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SPECIAL ISSUE

Quantitative Electroencephalography-Guided Versus Scott/Peniston Neurofeedback With Substance Use Disorder Outpatients: A Pilot Study

Roger J. deBeus, PhD, BCIA-EEG

Department of Psychiatry and Behavioral Sciences, Quillen College of Medicine, East Tennessee State University, Johnson City, TN

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The present study evaluated quantitative electroencephalogram (QEEG)-guided and Scott/Peniston neurofeedback compared with a wait-list control in the treatment of substance abuse in an outpatient setting. Participants completed an intake assessment, 40 neurofeedback sessions, and a posttraining assessment. Change scores of the clinical scales of the Personality Assessment Inventory were used for outcomes. Compared with controls, QEEG-guided neurofeedback resulted in improvement on three scales, whereas Scott/Peniston training resulted in improvement on two scales. Findings showed significantly decreased symptoms of anxiety, schizophrenia, alcohol problems, and drug problems. The changes in outcome scores related more strongly to the participants' diagnoses and predominant drugs of abuse than to the type of neurofeedback intervention. Due to the small numbers in this study, efficacy differences between the two neurofeedback approaches were inconclusive.

Introduction

The field of neurofeedback currently has two major approaches for treating substance abuse: (a) alpha-theta neurofeedback conducted with eyes closed and (b) sensorimotor (SMR)-beta neurofeedback conducted with eyes open, followed by alpha-theta. Alpha-theta neurofeedback has been used successfully with alcoholics (Peniston & Kulkosky, 1989) and with alcoholics with depressive symptoms (Saxby & Peniston, 1995), but not with stimulant users (Fahrion, 2002). The second approach, known as the Scott/Peniston protocol, has been used successfully with inpatient polysubstance users (Scott, Kaiser, Othmer, & Sideroff, 2005) and with inpatient, homeless crack/cocaine addicts (Burkett, Cummins, Dickson, & Skolnick, 2005). Neurofeedback training has helped substance-abusing patients remain in treatment longer and has improved abstinence rates (Scott et al., 2005). However, more rigorous scientific investigation is needed (Trudeau, 2005). Past research has failed to assess comorbidities that may impact outcomes and also has failed to collect or report any electroencephalogram (EEG) changes associated with training. Finally, protocols were developed from clinical theories and by trial-and-error, which is impressive given such success but clearly lacks a validated theoretical basis for making decisions about training protocols.

Using a quantitative electroencephalogram (QEEG) to direct neurofeedback interventions is a recent advance of this field. QEEG analysis takes EEG records beyond basic clinical application and quantifies brain waves into frequencies and amplitudes. This information is compared with established norms to determine statistical deviations, which may be used to prioritize sites and frequencies for making decisions about training for a given individual. Neurofeedback training is then targeted to moderate abnormalities in brain activation, as identified by the QEEG. As most neurofeedback clinicians know, everyone responds differently to training, but a QEEG assessment reduces much of the guesswork in deciding where and what to train.

There is an abundance of research, using QEEG as an assessment tool, on the effects of substance abuse on brain activity. Alcoholic patients typically exhibit alterations in beta activity (Bauer, 2001) and/or alpha activity (Finn & Justus, 1999). Drugs such as cocaine, cannabis, heroin, and methamphetamine present their own specific QEEG abnormalities, sometimes according to the phase of abstinence and recovery (see Sokhadze, Cannon, & Trudeau, in press, for a review). Abnormal QEEG patterns also are seen in mental disorders that often are comorbid with substance abuse, such as conduct disorder (Bauer & Hesselbrock, 1999), attention-deficit/hyperactivity disorder (ADHD) (Bauer, 1997), and bipolar or major affective disorder (Oluboka, Stewart, Sharma, Mazmanian, & Persad, 2002). Given all these possible presentations, it only seems prudent to utilize QEEG measures to drive an intervention designed for normalizing electrophysiology.

The purpose of this pilot study was to compare QEEG-guided and Scott/Peniston neurofeedback interventions with a wait-list control group. This study adds several new facets

to the investigation of neurofeedback with substance abusers. First, we conducted the study with outpatients attending sessions only four times per week. Second, every participant completed a diagnostic assessment. Third, QEEGs were collected before and after the intervention and were used to determine neurofeedback protocols. QEEG results will be presented in a later article. The primary hypothesis for this pilot study was that QEEG-guided neurofeedback would result in more improvements in personality functioning than the Scott/Peniston protocol.

Methods

Twenty-five participants were recruited from an outpatient substance abuse program. All participants were informed of the study parameters and signed an Institutional Review Board–approved informed consent document. Participants were required to remain in an outpatient substance abuse program during the study. Four participants dropped out before the initial assessment and two more dropped out after starting sessions.

The study consisted of three phases: (a) pretreatment assessment, (b) neurofeedback sessions, and (c) posttreatment assessment. Those performing the pre- and posttreatment assessments were blind to group membership. Both assessment points included a structured clinical diagnostic interview, intelligence testing, personality testing, ADHD rating scales, a continuous performance test, and a QEEG. For this study we focused our analysis on the clinical scales of the Personality Assessment Inventory (PAI) (Morey, 1991). One of the benefits of using the PAI is that all the scales are independent of each other, which helps to separate various clusters of symptoms more effectively.

After the preassessment, participants were placed randomly in one of three groups: (a) QEEG-guided neurofeedback; (b) Scott/Peniston neurofeedback; or a (c) wait-list control. In the QEEG-guided group, neurofeedback protocols were identified using the results from the NX-Link Neurometric database. A decision tree was used with absolute and relative power *z*-scores to identify which sites and bandwidths to reward and which to inhibit. Protocols were changed when the participant was able to change the average amplitude for a specified bandwidth by 20% in the desired direction within a session compared with baseline (i.e., first 3 minutes of session).

In the Scott/Peniston group, neurofeedback protocols were based on preassessment Integrative Visual and Auditory (IVA) (Sandford & Turner, 1995) test results and a symptom checklist for the initial 10 SMR-beta sessions, followed by the participants receiving 30 alpha-theta sessions. This is a minor variation of the Scott/Peniston protocol, because their participants completed an average of 13 SMR-beta sessions and used the Test of Variables of Attention (TOVA) (Greenberg & Waldman, 1993) at the beginning, as well as after sessions 10 and/or 15 to determine when to change the protocol (Scott et al., 2005).

The Scott/Peniston group starting protocol consisted of beta training 50% of the time and SMR training 50% of the time. These percentages might be altered based on preassessment IVA results, with inattentive or impulsive profiles resulting in increased beta or SMR training, respectively. Before each session, sleep symptoms were monitored to determine response levels to training and general asymmetric arousal (Tucker & Williamson, 1984). For example, if the participant was generally not feeling rested after his or her sleep, the amount of beta training would be increased. When the participant reported difficulties falling asleep, the amount of SMR training was increased. Beta training consisted of augmenting 15–18 Hz with active bipolar placement at C3-FpZ and SMR training augmenting 12–15 Hz at C4-PZ, based on the international 10-20 system of electrode placement (Jasper, 1958). At the same time, training inhibited elevated activity in the 2–7 Hz and 22–30 Hz ranges. The next 30 alpha-theta sessions were conducted with participants' eyes closed. The active electrode was placed at PZ with a left-ear reference (A1) augmenting alpha (8–11 Hz) and theta (5–8 Hz). The initial sessions were used to train down alpha levels that were above 12 μV (peak to peak), while augmenting theta until there was "crossover." This was defined as the point at which the alpha amplitude drops below the level of theta. Subsequent to the first achievement of crossover, both alpha and theta frequencies were augmented. Before initial crossover was achieved, excess EEG activity in the range of 15–30 Hz was inhibited. This was intended to reduce muscle tension and to quiet the mind. After crossover was achieved, the 2–5 Hz frequency range was then inhibited. This was intended to discourage the sleep transition during low-arousal states.

Both neurofeedback groups participated in 40 sessions, with 30 minutes of training for each session. Sessions occurred once a day, four times per week. A script based on Peniston's original protocol (Peniston & Kulkosky, 1989) was read at the beginning of each session for both treatment groups. Talk therapy was not included as part of neurofeedback treatment, because participants already were receiving this in their outpatient program. The wait-list control group received only standard outpatient substance abuse treatment. The control group returned after 3 months, retook the assessments, and was then offered treatment. Anyone in the group who accepted the offer was placed randomly in one of the two treatment groups.

Table 1. Demographics, substances of abuse, and diagnoses of participants

	QEEG Group *n* = 7	Scott/Peniston Group *n* = 6	Control Group *n* = 6
Gender: (male/female)	4/3	4/2	4/2
Age: (average/range)	44.9/39–57	38.3/22–57	44.3/37–58
Years education: (average/range)	15.4/12–18	14/12–18	15/12–18
Drugs of abuse:			
Alcohol (primary/secondary)	6/1	6/0	5/0
Cocaine (primary/secondary)	1/1	0/3	1/1
Cannabis (primary/secondary)	0/0	0/2	0/1
Axis I diagnoses:			
Alcohol dependence	6	6	5
Cocaine dependence	1	2	1
Cocaine abuse	1	0	0
Cannabis dependence	0	1	1
Posttraumatic stress disorder	2	0	0
Obsessive-compulsive disorder	1	2	0
Social anxiety	3	2	1
Depression	3	2	1
Axis II diagnoses:			
Borderline	1	1	0
Antisocial	0	1	0

Note. QEEG = quantitative electroencephalogram.

Results

Participant demographics, substances of abuse, and diagnostic information are listed in Table 1. There were 12 men and 7 women in the study, 10 of whom were polysubstance users. The primary drug of choice within each group was alcohol, which was used by 95% of participants. The other drugs of choice included cocaine (used by 37%) and cannabis (16%). All participants remained alcohol/drug-free (based on urine measures) while participating in this study. From a diagnostic standpoint, members of both treatment groups presented with more Axis I diagnoses than did controls, particularly in the anxiety spectrum.

Primary outcomes consisted of 11 clinical scales of the PAI. In order to assess treatment outcome, a change score was computed for each scale (i.e., posttreatment score minus pretreatment score). Positive values denote an increase in PAI symptom severity from pretest to posttest. Negative values denote a decrease in symptom severity. A zero value indicates that symptoms remained unchanged.

PAI scale scores have a mean of 50 and a standard deviation of 10. Reliability studies indicate that the PAI is stable over a period of 3–4 weeks (test-retest stability correlations range from .79–.92 for clinical scales) (Morey, 1991). From a clinical standpoint, a change of 10 points on a PAI scale score can indicate a significant clinical difference in functioning. On the PAI all participants responded in a forthright manner, were consistent in responding, and avoided making an effort to create a positive or negative impression.

Experimental groups were compared with controls on change scores by means of *t* tests for independent samples. Analyses between the two treatment groups found no differences. As shown in Table 2, compared with the control group, the QEEG-guided group improved on three scales and the Scott/Peniston group improved on two scales. Confidence intervals (CI) are reported, indicating the likely range of the true values 95% of the time with the type of population in this study. The lower (or numerically smaller) CI limit shows how small the effect might be in the population; the upper

Table 2. Between-groups change scores comparisons				
PAI Scale	QEEG vs. Control	95% CI	Scott/Peniston vs. Control	95% CI
Somatic complaints	–3.7	3.4, –10.9	–1.67	5.4, –8.7
Anxiety	–10.7**	–3.7, –17.7	–8.0**	–2.7, –13.3
Anxiety-related disorders	–6.5	2.0, –14.9	–4.8	3.2, –12.8
Depression	–8.1	2.0, –18.2	–1.0	8.0, –10.0
Mania	–3.0	3.7, –9.6	–3.7	4.0, –11.3
Paranoia	–5.6	1.6, –12.7	–3.5	3.7, –10.7
Schizophrenia	–6.6*	–1.2, –11.9	–5.8	0.1, –11.8
Borderline features	-4.0	4.9, –13.0	–6.8	1.4, –15.0
Antisocial features	–3.0	6.8, –12.8	–5.5	5.2, –16.2
Alcohol problems	–26.9***	–10.3, –43.5	–12.2	2.4, –26.8
Drug problems	–11.9	5.5, –29.2	–17.0*	–1.7, –32.3

Note. Means of change score differences are presented under each comparison heading. Negative numbers denote improvements in pre to post functioning compared with control group. QEEG = quantitative electroencephalogram; CI = confidence interval.

*$p < .05$, **$p < .01$, ***$p < .005$.

limit shows how large the effect might be. If the CI does not overlap zero, the effect is considered to be statistically significant. Effect sizes (ES) were calculated using the population standard deviation of the PAI, because group sizes were so small. The Figure shows a visual representation of the change scores' differences for the two comparisons.

The QEEG group significantly improved on three PAI scales compared with the control group: Anxiety (change difference = –10.7, $p < .01$, CI = –3.7 to –17.7, ES = 1.07), Schizophrenia (change difference = –6.6, $p < .05$, CI = –1.2 to –11.9, ES = .66), and Alcohol Problems (change difference = –26.9, $p < .005$, CI = –10.3 to –43.5, ES = 2.69).

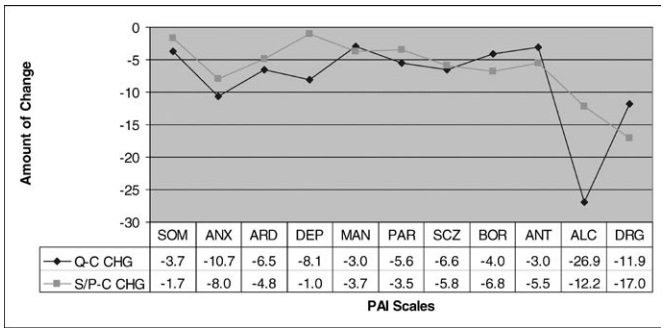

Figure. Change scores of neurofeedback treatment groups versus control group. Q–C CHG represents QEEG group change scores minus control group change scores. S/P–C CHG represents Scott/Peniston group change scores minus control group change scores.

The Scott/Peniston group significantly improved on two PAI scales compared with the control group: Anxiety (change difference = –8.0, $p < .01$, CI = –2.7 to –13.3, ES = .80) and Drug Problems (change difference = –17.0, $p < .05$, CI = –1.7 to –32.3, ES = 1.70).

Discussion

This study was successful in applying a neurofeedback intervention with abstinent, substance use disorder outpatients participating in sessions four times per week. We also found that concurrent participation in standard substance abuse treatments (i.e., Alcoholics Anonymous/ Narcotics Anonymous meetings, group therapy, and individual psychotherapy) was beneficial to the participants. After completing neurofeedback the participants made such statements as: "I feel like the 'edge' is no longer there"; "I still have some cravings, but the severity and frequency have significantly diminished"; "The cravings no longer drive me like they use to, 'I' am in control now."

One consideration of whether neurofeedback is successful with this population is obviously motivation. Our participants were motivated to do something different, and most found that neurofeedback helped them. Although we did not formally measure motivation, coming to our clinic

four times per week for 10 weeks is one indication. With the few who dropped out, it was evident within the first 2 weeks that motivation was lower, because they showed up inconsistently for their appointments. Future research may benefit from measuring motivation as a mediating factor for neurofeedback.

The study consisted mainly of alcoholics (95% using), some cocaine users (37%), and cannabis users (16%). Of the participants, 53% were polysubstance users. The primary hypothesis for this study was that QEEG-guided neurofeedback would result in more improvements in personality functioning than the Scott/Peniston protocol. Results indicated the QEEG group improved more than the Scott/Peniston group did, but only marginally. Analyzing change scores between the treatment groups and control group showed improvements in symptoms of anxiety, schizophrenia, alcohol problems, and drug problems.

Anxiety disorders and substance use disorders commonly co-occur. Data from the National Comorbidity Survey (Kendler, Gallagher, Abelson, & Kessler, 1996) indicate that individuals with anxiety disorders are 2–3 times more likely than the general population to have a substance use disorder at some time in their lives. Brady (2001) described the relationship between anxiety disorders and substance use as complex and varying greatly among individuals. In some cases, the substance use disorder may develop as an attempt to self-medicate anxiety symptoms. In others, anxiety symptoms are side effects of substances of abuse and/or occur during withdrawal states. In some individuals there is likely to be a cyclic interaction: Depressants, such as alcohol and opiates, may be used in an attempt to decrease anxiety, but during withdrawal states anxiety is increased, leading to an exacerbation of the anxiety disorder and making relapse to substance use more likely.

Other than substance use diagnoses, it is of interest to note that participants in this study showed more anxiety-related diagnoses than depression (see Table 1). In relation to the PAI Anxiety scale, both treatment groups reduced ruminative worry, subjective feelings of apprehension, and physical signs of tension and stress. The relationship between reduction of anxiety scores and the amount of patient anxiety-related diagnoses is one of the main findings in this study. These findings further support that neurofeedback can be an important coping strategy and method by which substance abusers can manage some of their anxiety without the use of medications or substances of abuse.

The QEEG group showed improvements on the PAI Schizophrenia scale. None of the participants in this group were schizophrenic, however, a review of the subscales indicated that participants had subclinical difficulties with these symptoms. Results indicated improvements in the areas of poor social competence and social anhedonia, as well as disturbances in attention, concentration, and associational processes. These findings may be related to the social anxiety and attentional problems found with substance abusers (Thomas, Randall, & Carrigan, 2003).

The alcohol and drug problem improvements were divided between the two treatment groups. In relation to the Alcohol Problems scale, the QEEG group improved in behaviors and consequences of alcohol use, abuse, and dependence, including loss of control and alcohol-related cravings. Although the Scott/Peniston group showed a large ES on this measure, it comprised more polysubstance users (5 of 6) than alcohol-only users (1 of 6). The QEEG group had fewer polysubstance users (2 of 7) than alcohol-only users (5 of 7). This may explain why the Scott/Peniston group improved on the Drug Problems scale whereas the QEEG group did not, at least statistically. The Scott/Peniston group improved in behaviors and consequences of drug use, abuse, and dependence, including loss of control and drug-related cravings.

Another of the main findings of this study indicated that the presenting drugs of abuse impacted the respective measured outcomes but not necessarily the type of neurofeedback intervention. More specifically, the QEEG group improved more on the Alcohol Problems scale because the group had more participants who used alcohol only versus multiple drugs. The Scott/Peniston group improved more on the Drug Problems scale, because the group contained more polysubstance than alcohol-only users. Given the large ESs seen on the alcohol and drug measures that did not reach statistical significance, we can speculate that either neurofeedback modality would impact both alcohol and drug functioning. However, our conjecture is limited due to the small size of this study.

In summary, as in previous studies, this study has continued to show that neurofeedback is an effective intervention with substance abusers. The changes in outcome scores related more strongly to the participants' presenting diagnoses and predominant drugs of abuse than to the type of neurofeedback. Significant improvements were found with symptoms related to anxiety, alcohol problems, and drug problems with moderate to very large ESs. Because the participant numbers in this study are small, it is not possible to draw conclusions regarding any advantage regarding QEEG- versus Scott/Peniston-specific training using this current analysis. Future directions of this research include analyzing the QEEGs to determine the impact of these two types of interventions on electrophysiological functioning.

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Roger J. deBeus

Correspondence: Roger deBeus, Center for the Advancement of Human Potential, 30 Garfield Street, Suite D, Asheville, NC 28803. Email: roger.debeus@att.net.