

SPECIAL ISSUE

Clinical Outcomes in Addiction: A Neurofeedback Case Series

Jay Gunkelman, QEEG-Diplomate,¹ and Curtis Cripe, PhD²

¹Q-Pro Worldwide, Crockett, CA; ²Q-Pro Worldwide, Crossroads Institute, Scottsdale, AZ

Keywords: EEG/QEEG, addiction, phenotype, neurofeedback

This case series (N = 30) shows the impact of an addiction treatment approach that uses phenotype-based neurofeedback in an integrated clinical treatment (Crossroads Institute), which combines targeted brain recovery exercises and neurotherapy. We present pre- and post-neurocognitive testing and electroencephalography/quantitative electroencephalography measures of the phenotype findings in this polysubstance-based addict population. The electroencephalography phenotypes identify two separate drive systems underlying individual addiction: central nervous system overactivation and obsessive/compulsive drives. In addition to sobriety and abstinence, the neurocognitive improvements documented are particularly impressive.

Background

According to a survey by the National Institute on Drug Abuse (NIDA), addiction is characterized by compulsive cravings, drug seeking, and drug use, which persist in the face of consequences (Substance Abuse and Mental Health Services [SAMHSA], 2006). For many, addiction is a chronic condition, with relapses occurring even after long periods of abstinence. Relapse rates are quite strikingly similar to rates for other chronic medical illnesses such as asthma. Like any chronic illness, addiction treatment generally requires repeated and persistent intervention to extend the time between relapse as well as to diminish the relapse severity and duration. Through treatment, people with drug addiction can lead productive lives.

The U.S. Substance Abuse and Mental Health Services Administration states that chemical dependency, along with associated mental health disorders, has become one of the most severe health and social problems facing the United States. In the United States, 12.5% of the population has a significant problem with alcohol or drugs, with 40% of these individuals having a concurrent mental/nervous disorder (the so-called dual diagnosis). The medical costs are approximately 300% higher for an untreated alcoholic than for a treated alcoholic. About 70% of addicts are employed, with their addiction contributing substantially

to absenteeism, turnover costs, accidents/injuries, decreased productivity, increased insurance expenses, and even workplace violence.

Costs related to addiction include those related to violence and property crimes, prison expenses, court and criminal costs, emergency room visits, health care utilization, child abuse and neglect, lost child support, foster care and welfare costs, reduced productivity, and unemployment. Of Americans aged 12 years or older, 22.5 million need treatment, but only 3.8 million people receive it (SAMHSA, 2006).

The 2004 survey also shows an estimate of the costs to society of illicit drug abuse alone, which is estimated at \$181 billion. If you include health care, criminal justice, and lost productivity, the costs exceed \$500 billion. Successful treatment can help reduce this cost, the associated crime, and the spread of many infectious diseases (SAMHSA, 2006).

It is estimated by NIDA that for every dollar spent on addiction treatment programs, there is a \$4 to \$7 reduction in the cost of drug-related crimes. With some outpatient programs, total savings can exceed costs by a ratio of 12:1 (SAMHSA, 2006).

For most programs, the ultimate goal of treatment is to enable an individual to achieve abstinence. Of equal importance is the patient's ability to improve function and minimize medical and social complications of the addiction. This allows the individual to reintegrate back into society in a productive manner. Abstinence from drug exposure itself is important, but the quality-of-life measures are also critical to true recovery, which is the thrust of this case series.

Neurofeedback for Addictions

Traditionally, addiction is treated as a behavioral disorder; however, it has recently been recognized as having brain-based components as well. In the field of neurotherapy, the efficacy of treatment for addiction using neurofeedback alone has not yet been fully established based on the quality of the research required for proof of efficacy in our field (La Vaque et al., 2002). It may never be established if clinicians

apply a single-modality treatment program, because of the multifaceted aspects of the disorder.

Early work using neurofeedback by Eugene Peniston provided a solid hint that our field may prove to have an enhanced tool that will add to the efficacy of treatment over traditional 12-step and counseling approaches to addiction, although these early studies were admittedly flawed. Peniston's treatment protocol is a multimodal intervention, integrating alpha-theta neurofeedback, thermal biofeedback, diaphragmatic breathing, autogenic training, emotional catharsis, and guided imagery and visualization (Peniston & Kulkosky, 1989, 1990). Later studies (Bodenhammer-Davis & DeBeus, 1995; Scott & Kaiser, 1998) have replicated the Peniston approach and have added to our understanding a need to include a modified Peniston approach for some individuals whose nervous systems are not ready for the full Peniston protocol. Sokhadze, Cannon, and Trudeau (2008) have reviewed the entire scientific literature on the use of neurofeedback for substance abuse in a white paper approved by both the Association for Applied Psychophysiology and Biofeedback and International Society for Neurofeedback and Research and concluded that both the Peniston alpha-theta protocol and the Scott-Kaiser modification of the Peniston protocol can be regarded as "probably efficacious," using the efficacy categories and criteria established by La Vaque et al. (2002).

The Concept of Phenotypes

The diagnosis of addiction is behavior based, yet current research shows that addictions have a biological basis and that this basis has a genetic component. This linkage between addiction and genetics has commonly been a vague reference, but solid evidence now exists for a genetic component, and this genetic component links electroencephalography (EEG) and genetics to addiction. This linkage between genetics and behavior is not direct: It has an intermediate step, and not all genes are expressed. This intermediate step is the endophenotype or, to use a more commonly used term, *phenotype*.

Many researchers in the neurofeedback field think of EEG subtypes within diagnostic groups, such as an alpha subtype of attention-deficit disorder (ADD)/attention-deficit hyperactivity disorder, or an alpha subtype of depression. Yet these subtypes are not specific to these disorders and are better conceptualized as phenotypical divergence patterns (see Johnstone, Gunkelman, & Lunt, 2005), or phenotypes. In other words, one divergent EEG pattern, or phenotype, may contribute to the disturbance in several different diagnostic categories.

These EEG phenotypes can be used to predict the optimal therapy for an individual, including choice of

medication or choice of neurofeedback training protocol. Medication prediction has been documented in the work of Arns, Gunkelman, Breteler, and Spronk (2008) and the neurofeedback predictions in the work of Gunkelman (2007) and Gunkelman and Johnstone (2005).

The reader should note that the full treatment for this clinical population is not restricted to neurofeedback but rather follows a biopsychosocial model encompassing a multimodality approach. This study is intended to add to the biopsychosocial model the understanding that phenotype-based neurofeedback, done in combination with targeted brain recovery exercises, form the basis for a tool to assist addiction-related recovery.

Methods

This case series represents our experience with clinical outcomes within our addicted population completing therapy with the Crossroad Institute's addiction program. The outcomes represent a noncontrolled study addressing observed results for a clinical case series, comprising the first 30 clients who completed therapy at Crossroads Institute in their Brain Recovery Program for substance abuse disorder. The program uses a biopsychosocial model that includes a core component of phenotype-directed neurofeedback, along with targeted individualized neurocognitive rehabilitation programs and nutritional and counseling approaches. As a case series, it was not designed as a randomized controlled study. This study does not allow us to make any claim of superiority for this approach over any other clinical approaches, nor is that our intent. We provide these data to show pilot-level outcomes to prospective granting agencies. The case series illustrates the pathophysiology associated with addiction and generally shows the impact of this approach on the various neurocognitive outcome measures of functional performance for our participants as well as the impact in their addictive behavior.

Because of the small sample size ($N = 30$), this case series does not validly show a detailed incidence of phenotypes across the clinical population, as many more clients are needed to achieve any predictive validity for some of the smaller phenotype clusters. It does generally characterize the phenotypes in this population as well as the effects on client's outcomes.

No claim of treatment efficacy is being made for neurofeedback based on this work alone (see the field's efficacy hierarchy for appropriate standards, and Yucha and Montgomery's new 2008 summary of evidence-based practice). These case outcomes show more than merely having clients with sobriety/abstinence and quantitative EEG (QEEG) patterns. People's lives are changed when the underlying phenotypical patterns are addressed.

EEG Phenotypes in Addiction

The actual distribution of phenotypes in addiction is not yet fully described and will require many more cases to allow the smaller groupings to populate reliably. However, based on this preliminary work, it appears that two different neural factors underlie the preponderance of addiction cases, likely representing separate pathophysiologic drives for addictive behaviors: (a) central nervous system (CNS) overarousal ($n = 21$) and (b) cingulate issues (obsessive-compulsive; $n = 9$).

Neurofeedback protocols are all derived from the EEG phenotype(s), not the behavior of addiction. The two addictive drive factors contain a few phenotypes, with the neurofeedback intervention for each phenotype described previously (Gunkelman, 2007; Johnston et al., 2005), and the interventions are generally described below:

CNS overarousal-based drives ($n = 21$) were ascribed to the following phenotypes:

- low-voltage fast EEG phenotype ($n = 8$), which gets sensory motor rhythm (SMR) and alpha-theta training;
- faster alpha frequencies ($n = 7$), which get alpha-theta training; and
- beta spindle ($n = 18$), which gets SMR training and then alpha-theta.

Cingulate-based obsession-and-compulsion-based drives ($n = 9$) were ascribed to anterior midline divergences, centered at Fz, with the neurofeedback approach used to suppress the individual's localized divergent EEG pattern. Specific phenotypes were as follows:

- Alpha ($n = 3$)
- Slow (theta) ($n = 2$)
- Beta spindling ($n = 4$)

Following neurofeedback training, the EEG changes are observable and measurable, and although this is not a formal analysis, we can report our general experience. We find that the effect of alpha-theta training on the EEG is to decrease beta levels following training, which is similar to the findings of Tobias Egner at The Royal College of Medicine in London. The alpha and theta training corresponds to training hypoperfusion, and the result is decreased beta (which is associated with hyperperfusion). With SMR training, we see a reduction of cortical excitability, much like Serman and Egner's (2006) published findings. For the cingulate-related training (Congedo, Lubar, & Joffe, 2004), we see EEG improvements and behavioral improvements associated with cingulate function, including improvements in cognitive and emotional flexibility and reductions in obsessive/compulsive and oppositional behavior.

Detailed pre-post QEEG analysis will await grant funding, although our experience shows substantial reduction in overarousal and improvements in cingulate function, as

appropriate to the individual case's findings, following the completion of the training and therapy.

Clinical Outcome Data

Our outcomes are not merely abstinence or sobriety from the client's drug of choice but reflect a more fundamental improvement in neurocognitive function, as seen in our routine pre-post neurocognitive testing.

Although it should be noted that these are relatively short-term outcome results, the average time of abstinence for these 30 clients at the time of the write-up was more than 18 months, far beyond the commonly discussed 6-month placebo washout time. Longer-term follow-up will be provided over time.

Three case reviews are included here to help provide some perspective on the treatment programs our clients are actually involved in.

Paul is a 36-year-old male opiate addict with a long history of relapse and treatment failure. Paul entered the drug rehabilitation program, where he was given a brain map and treated with the neurodevelopment program, psychotherapy, Alcoholics Anonymous (AA) recovery program, and group therapy. He was randomly drug tested throughout his treatment. Paul's EEG/QEEG results revealed physiological correlation with his memory impairment, cognitive ability impairment, and his extremely poor impulse control. Paul worked the program for 5 months of inpatient treatment and then stepped down to sober living for 10 months. He continued to undergo random drug testing. The work Paul did in the neurodevelopment program included resolution of his memory and cognition problems and his poor impulse control issues. Paul was able to improve his cognitive abilities by at least 45% overall, based on the Woodcock-Johnson III scales used in this study (Woodcock, McGrew, & Mather, 2001). Paul currently is 15 months sober and is working a strong recovery program.

Nick is a 22-year-old drug addict who has had several prior treatment failures elsewhere. Nick's treatment program included the EEG/QEEG, psychotherapy, neurodevelopment program, and an AA recovery and sober living program with random drug testing. Nick's EEG/QEEG showed strong a correlation with his very poor impulse control, severe memory problems, adult ADD, and developmental delay related to very low cognitive abilities. After 8 months of treatment, Nick was able to resolve his memory, learning, developmental delay, and adult ADD problems. His cognition has increased by 48% (also based on the Woodcock-Johnson III scales used in this study). His functional IQ has increased 22 points, and Nick's impulse control has stabilized him to the point at which, coupled

with a solid AA recovery program, he currently is sober going on 9 months.

Jerry is a 20-year-old man given a diagnosis of drug addiction and schizophrenia. Jerry spent 6 months in a drug rehabilitation center, where he was treated with psychotherapy, psychopharmacological medications, and an AA recovery program. Jerry was referred for an evaluation including EEG/QEEG, and he entered the neurodevelopment program, psychotherapy, sober living, and AA recovery. Jerry's EEG/QEEG suggested that the diagnosis of schizophrenia was incorrect. The EEG data suggested that Jerry's semantic memory systems were impaired, and he had evidence consistent with developmental delays, poor impulse control, and severe learning problems. The focus of Jerry's treatment program for the next 13 months was neurodevelopment, psychotherapy, AA recovery program, and sober living with random drug testing. Jerry's goal was to be able to attend college.

After 13 months in the program, Jerry was able to build a sober network of support. His cognition has improved by 44%, and his developmental and learning problems were resolved. Jerry has met his goal in college: He is an A student. He has been sober for 2 years.

Some quantification of the neurocognitive testing changes associated with our phenotype-based neurotherapy approach is seen in measures that were taken both before and after treatment. Measurements are expressed as standard scores, with 100 being a normal performance. The Table lists the change in the group mean values comparing the various measurements before and after treatment.

These measurements are taken from the Woodcock-Johnson III scales (Woodcock et al., 2001). The first metric is a General Intellectual Ability measure. This score is considered a measure of the fluid intelligence of the individual and is crudely equivalent to an IQ score. The Thinking Ability metric measures thinking processes used when short-term memory information cannot be processed automatically. Cognitive Efficiency measures the ability to

process information automatically, with tasks requiring that information be held in working memory and also visual perceptual speed. Audio-Visual-Learning Ability measures learning in situations in which information is presented both orally and visually. Delayed Recall is a measure of both auditory and visual recall after a 30-minute delay. Working Memory measures the ability to hold information in immediate awareness while performing mental operations on the information. The following graphics represent the groups' before and after average scores.

Discussion

Some perspective is needed regarding what we are showing with our case series of addiction clients.

The value of a case series is in providing pilot data to guide the design of more formal and well-designed studies. As such, we are reporting our outcome experience with 30 clients who completed a full biopsychosocial-based multimodal therapy, detailing the logic of our clinical approach, and showing the impact of our clinical approach on the EEG and the impacts on the various behavioral measures, including abstinence/sobriety. These data may prove valuable in designing a more rigorous controlled study, and we hope it is helpful in understanding the pathophysiology underlying addiction.

This case series features the impact of the use of neurofeedback when it is based on phenotypes (applied to addiction in this circumstance), although it should be kept in mind that these phenotypes are not restricted to this diagnosis. The client's phenotype(s) drive the treatment approach selected, regardless of the diagnosis, as detailed in a prior publication by the first author in *Biofeedback* (Gunkelman, 2007).

After reviewing the 30 cases, it became obvious that two basic factors formed the pathophysiologic basis for addictive behaviors: Overarousal of the CNS is one drive mechanism, and a separate drive factor is based on a cingulate disturbance, associated with an obsessive-and-compulsive-based addiction.

The actual treatment was based on a biopsychosocial-based multimodal therapy and was more than just neurofeedback. This case series is representative of expected outcomes from real therapeutic environment clients, drawn from our experience at the Crossroads Institute's centers, and is not a controlled experiment. No claim of efficacy is made for neurofeedback based on this work alone.

Outcomes include more than merely having clients with sobriety/abstinence and happy therapists watching QEEG patterns change. People's entire lives are changed when the underlying phenotypical EEG failure patterns are addressed, as seen in the dramatic changes in the behavioral measures we presented.

| Table. Changes in neurocognitive abilities | | |
|--|------|-------|
| Test | Pre | Post |
| "IQ" (Woodcock Johnson III Cognitive Abilities Test) | 99 | 120 |
| Thinking Ability | 103 | 122 |
| Cognitive Efficiency | 94.7 | 118 |
| Audio-Visual-Learning Ability | 88 | 112 |
| Delayed Recall Ability | 65.8 | 103.6 |
| Working Memory | 93 | 122 |

Acknowledgment

Special thanks to the clinical and support teams at Crossroads Institute, Scottsdale, Arizona.

References

- Arns, M., Gunkelman, J., Breteler, M., & Spronk, D. (2008). EEG phenotypes predict treatment outcome to stimulants in children with ADHD. *Journal of Integrative Neuroscience*, 7(3), 1–18.
- Bodenhamer-Davis, E., & DeBeus, M. (1995, Fall). *Neurotherapy for the treatment of alcohol and drug abuse: Results of a two-year study*. Paper presented at the meeting of the Society of Study of Neuronal Regulation, Scottsdale, AZ.
- Congedo, M., Lubar, J. F., & Joffe, D. (2004). Low-resolution electromagnetic tomography neurofeedback. *IEEE: Transactions on Neural Systems & Rehabilitation Engineering*, 12, 387–397.
- Gunkelman, J. (2007). Transcend the DSM using phenotypes. *Biofeedback*, 34, 95–98.
- Gunkelman, J., & Johnstone, J. (2005). Neurofeedback and the brain. *Journal of Adult Development*, 12(2–3), 93–98.
- Johnstone, J., Gunkelman, J., & Lunt, J. (2005). Clinical database development: Characterization of EEG phenotypes. *Clinical EEG and Neuroscience*, 36(2), 99–107.
- La Vaque, T. J., Hammond, D. C., Trudeau, D., Monastra, V., Perry, P., & Lehrer, P. (2002). Template for developing guidelines for the evaluation of the clinical efficacy of psychophysiological interventions. *Applied Psychophysiology and Biofeedback*, 27, 273–281.
- Peniston, E., & Kulkosky, P. (1989). Alpha-theta brainwave training and B-endorphin levels in alcoholics. *Alcoholism: Clinical and Experimental Research*, 13, 271–279.
- Peniston, E. G., & Kulkosky, P. J. (1990). Alcoholic personality and alpha-theta brainwave training. *Medical Psychotherapy: An International Journal*, 3, 37–55.
- Scott, W., & Kaiser, D. (1998). Augmenting chemical dependency treatment with neurofeedback training. *Journal of Neurotherapy*, 3(1), 66.
- Sokhadze, T., Cannon, R., & Trudeau, D. (2008). EEG biofeedback as a treatment for substance use disorders: Review, rating of efficacy, and recommendations for further research. *Applied Psychophysiology and Biofeedback*, 33, 1–28.
- Sterman, M. B., & Egner, T. (2006). Foundation and practice of neurofeedback for the treatment of epilepsy. *Applied Psychophysiology & Biofeedback*, 31(1), 21–36.
- Substance Abuse and Mental Health Services Administration (SAMHSA). (2007). *Results from the 2006 National Survey on Drug Use and Health: National findings* (Office of Applied Studies, NSDUH Series H-32, DHHS Publication No. SMA 07-4293). Rockville, MD: Department of Health and Human Services.

Woodcock, R. W., McGrew, K. S., & Mather, N. (2001) *Woodcock-Johnson III*. Itasca, IL: Riverside.

Yucha, C., & Montgomery, D. (2008). *Evidence-based practice in biofeedback and neurofeedback 2008*. Wheat Ridge, CO: Association for Applied Psychophysiology and Biofeedback.



Jay Gunkelman



Curtis Cripe

Correspondence: Curtis Cripe, PhD, Crossroads Institute, 9328 E. Raintree Dr., Scottsdale, AZ 85260, email: curtis@myhealthybrain.com.

JOIN! Join the discussion on
AAPB list servs.

People are talking. As a special member benefit, you can join the conversation via the AAPB list servs. The first is for the entire membership and is called AAPB-membership. And the second is for the Neurofeedback Division members and is called AAPB-neurofeedback. Plus, many of the individual sections have their own list serv available.

To join the list(s) go to:

AAPB_Members-join@lists.aapb.org
AAPB_Neurofeedback-join@lists.aapb.org



We hope you enjoy
this new member
benefit of AAPB.