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Brainwave Biofeedback for Addictive Disorder

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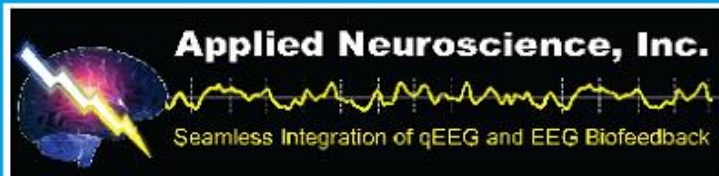
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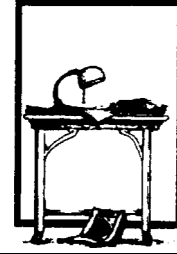
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Brainwave Biofeedback for Addictive Disorder

The first published account of brainwave biofeedback for addictive disorder appeared 33 years ago (Goslinga, 1975). Since then, much of brainwave biofeedback has focused on occipital alpha-theta training in a transformational paradigm of addiction recovery, described in this issue (Callaway & Bodenhamer-Davis; White) and critically reviewed (Sokhadze, Cannon, & Trudeau, 2008) in a recent issue of the *Journal of Neurotherapy*.

My interest in brainwave biofeedback for addiction dates back to 1991, when I first learned of the work of Eugene Peniston. As a career addictionist I was in private practice and had come to a mixed clinical and academic setting, the addictive disorders section of the VA Medical Center in Minneapolis, with an interest in learning more about why some recovered from addictions with standard treatment whereas others did not. I was especially interested in research opportunities and found nurturance and support to pursue these interests from the Chief of Psychiatry, Richard Magraw, MD, and later, Joseph Westermeir, MD, PhD. When I read Peniston's early work (Peniston & Kulkosky, 1989, 1990), I was struck with the possibilities of his method and the need for further research to validate his findings.

The guided trance state of alpha-theta feedback is subjectively impressive. I recall with clarity the first time I was "treated" to an alpha-theta session at the home of one of my colleagues from the Minneapolis Veteran's Affairs Hospital. I was given a quiet room and a reclining lounge chair, and I was hooked up to an analog trainer. There was no display of EEG, and thresholds were arbitrary dial settings. It was very crude by today's standard. I was told to relax and let myself have a reverie, allowing the tones to guide my state of consciousness. The 30-min session went by and I lost all sense of the passage of time while remaining awake and aware throughout. But what I best recall is the vivid imagery I had of my self in an area of emotional conflict that I was experiencing at the time. I was amazed at this experience, and I soon learned everything I could about alpha-theta training, EEG biofeedback, and acquiring the use of equipment.

My early experiences with others using alpha-theta were equally dramatic. Attending workshops at Menninger I found that vivid recall and transformational insights were common. I read the literature of the day, which is well summarized and documented in this issue by Callaway and Bodenhamer-Davis and by Sokhadze, Cannon and Trudeau (2008) in Volume 12, Issue 1.

As a result of these experiences I became interested in the following research questions: Does alpha-theta really work for addictions? How well does it work? Is there anything that makes it work better? Does it work for all kinds of addictions? Is its effect contingent on comorbid conditions? What exactly does it do to speed recovery? Does it promote insight, improve motivation to change, or remediate abnormal brain physiology in some way? Are there other forms of brainwave biofeedback that are as effective? How does one begin to parse the apparent phenomenon of addiction recovery being facilitated by brain wave biofeedback?

This issue of *Journal of Neurotherapy* contains a number of articles that begin to address these questions and add to the growing body of knowledge of neurotherapy for addictive disorders. Calloway and Bodenhammer Davis present long-term outcome findings in an independent cohort of alcoholic individuals treated with alpha-theta feedback. Although uncontrolled and a small sample size, this descriptive study illustrates the durability of the effect of the Peniston protocol. White offers a commentary on the dilemma of the clinician whose focus is on clinical methods utilizing alpha-theta feedback, when asked to consider critical analysis and empiric evaluation of the method. Both of these articles reinforce the classic views of the mechanism of the Peniston protocol as a psychotherapeutic process mediated by suggestibility and personal transformation that takes place in alpha-theta feedback sessions.

Three other articles in this issue report on approaches to addictive disorders that have the potential to become important clinical tools. Cannon, Lubar, Sokhadze, and Baldwin explore in a case study the effect of LORETA neurofeedback of the anterior cingulate cortex on the relationships between frontal and limbic structures. As part of a larger controlled study this work may have important implications for the relationships of personality traits and electroneurophysiology and neurofeedback in emerging models of addictive disorder. Sokhadze, Stewart, Hollifield, and Tasman report on executive dysfunction in cocaine addicts, using event related potentials and a dense

array. Their findings suggest that hypofrontality in cocaine addicts may be associated with poor ability to make choices when faced with drug cues or cravings. These findings are important for devising clinical strategies for cocaine addicts and also for assessing response to interventions. In the third study Sokhadze and colleagues look at cocaine addicts with and without posttraumatic stress disorder (PTSD). Patients with cocaine dependence and PTSD, as compared to patients with only cocaine addiction and control individuals, showed excessive cue reactivity to both drug- and trauma-related visual stimuli. These findings suggest that we must devise and assess interventions for cocaine addicts with PTSD, a common comorbidity. Both of these studies demonstrate frontal dysfunction in cocaine addicts, and their findings may be related to a model of why the Scott-Kaiser modification of Peniston's protocol (Scott, Kaiser, Othmer, & Sideroff, 2005)—namely, frontal theta suppression and beta enhancement prior to alpha theta training—works in mixed drug abuse populations.

Rather than considering addictive disorder as a unitary disorder and seeking a broad-brush treatment approach, the last three studies are considering the many variables that are unique to types of substance and coexisting conditions. They employ EEG technology that has evolved in recent years, such as source localization and evoked potential and dense arrays. They look to neurotherapy to help evaluate specific elements of a recovery process. But they are also similar to the alpha-theta studies in that they are seeking to bridge models of psychologic dysfunction with electrophysiologic findings. These new approaches represent the expected evolution of neurotherapy for addictive disorders over the course of 33 years. I am heartened to see this evolution happening.

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Guest Editor

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