theta neurofeedback training for problem drinking among the Dine' (Navajo) people (Kelley, 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.

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. Unfortunately, these results were never published.

3. The CRI-Help Study

During the mid-90's, the group at EEG Spectrum (headed by Siegfried Othmer, author herein) decided to undertake a large-scale replication of Peniston's work, with the principal modification that EEG training would replace thermal training prior to the Alpha-theta work. This transpired in collaboration with CRI-Help, a residential treatment center in Los Angeles. The intent was to assess the role of neurofeedback in as realistic a

situation as possible; hence staff at CRI-Help were trained to provide the service.

The study was intended to focus on alcoholism, but there weren't enough cases to meet group size objectives in reasonable time, so the study was opened up to include other drugs as well. Heroin, crack cocaine, and methamphetamine were represented at a nominal 30% in the treatment population, with alcoholism at 10%. Nearly all were multi-drug users. Everyone received the standard Minnesota Model treatment (individual and group psychotherapy imbedded in a 12-step program), with the experimentals also getting the neurofeedback, while the controls received additional individual and group psychotherapy as an attention control.

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 the slight modification that 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. Electrode placement was bipolar at C3-Fz for left-hemisphere training, and at C4-Pz for right hemisphere training. The principal objective in 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.

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 gets the frontal region explicitly involved. Right hemisphere training was done with a parietal bias to calm down phasic arousal. For more extensive treatment of this protocol and of the clinical results that were obtainable with it in this time frame, see Othmer (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.

With 121 participants entering the program, group size was adequate to withstand any potential 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 immediately when the training began. Even within the first couple of weeks of training at a rate of two sessions per day, a statistically significant group difference manifested in the retention rate. This is shown in **Figure 1**. During this period of time, only the SMR-beta training was done; this argues for a significant role for the SMR-beta training in the overall protocol. The proximate goal in this training was to normalize the measures on a continuous performance test (CPT) — the TOVA (Test of Variables of Attention). Participants were retested 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 (i.e., the median was 10).

The average TOVA scores for the two cohorts are reproduced in **Figure 2**. Remarkably, all the 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 had been homeless. About 30 percent 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.

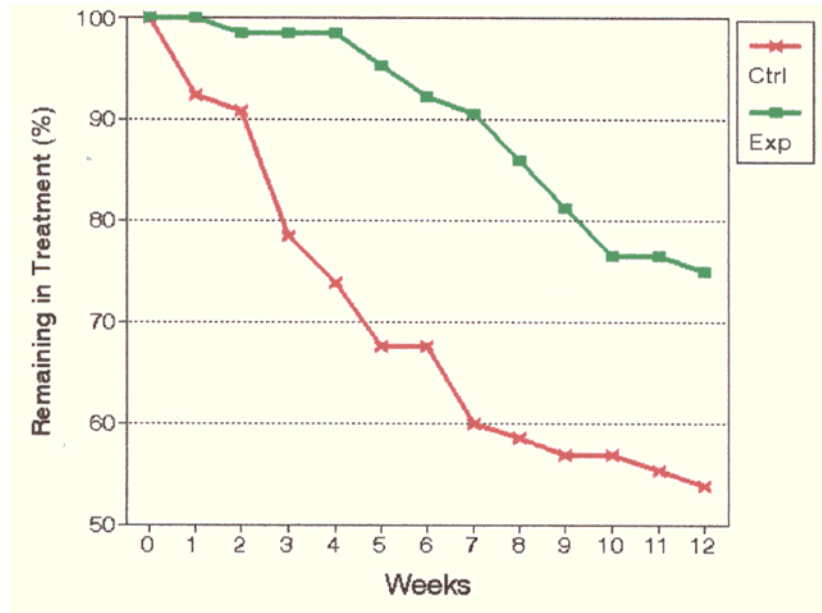


Figure 1. Retention in therapy for the experimental and control groups over the duration of the treatment program. Attrition rate for the controls was twice that of the experimentals. Significant differences appeared even during the SMR-beta phase of the EEG training.

A neurocognitive test battery was also given to all participants, but here the experimentals distinguished themselves from the controls significantly only with respect to the test of delayed recall. Though memory is one excellent test of brain function, on the basis of the good CPT data, one might have expected a broader range of improvements. Gains were also observed on immediate recall and the Tower Test, but there was no significant treatment interaction.

Following Peniston's method, the MMPI was also administered pre-post in this study, and the results mirrored his earlier data. The results are shown in **Figure 3** for the experimentals, and in **Figure 4** for the controls. A common thread that runs through all of these studies is a large change in the depression scale. Additionally, the CRI-Help study confirmed Peniston's work with respect to significant changes in the Hypochondriasis, Conversion Hysteria, and Schizophrenia scales. The consistent trends of these changes over all the studies indicate that this training clearly improved a number of aspects of the trainees' mental health status.

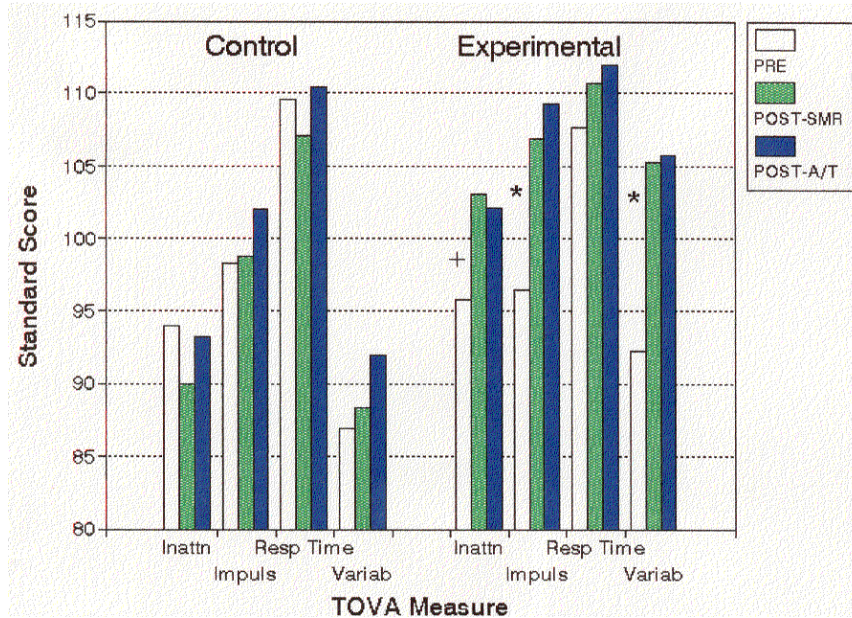


Figure 2. Pre-post standard scores are shown for the Test of Variables of Attention (the TOVA). Experimental subjects normalized all of their TOVA subtest scores with an average of 13 training sessions.

Relapse was tracked in these individuals over a three-year period, but has been formally reported only for the first year. The data are shown in **Figure 5**. In interpreting the data, one must consider that, at the end of the program, the graduating cohorts are no longer equivalent, due to the much higher attrition among the 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 one year than someone who just received the standard treatment alone. This turns a success rate of nominally 20% into a success rate of more than 60%.

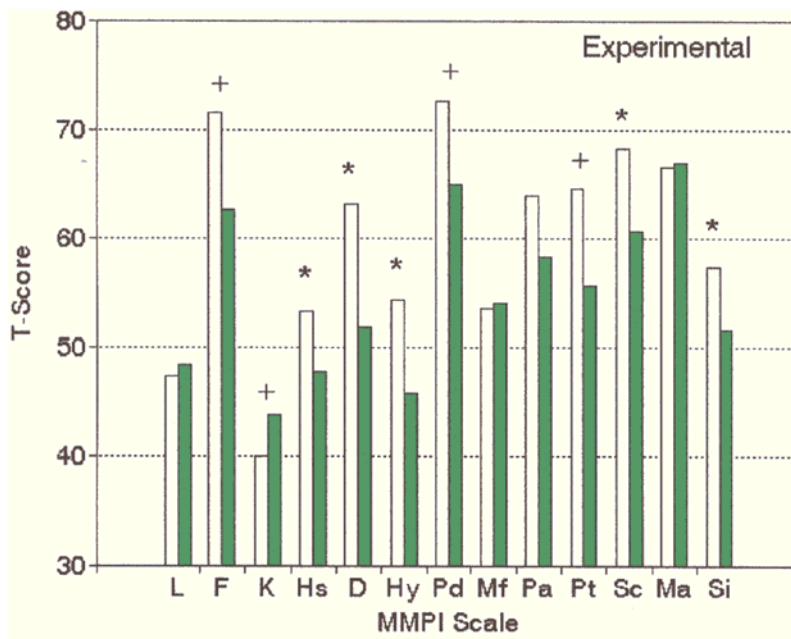


Figure 3. Pre-post MMPI data are shown for the experimentals. Substantial normalization of elevated scores is indicated. A star is used to indicate a significant treatment interaction, whereas a plus indicates significant change that nevertheless did not reach the level of significant treatment interaction.

Given the novelty of the neurofeedback approach, it was difficult to get this work published in a mainline journal. It finally appeared in the *American Journal of Drug and Alcohol Abuse* in August of 2005. The study had taken four years, and the subsequent path to publication had taken about five years.

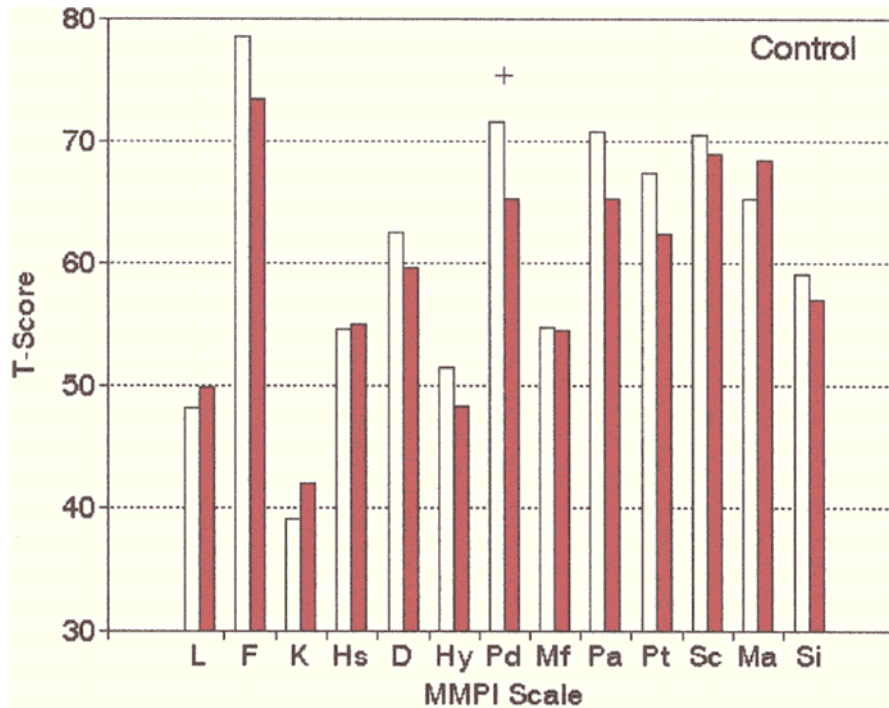


Figure 4. Comparative pre-post MMPI data are shown for the controls.

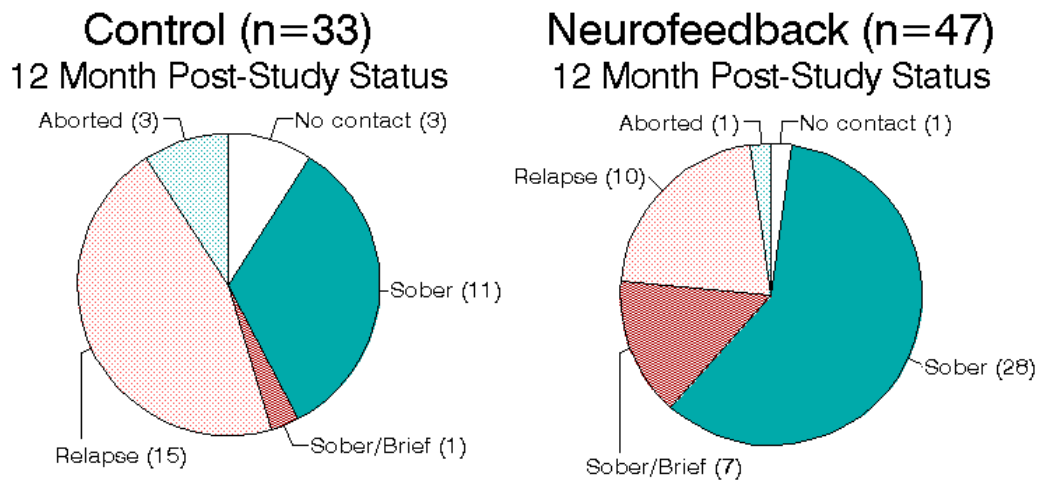


Figure 5. Relapse data are shown for the period 12 months post-graduation. Experimentals exhibit a three times higher success rate in maintaining abstinence if brief relapsers are included (35 versus 12)

4. The Replications of Peniston and of CRI-Help

Several replications of Peniston have been published in recent years. Bodenhamer-Davis published follow-up results for the Peniston protocol as used with chemically dependent probationers considered to be at high risk for re-arrest. They had undergone an average of 30 sessions of Alpha-theta training in the 1993-5 timeframe. Over the course of training, Beck Depression Inventory scores normalized from a pre-training range of mild to moderate, and MMPI-2 scores showed substantial improvements on seven sub-tests. Follow-up after seven to nine years confirmed 80% maintenance of abstinence. Rearrests over that timeframe were 40% versus 80% for a comparison group. (Bodenhamer-Davis, 2004).

Burkett et al. reported results mirroring those of Peniston and CRI-Help for a tough clinical population, namely homeless crack addicts (Burkett et al., 2005). A modified Peniston neurofeedback was added to a faith-based mission to the homeless in Houston. As in the CRI-Help study, the Alpha-theta phase was preceded by reinforcement in the low-beta range of frequencies (13-15 Hz in this case), with concurrent inhibition in the theta range. Placement was bipolar at T4-Fp1.

With the introduction of the neurofeedback, length of stay increased from 30 to 100 days. Substantial success was achieved according to multiple criteria at one-year follow-up: Some 90% of participants were housed; were either employed or in school; had remained arrest-free; and had experienced fewer than ten instances of drug use. Of these 50 percent had remained drug-free. The already startlingly good results obtained originally by Peniston had been exceeded, and this had been accomplished with an even more dysfunctional population.

Smith and Sams reported on a pilot study of neurotherapy in application to youthful offenders who were also substance abusers. The study was done in a therapeutic community where the youngsters typically stayed for twelve 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 (Smith, Sams, 2004).

Neurofeedback was also inserted into a 12-step-based program. Training was at a rate of four sessions per week; this program also involved heart rate variability (HRV) training, which is thought to impinge upon emotional responsivity. No Alpha-Theta training was done with this group. Both the neurofeedback protocols and the HRV training should be seen as primarily serving the objective of enhanced state regulation. The HRV training involves a more direct appeal to autonomic regulation, with its more direct connection to affective states.

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 CPT scores improved to normative expectations, with all effect sizes exceeding one standard deviation. Average MMPI scores (n=30) improved

from 64.5 prior 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 is 10 points. Average changes of ten 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 EMG biofeedback, Transcendental Meditation (TM), and Alpha-theta feedback with standard treatment in application to alcoholism (Taub, 1994). Whereas enhanced clinical success was achieved within the EMG and TM arms of the study, this success was not shared 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 failure was a university-based study 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 and 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, and they 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 averred that narrowly focused 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 the 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 (Graap and Freides, 1998). Peniston defended himself, but the controversy did not entirely subside. In any event, Graap and Freides never published the results of their study.

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.

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.

For completeness, it should be mentioned that a very different EEG training technique was applied to the problem of alcohol dependency in a European study. The technique of transiently altering the slow cortical

potential, which had been developed in application to seizure management, was employed with seven patients hospitalized for their alcoholism. In follow-up at four 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, 2003).

The Current Status

Since the time of Peniston's work, numerous enhancements to the protocol 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 changes have occurred since.

In the CRI-Help study, William Scott found a number of people to react badly to alpha reinforcement. Often, these people would show high alpha amplitudes at inception. In these cases, it was found beneficial to first train alpha down before the standard protocol was administered later. One may think of this as an additional training toward functional normalization and improved state regulation, as 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 under conditions of moderate arousal, and the second takes place in a state of low arousal, or deep-state training.

The observation that what is involved in deep-state training is mostly state shifts rather than EEG change specifically or functional normalization generally has found support in a study of Alpha-theta training unconnected to the problem of addictions. Egner and Gruzelier used a combination of SMR-beta training and Alpha-theta training with students at the Royal College of Music in London. In these studies, a substantial enhancement of musical performance was demonstrated, as judged by blinded professional musicians. More specifically it was found that the beneficial effects of the protocol could be attributed entirely to the Alpha-theta component (Egner and Gruzelier, 2003). A subsequent characterization of EEG band amplitudes throughout these trainings revealed that the secular trend of the alpha amplitude was actually downward during the training rather than up, and surprisingly the same tended to hold true even for the theta component. The theta-alpha ratio showed the influence of the reinforcement most unambiguously, and the common tendency of this ratio to increase over the sessions is taken as an indication of movement toward states of lower arousal (Egner and Gruzelier, 2004).

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 worthwhile.

A second change arose from the work of Les Fehmi and of Jim Hardt, who consistently rewarded alpha in a multi-channel configuration. The multi-channel setup 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 contra-indicated when the alpha band appears to be a zone of vulnerability, as it may be in cases of minor traumatic brain injury, fibromyalgia, and migraines.

The more significant evolution has occurred in the first stage of EEG 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 and shifting the reward frequency based on the person's response to the training has stretched the effectively reinforcing EEG spectrum up to 40 Hz and even down to 0.001 Hz (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 CPT. 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 the lower frequencies. The impact on affective states is much more immediate and compelling at low frequencies, with the additive benefit of a greater impact on felt states, which in turn promotes engagement with the process. One might term this *affective arousal*, in that the training productively addresses fear conditioning, trait anxiety, and affect dysregulation.

Functional Normalization and the Trauma Response

With the option of training at low frequency in conjunction with the optimization strategy, trainees naturally gravitate toward the training that they most need at a particular moment. 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 remediate. In turn, a two-pronged strategy has evolved for addressing the trauma formations that underlie many intractable addictions: The first training, currently referred to as *alert-state* training or *awake-state* training (formerly SMR-beta), addresses the physiological dysregulation, and the *deep-state* (Alpha-theta) training tackles the psychodynamic aspects. (Paradoxically, the alert-state training may transpire at lower EEG frequencies than the deep-state training, albeit under eyes-open conditions.)

The intimate connection between intractable addictions and trauma recapitulates neurofeedback application to a number of other conditions. The trauma connection is strikingly in evidence for severe chronic pain conditions, for the major eating disorders, and for intractable, severe migraines. It is also endemic in treatment-resistant fibromyalgia, chronic fatigue, and chemical sensitivities. It may also be fundamental in severe, intractable Tourette Syndrome. Finally, there is also a correlation of psychological trauma with disease mortality (Boscarino, 2008).

The significance of psychological trauma as a mediator of addiction is consistent with the model on which neurofeedback rests, in that its first impact on the organism is via the networks that perceive and process the

trauma. There is no physical injury involved. Psychological trauma starts as a “software” problem, which may then become progressively encoded in pathological network relations. This also serves as a priming event for subsequent traumas, thus escalating the person's vulnerability. The clinical intractability of the resulting syndromes gives the impression that the encoding of trauma must be structural and hence immutable. The efficacy of neurofeedback strongly implies that many pathologies are therapeutically accessible as a “software” problem. That is to say, they are immediately modifiable within the plasticity that is the natural property of neural networks through adaptive modification of synaptic strengths. And, this occurs even prior to new neuronal growth.

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 sub-cortical dysfunctions are accessible in imagery such as SPECT and PET. And, the stationary or steady-state properties of network linkages between different cortical sites can be derived from the quantitative EEG. Some network linkages become too intimate; others become too estranged.

From the standpoint of control system theory, a hierarchical needs assessment shows that the first priority must be to restore stability to the system. In cognizance of the network model of cerebral function, the second priority must be to restore the (vertical) hierarchy of regulation, from brainstem to cortex; this is in the service of state regulation generally. The third priority is to normalize the (horizontal) linkages between cortical regions where these may be deviant; this addresses specific deficits in cognitive function, sensory processing, and sensory integration.

By virtue of the high level of integration of neural architecture, any of the standard neurofeedback approaches will impinge on all three of these dimensions. 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 best assured by targeting inter-hemispheric timing relationships, etc. There are also known frequency relationships. Left-hemisphere cognitive function has a higher-frequency bias; right hemisphere affect regulation has a lower-frequency bias. In fact, the left hemisphere has a higher-frequency bias versus the right generally, and so forth.

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 *essential* self, the existential self that moves through time as an entity whose essential character is somewhat buffered from raw experience, maintaining continuity through all the vicissitudes of life. In this core self, organized around the limbic system, the stabilizing internal gyroscope is wound up, the moral compass is set, and the range and depth of emotional sentience are tuned. The addict requires attention to both domains, and both are

directly accessible through interaction with the neuronal networks (Freeman, W.J., 1999).

The Stealth Factor

In addition to psychological trauma, there is also the problem of minor traumatic brain injury (MTBI). These head injuries don't involve skull fracture, 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, 2008). So, MTBI could be a significant etiological factor in intractable addiction. The lack of structural evidence of injury (even in the rare cases it is sought) indicates that, as with psychological trauma, the principal mechanism of dysfunction must lie in the disturbance of network relations. There is much evidence for this in functional imaging, and we only allude here to by reference (Hoffman, 1996; Thornton, 2005). The excellent recovery achieved through neurofeedback further supports the functional model of MTBI (Walker, 2002).

One may consider both psychological trauma and minor brain injury as *stealth* conditions that have gone largely unrecognized when patients present for addictions treatment. We can attribute deficits related to both classes of trauma to degradations in network relations and to deterioration in the brain's capacity to self-regulate. The remediation of these deficits is necessary as part of addictions treatment. Fortunately, neurofeedback training substantially addresses these deficits, but they must be recognized and tracked through the therapy process.

This understanding also allows us to explain why a few of the early studies were not successful. Alpha reinforcement training is not likely to help people with unremediated traumatic brain injury, and may well even be counterproductive. This alone is sufficient to explain the ambiguous outcomes reported by Watson and Herder in application to psychiatric patients, as well as the negative outcome reported by Taub. For the vast majority of substance abuse patients, a narrow focus on the addiction status in treatment is unlikely to yield success. It is vital to address the whole penumbra of the condition. Much of this penumbra may have a trauma basis, but the broader implication is that has network regulation must be restored, irrespective of etiology.

Eugene Peniston brought all the necessary elements of the recovery process together systematically, perhaps for the first time. Subsequent attempts to distill from this work the singular "active ingredient" were flawed from their conception. There were many ingredients to the restoration of wholeness, all contributing to the one indispensable element of recovery: the restoration of adequate self-regulatory status as a preparatory step for the journey toward self-actualization. Succeeding developments have only built upon and refined the Menninger/Peniston procedure.

The Transformative Event

There is by now abundant evidence that the process of addiction recovery responds well to direct intervention using historically viable biofeedback techniques. Success is now fairly predictable, even for populations previously thought to be intractable. However, global appraisal of this process makes it clear that the treatment effects are far more propitious and far more general than one would expect from such limited direct intervention. 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 ultimately means a transformed life, a life in which addiction no longer fits.

This extension of healthy adaptation is a benefit that accrues well beyond the specific neurofeedback treatment protocols.

Sometimes the process of transformation is traceable to a singular event, one that typically occurs in the Alpha-theta phase of the training. This is nicely illustrated by an example reported by a therapist:

“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. 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 awhile, 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 become warm, friendly, and engaging since his Alpha-theta training experience.

This type of experience is not uncommon in the Alpha-theta training. Even more than with the training toward physiological normalization, one has the sense that neurofeedback simply sets the stage for this kind of growth. Witnessing such healing in patients leaves the therapist more humble in regard to his own role in this 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. Arguably, 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 hardly compatible. This misunderstanding may also have contributed to the failed replications of Graap and Freides, and of Taub.

It is surprising how some researchers can approach this paradigm with both skepticism and research procedures that could only produce positive results if the independent variables possessed magical powers. When the flawed designs fail to manifest desired outcomes, their suspicions are confirmed. Their original skepticism is assuaged and they move on.

However, treatment efficacy is more 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 striving for it. Having discovered that a modest yielding to the process was the path to success, he tried to practice that, but failed as well. It was necessary for him to truly surrender to the process, without any efforting or overt engagement of any kind. So, merely subjecting someone to this procedure for a certain length of time is not necessarily a valid test. It is crucial to determine whether the reinforcement actually eventuated in state change. That is likely to depend on variables such as trust, or a sense of safety, and even, as was seen in Fehmi's own case, a complete absence of striving toward an objective. These may not have been arranged for or adequately implemented in research settings.

One of the present authors (Othmer) had a similar experience with Les Fehmi's instrumentation: alpha amplitudes just would 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 puts the Taub study in a very different light. The success of the EMG and TM arms in that study just illustrates different pathways of getting to what is likely 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). When LSD was used in addictions treatment in the sixties, the experience of one or two LSD experiences during an extended six-month course of therapy made a large difference in outcomes that can only be attributed to these singular events. In three different universities, recovery at a 50% level was documented (Abrahamson, 1961).

Recovery is often dependent upon what is described as a “peak experience” (PE). More specifically, it is the affective component of the experience that matters. When subjects were first given penicillamine, their LSD experiences were identical except for a dampening of affect. The advantage for recovery, however, was thereby lost (Maslow, 1971).

Stanislov Grof routinely observed transformative events in the course of his holotropic breathwork (Grof, 2000). In this approach, hyperventilation leads to a state of hypocapnia (reduced CO₂ 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 entry into altered states. The peak experience, or transformative event, gives the person a glimpse of the actualized self, which then sets life upon a different trajectory.

The technique of eye movement desensitization and reprocessing (EMDR) similarly works by stimulating the low EEG frequencies where traumas are preferentially accessed. And, hypnosis typically moves subjects into states where the low EEG frequencies are dominant. Indeed, one of the present authors refers to Alpha-theta deep-state training as *EEG hypnosis*.¹

What differentiates modern Alpha-theta training from these other techniques is the precision with which intervention may be directed when one has access to the EEG. And what distinguishes the neurofeedback approach generally is the fact that one technique can address the various aspects of the dysregulated brain — the brain that also happens to be addicted.

The transformative event, when it occurs, leaves little doubt in any observer’s mind about its significance. But, it is not essential to the process, and it should not be an explicit objective. When we asked successful research participants at the three-year follow-up point of our CRI-Help study about what they credited for their success in maintaining abstinence, all of them cited the 12-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 remain beneath the person's own radar. Because of the way that this progress integrates with the developing self, accommodations are made in which the biofeedback contributions are taken for granted and subsumed in the newer version of the self that the person internally narrates.

Summary

This chapter provides a compendium of evidence that jointly illuminate the problem of addiction in a larger framework of what may be labeled *disorders of dysregulation*. Such disorders may have both a structural and a functional basis. However, addressing just the functional aspect of these disorders results in a surprising degree

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.

of recovery. Observing and modifying the EEG using the neurofeedback methodologies can change the dysregulation status of brain networks; this operant reinforcement strategy works toward the recovery of numerous domains of function. Such an approach represents an entirely new resource for conditions such as addiction that currently do not yield readily 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 treatment approaches. A comprehensive approach that addresses itself to all aspects of neuronal dysregulation manifestly enhances success in addictions treatment. The data compel consideration of EEG neurofeedback as an integral component of 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

The systematic recovery from addictions is more probable with the advances made available by combining modern neurophysiological and behavioral technology with traditional human care and connection. The model for these advances is the neural network encoding that makes the brain therapeutically accessible to direct intervention at the neuronal level. The technique of operant conditioning on EEG variables, combined with psychotherapy and pharmacological support, works to enhance clinical outcomes significantly, even with the most challenging clinical populations. The technique targets improved mental functioning generally, rather than the physiological dependence on drugs specifically.

The support for the larger therapeutic project of remediating mental functioning comprehensively— rather than treating addiction in isolation — is compelling in light of the prevalent comorbid conditions that are so therapeutically challenging: psychological trauma, minor traumatic brain injury, personality disorders, impulse control problems and behavioral disinhibition, anxiety, and depression. These comorbidities are also deficits in network functioning, and thus make possible a common approach to their substantial resolution.

The process of restoring the brain’s regulatory and cognitive functions using neurofeedback also heals psychological wounds by facilitating and inducing particular network states that favor trauma resolution, which in turn sets the stage for progress toward self-actualization.

References

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

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

- Boscarino, Joseph A., Psychobiologic Predictors of Disease Mortality After Psychological Trauma: Implications for Research and Clinical Surveillance, *Journal of Nervous & Mental Disease*. 196(2):100-107, February 2008.
- Budzynski, T. H., & Stoyva, J.M. (1972). Biofeedback techniques in behavior therapy. In D. Shapiro, T.X. Barber, L.V. DiCara, J. Kamiya, N. B. Miller, and J.M. Stoyva (Editors), *Behavior and Self-Control*, pp. 437-459. Aldine, Chicago.
- Budzynski, T. H. (1972). Some applications of biofeedback-induced twilight states. In D. Shapiro, T.X. Barber, L.V. DiCara, J. Kamiya, N. B. Miller, and J.M. Stoyva (Editors), *Behavior and Self-Control*, pp. 437-459. Aldine, Chicago.
- 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. *J. Neurotherapy*, 8(2), 138-140
- Cattell, R.B., Ebner, H.W., & Tatsuoka, M.H. (1970). *Handbook for the Sixteen Personality Factor Questionnaire (16PF)*. Institute for Personality and Ability Testing, Chicago.
- Egner, T., and Gruzelier, J.H. (2003). Ecological validity of neurofeedback: Modulation of slow-wave EEG enhances musical performance. *Neuroreport*, 14(9), 1221-1224
- Egner, T., and Gruzelier, J.H. (2004). The temporal dynamics of electroencephalographic responses to Alpha/Theta neurofeedback training in healthy subjects, *Journal of Neurotherapy*, 8(1), 43-57
- 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. *Alcohol. Clin. Exp. Res.*, **16**, 547-552.
- Farmer, S.F., Neural rhythms in Parkinson's disease, *Brain*, 125, 1175-1176 (2002)
- Fehmi, L.G. (1978). EEG biofeedback, multi-channel synchrony training, and attention. Sugarman, A., & Tarter, R.E. (Eds.). *Expanding Dimensions of Consciousness*. New York: Springer Publishing Company, pp. 152-182.
- Fehmi, L.G., & Robbins, J. (2007). *The Open-Focus Brain: Harnessing the Power of Attention to Heal Mind and Body*. Trumpeter.
- Freeman, W.J. (1999). Consciousness, Intentionality, and Causality. In *Reclaiming Cognition*, Nunez, R., and Freeman, W.J., editors. Imprint Academic, Thorverton UK
- Freeman, W.J. (2000). *How brains make up their minds*, Columbia University Press, p.109

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., Ready, D.J., Freides, D., Daniels, R., & Baltzell, D. (1997). EEG Biofeedback Treatment for Vietnam Veterans Suffering from Post Traumatic Stress Disorder, *J. Neurotherapy*, 2, 65-66

Graap, K., & Freides, D. (1998). Regarding the database for the Peniston Alpha-theta EEG biofeedback protocol. *Applied Psychophysiology and Biofeedback*, 23(4), 265-272.

Green, E.E., Green, A.M., and Walters, E.D. (1970). Voluntary control of internal states: psychological and physiological. *J. Transpersonal Psychol.*, 2, 1-26.

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

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

Hammond, D.C. (2009). *Comprehensive Neurofeedback Bibliography*,
<http://www.isnr.org/ComprehensiveBibliography.cfm>

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

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. C.T. Tart (Ed.), *"Altered States of Consciousness"*, Wiley, New York, 519-529

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

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

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

Nunez, R., & Freeman, W. J. (2000). *Reclaiming Cognition: The Primacy of Action, Intention, and Emotion*, Academic Press, New York

Othmer, S., Othmer, S.F., and Kaiser, D.A. EEG Biofeedback: An Emerging Model for Its Global Efficacy, Siegfried Othmer, Susan F. Othmer, and David A. Kaiser, in *Introduction to Quantitative EEG and Neurofeedback*, James R. Evans and Andrew Abarbanel (Eds.), Academic Press, San Diego, pp 243-310 (1999)

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.). Haworth Medical Press, New York, 109-136.

Passini, F.T., Watson, C.B., Dehnel, L., Herder, J., & Watkins, B. (1977). Alpha wave biofeedback training therapy in alcoholics. *J. Clin. Psychol.*, **33**, 292-299.

Peniston, E.G. & Kulkosky, P.J. (1989). Alpha-theta brainwave training and beta endorphin levels in alcoholics. *Alcoholism: Clinical and Experimental Results*, **13**(2), 271-279.

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. (1992). Alpha-theta EEG biofeedback training in alcoholism and posttraumatic stress disorder. *The International Society for the Study of Subtle Energies and Energy Medicines*, **2**, 5-7.

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.

Peniston, E.G. & Kulkosky, P.J. (1989, 1995). *The Peniston/Kulkosky Brainwave Neurofeedback Therapy for Alcoholism and Posttraumatic Stress Disorders: Medical Psychotherapist Manual*. Certificate of Copyright Office. The Library of Congress, 1-25.

Peniston, E.G., & Kulkosky, P.J. (1999). *Neurofeedback in the Treatment of Addictive Disorders*. Evans, J.R., & Abarbanel, A. (Eds.). *Introduction to Quantitative EEG and Neurofeedback*. Academic Press, San Diego.

Schneider, F., Elbert, T., Heimann, H., Welker, A., Stetter, F., Mattes, R., Birbaumer, N. and Mann, J. (1993). Self-regulation of slow cortical potentials in psychiatric patients: alcohol dependency. *Biofeedback Self Regul.*, **18**(1), 23-32

Singer, W. (2007). What do disturbances in neural synchrony tell us about autism? *Biol. Psychiatry*, 62, 190-191

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), 137-162

Twemlow, S.W., & Bowen, W.T. (1976). EEG biofeedback induced self-actualization in alcoholics. *Journal of Biofeedback*, 3, 20-25.

Twemlow, S.W., & Bowen, W.T. (1977). Sociocultural predictors of self actualization in EEG biofeedback treated alcoholics. *Psychological Reports*, 40, 591-598.

Twemlow, S.W., & Bowen, W.T. (1977). 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. *J. Neurotherapy*, 6, 31-43.

Watson, C.G., Herder, J., Passini, E.T. (1978). Alpha biofeedback therapy in alcoholics. An 18-month follow-up. *J. Clin. Psychol.*, 34, 765-769