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Neurofeedback in Treatment of Substance Abuse

By Dr. Stephen Sideroff, PhD

Over the last two decades a new research and clinical approach--neurofeedback--has shown promise in the treatment of substance abuse. This article addresses how it works, what makes it so effective, why it is a potentially important tool in addiction, the neurophysiological issues it might address, the existing promising research and, most importantly, that neurofeedback can be a significant adjunct...

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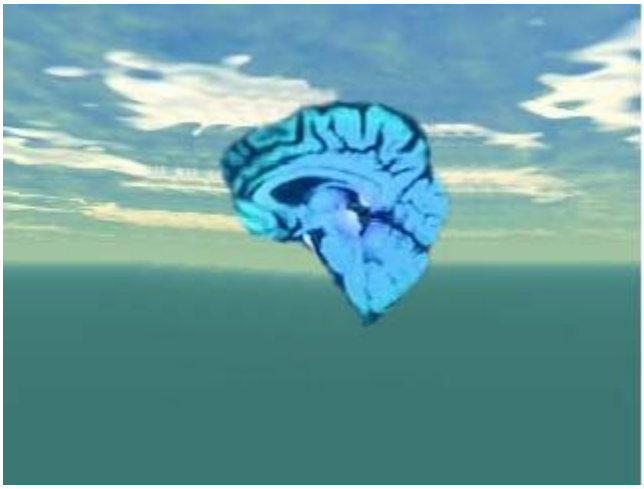

Editor's Note: This article is the first in a two-part series on Neurofeedback in the Treatment of Substance Abuse. This article presents evidence of the neurological basis, specifically EEG dysfunction, underlying addiction that makes it such a complicated condition to treat, and explains how neurofeedback addresses cognitive, emotional and physical symptoms. The second part of this article will include a discussion of the efficacy models of neurofeedback and a review of the research applying neurofeedback to substance abuse treatment, as well as address the possible mechanisms of its effectiveness in addiction.

Over the last two decades a new research and clinical approach--neurofeedback--has shown promise in the treatment of substance abuse. This article addresses how it works, what makes it so effective, why it is a potentially important tool in addiction, the neurophysiological issues it might address, the existing promising research and, most importantly, that neurofeedback can be a significant adjunct to the therapeutic and counseling process with addicts.

The category of disorders associated with substance abuse is the most common psychiatric set of

conditions affecting an estimated 22 million people in this country (SAMHSA, 2004). Furthermore, the disorder is accompanied by serious impairments of cognitive, emotional and behavioral functioning. These conditions and symptoms so significantly alter a person's brain and its functioning, that we often refer to the drug as hijacking the brain, making it very difficult to think logically and appropriately weigh the consequences of the drug related behavior.

Detoxified addicts have been shown to have significant alterations in brain electroencephalographic (EEG) patterns and children of addicts also exhibit EEG patterns that are significantly different than normal (Sokhadze et al., 2008, for review). This indicates that, not only are we dealing with the neurological consequences of drug-related behavior, but there appears to be a genetic pattern as well, that places certain people at greater risk for addictive behaviors. The complexity of these factors makes the treatment of addiction one of the most difficult areas of mental, emotional and physical rehabilitation.

Multiple factors in addiction

Treating addiction is compounded by the many factors contributing to its onset and maintenance. Furthermore, the addiction itself masks many other clinical conditions that become more evident once the drug user becomes abstinent. In fact, it is frequently other psychiatric problems that lead to drug abuse as the addict attempts self-medication. It has also been shown that people with cognitive disabilities are more vulnerable, and more likely to have a substance abuse disorder (Moore, 1998). These impairments appear to include attentional issues as well as the hypo-functioning of the frontal cortex, sometimes referred to as the executive brain, where decision making takes place (Fowler, et al., 2007).

As a result, we are learning that no one approach has all the answers. Multiple mechanisms require multiple considerations and approaches. In addition, addicts are a diverse group, resulting in the need for many tools and approaches. It appears that programs offering the most diversified array of treatment modalities are the most effective (Vaccaro & Sideroff, 2008). That is also why, for example, most programs urge the inclusion of a 12-step program for ongoing support.

But how do you address the biological and genetic aspects while also addressing the traumatic and emotional factors, the social cognitive and attentional factors? How do you deal with the apparent "procedural memory" and conditioned factors that cause an abstinent addict, on his or her way home from work, to all of a sudden take an inappropriate turn and end up at the drug dealer? Neurofeedback appears to be a tool, a training that has the facility to address many of these factors associated with addiction.

History of promising treatments

Over the years, there have been a number of developments that have been promising in the treatment of addiction. Each time a new approach is identified, it is immediately seen as being the long sought after "silver bullet" that will solve the addiction problem. This occurred with the development of methadone, and later Levo-Alpha Acetyl Methadol (LAAM). When I entered the field in 1976, as a post-doctoral fellow of the National Institute of Drug Abuse, Naltrexone was gaining popularity. Naltrexone is a long-acting opiate antagonist that blocks the effects of opiates, such as morphine, heroin and codeine.

It was around this time that the importance of addiction-related stimuli was becoming widely recognized (Wikler, 1984). In research examining the conditioned aspects of addiction, it was found that stimuli associated with the drug using behavior could serve as conditioned stimuli that would trigger an unconditioned psychophysiological response that had similarities to withdrawal and included anxiety, fear and physiological arousal (e.g. Sideroff & Jarvik, 1980). This conditioned patterning of response lead to the proposal that relapse liability might be determined by exposing addicts to these conditioned stimuli and monitoring their responses (Sideroff, 1980).

Following this conditioning model, one potential mechanism of Naltrexone treatment would be the behavioral extinction of some of the conditioned associations of addiction. In other words, if the addict

attempted to get high while on Naltrexone, the lack of reinforcing effect might lessen the conditioned effects of drug related stimuli. This, in turn, might reduce readdiction liability. All that needed to happen was for the addict to use, without experiencing any effect; a perfectly reasonable theoretical assumption. So, not only was Naltrexone expected to be successful in keeping addicts from using, but it also could address conditioned aspects of addiction.

When I arrived at UCLA and the Veterans Administration at Brentwood in 1976, I was surprised to discover that the treatment program to which I had been awarded a fellowship, was already eliminated--almost before it began. With the help of the director of the methadone clinic, I started a new experimental Naltrexone treatment program, drawing recruits from the VA's methadone maintenance population.

Unfortunately, Naltrexone did not meet its high expectations. While many methadone patients expressed interest in using Naltrexone, the long process of withdrawing from methadone--necessary in order to begin taking the opiate antagonist--eliminated more than 80 percent of volunteers. Also, as we enrolled volunteers, we found that 90 percent of the addicts who began using Naltrexone never used opiates while on the antagonist; and the 10 percent who did use, only used once. It was as if the addict immediately experienced this "no reward" condition and thus didn't bother to waste his money. This, in itself, was an interesting finding, as it showed this population to be able to demonstrate impulse control under certain circumstances (Sideroff et al., 1978). As a result, we never had the opportunity to test our theory of extinction.
The use of Naltrexone for opiate addiction has subsequently been viewed as an unworkable model. Yet, for the small fraction of individuals who were able to detox and begin taking Naltrexone, it did change their lives.

Typically, the "Silver Bullet" has been thought of in terms of a drug; something that could either eliminate craving or eliminate the high of the drug of abuse. What have become most useful, have been drugs of substitution, such as buprenorphine, (Johnson, et al., 2000), as we continue to search for an effective treatment combination that includes psychotherapy.

EEG and addiction
The EEG is one objective representation of how the brain is functioning. The EEG is recorded from scalp electrodes, and is a representation of electrical activity produced by the collective firing of populations of neurons in the brain, in the vicinity of the electrode. Figure 1 presents a chart of brain wave frequencies and the primary functions associated with their production. It should be pointed out that this is a gross representation and that more precise differences--beyong the scope of this article - can be found when you look at specific single frequencies within each range. While all frequencies and frequency ranges are important and necessary, problems arise when there is too much or too little of a particular type of brain wave; there is difficulty shifting in response to changing needs; or the EEG is to reactive.

For example, in a healthy functioning brain, if we look at the amount of theta being produced and we compared it (using 4-8 Hz) with beta frequencies between 13 and 21 Hz (cycles per second), there is approximately a 2 to 1 ratio. When we assess the EEGs of people with Attention Deficit Disorder (ADD), we see ratios that are 3 to 1 and much higher (Lubar, 2003).

These higher ratios indicate that the brain is producing too much of the slow waves relative to the beta waves, where the beta waves represent a more focused and engaged brain. In other words, these brains are under-activated. On the other hand, if we look at the EEG patterns of people with anxiety, worry and tension, there is typically too much activity occurring in the higher frequencies, usually between 24 and 35 Hz. The EEGs of people with substance abuse problems can show both of these patterns.

It has been demonstrated that the EEGs of addicts show specific abnormalities when compared to normative data. Studies of detoxified alcoholics indicate an increase in absolute and relative power in the higher beta range, along with a decrease in alpha and delta/theta power (Saletu, et al., 2002). Low

voltage fast desynchronized patterns (high beta) may be interpreted as demonstrating a hyper arousal of the central nervous system (Saletu-Z et al., 2004); and Bauer, showed a worse prognosis for the patient group with a more pronounced frontal hyper-arousal (Bauer, 2001).
The fact that these EEG patterns as well as alcohol dependence itself are highly inheritable further supports the biological nature of this disease (Gabrielli et al., 1982; Schuckit & Smith, 1996; Van Beijsterveldt & Van Baal, 2002).

These specific abnormalities show both a worse prognosis and a predisposition to development of alcoholism. Individuals with a family history of alcoholism were found to have reduced relative and absolute alpha power in occipital and frontal regions and increased relative beta in both regions compared with those with a negative family history of alcoholism. In another study, these abnormalities also were associated with risk for alcoholism (Finn & Justus, 1999).

It is a common belief that at least part of the cause of addiction is an attempt at feeling better--self-medicating. When someone with reduced or an absence of synchronous alpha rhythm takes a drink of alcohol, it results in the generation of an alpha rhythm or what is referred to as alpha synchrony, which a normal functioning brain has much greater capacity to produce (Pollock et al., 1983). Thus, it appears that the alcohol is helping the addicted person compensate for their brain's inability to produce an alpha rhythm which is associated with a state of calmness. This mechanism helps to explain the use of alcohol by this group of addicts.

In related research on abstinent heroin-dependent subjects, it is interesting to note similar abnormalities of deficits in alpha frequencies, along with excessive high beta EEG activity (Franken et al. 2004; Polunina & Davydov, 2004). Although it appears that in some studies, these changes found in early abstinence normalize after several months of abstinence (Shufman et al., 1966; Polunina & Davydov, 2004). Cocaine-dependent subjects may show similar increases in beta activity, but in addition show increases in frontal alpha (Herning, et al., 1994). These changes, specifically the elevation of fast beta activity, appear to be correlated with relapse in cocaine abuse (Bauer, 2001). In contrast, methamphetamine abusers have been shown to have significant increases in delta and theta frequency bands (Newton et al, 2003).

There are many questions that this research does not answer with regard to the relationship between abnormal EEG patterns and addiction. For example, it is not known if these dysfunctional elements are coincidental or causal. In addition, these EEG patterns are found in many mental disorders, some that are typically coincident with substance abuse. These questions do not minimize the probable conclusions that the EEG dysfunction creates specific vulnerabilities of these subjects. For example, frontal alpha, which is also found with some types of ADD, results in impairment of executive functions, such as decision making; and excessive fast beta activity can result in excess emotional and physical tension as well, as obsessive qualities.

Other substances of abuse have also been shown to correlate with abnormal EEG patterns. For example, studies have demonstrated that subjects with a chronic history of marijuana use demonstrate EEG patterns of frontal elevations of alpha frequencies. (Struve, Manno, Kemp, Patrick, & Manno 2003). This is referred to as "alpha hyper-frontality." Another common feature of the EEG of chronic users is a reduction of alpha mean frequency, which may indicate some deficits in intellectual functioning.

Neurofeedback

Neurofeedback, as a subset of biofeedback, monitors a subject's brain waves and feeds back selective information about these brain waves, in order to gain control over these patterns. Neurofeedback programs typically allow for the setting of thresholds within specific frequency bands or ranges so that when the EEG either rises above the threshold or drops below the threshold, some form of signal or reinforcement is presented to the subject. This feedback lets the brain know when it has been successful, thus, in an operant conditioning model, encourages this rewarded brain wave response. When the goal is to have the signal go above a threshold, we refer to this as "up training" or rewarding. When the goal is to reinforce signals that drop below a threshold, we refer to this as "down

training," or inhibiting this component of the EEG.

Joe Kamiya, a researcher at the University of Chicago, was the first researcher to discover that when a subject was informed that he was producing alpha brain wave frequencies, he would then be able to learn to detect, on his own, when he was in alpha (Kamiya, 1968). As a result of this finding, he designed a study in which he similarly gave feedback to the subjects as to their production of alpha, with the instruction to produce alpha. He found that when given this feedback, subjects were able to increase their production of synchronous alpha waves (Nowlis & Kamiya, 1970). Interestingly, his success led to the popularity of alpha training in mass culture, which coincided with its loss of credibility in the academic community.

Neurofeedback research and its acceptance took on a new impetus when Sterman, working with cats, was able to train these animals using a similar operant conditioning model, to increase the amount of synchronous spindle activity in the 14 Hz frequency range (Sterman, 2000). Since these spindles occurred over the sensorimotor cortex, he labeled them sensorimotor rhythm (SMR). These studies confirmed that the production of these brain waves--associated with motoric stillness--resulted in animals that were more resistant to the triggering of seizures. Sterman, then adapted this EEG biofeedback procedure with epileptic patients and demonstrated its effectiveness in reducing the frequency and intensity of seizures.

When a subject produces SMR activity, he is mentally alert with relaxed muscles (lower muscle tone). Lubar, working in Sterman's laboratory, recognized the potential of this discovery, and in a series of research studies, he and his colleagues were able to train children with hyperactive disorder to increase their production of SMR activity with feedback, resulting in reduced hyperactivity (Lubar, 1985).

The training procedures have evolved so that in addition to reinforcing SMR frequencies, the training of ADD also typically reinforces slightly higher frequencies of either 15 to 18, or 15 to 20 Hz activity, and at the same time, down trains the slower (theta) frequencies. The protocols address the ratio between the slower (theta) brain waves, with the faster brain waves, with a goal of training greater activation of the brain, which translates into improved attention. In one follow up study, Lubar and associates were able to demonstrate that gains made in variables of attention were maintained in subjects 10 years following training (Lubar, 1995; 2003).

At the same time that neurofeedback was being used to address attentional and cognitive deficits, primarily by training the activation of the brain, it also was being used to help people relax and establish autonomic and neuromuscular balance. With populations demonstrating aspects of anxiety, obsessive compulsive disorder and tension, the procedure has been to train increases in alpha frequencies (8-12 Hz) or a combination of alpha and theta (Moore, 2000). In these cases, the process is one of training a lowering of activation of the brain. A wide range of neurofeedback protocols have now been applied to cognitive, emotional and physical symptoms and conditions with a growing range of positive results. A bibliography covering these studies is available (Hammond 2008).

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