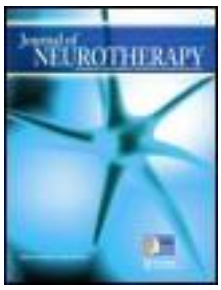


Journal of Neurotherapy: Investigations in Neuromodulation, Neurofeedback and Applied Neuroscience

QEEG-Based Protocol Selection: A Study of Level of Agreement on Sites, Sequences, and Rationales Among a Group of Experienced QEEG-Based Neurofeedback Practitioners

Mark L. Johnson MS [a] & Eugenia Bodenhamer-Davis PhD [a]

[a] University of North Texas , Denton

Published online: 26 Feb 2009.

To cite this article: Mark L. Johnson MS & Eugenia Bodenhamer-Davis PhD (2009) QEEG-Based Protocol Selection: A Study of Level of Agreement on Sites, Sequences, and Rationales Among a Group of Experienced QEEG-Based Neurofeedback Practitioners, Journal of Neurotherapy: Investigations in Neuromodulation, Neurofeedback and Applied Neuroscience, 13:1, 41-66

To link to this article: http://dx.doi.org/10.1080/10874200802668416

Journal of Neurotherapy, 13:41–66, 2009

ISSN: 1087-4208 print/1530-017X online
DOI: 10.1080/10874200802668416

Routledge
Taylor & Francis Group

CLINICAL CORNER

QEEG-Based Protocol Selection: A Study of Level of Agreement on Sites, Sequences, and Rationales Among a Group of Experienced QEEG-Based Neurofeedback Practitioners

Mark L. Johnson, MS
Eugenia Bodenhamer-Davis, PhD

ABSTRACT. *Background.* The history of neurofeedback is marked by a diversity of theoretical bases and specific protocol development approaches, including standard protocols based on research, symptom/neurophysiological function-based approaches, and approaches based on quantitative electroencephalography (QEEG) assessment (Budzynski, 1999; Demos, 2005). Although this diversity of approaches currently characterizes clinical practice within the field, one might assume that a certain degree of uniformity exists among practitioners who follow one particular treatment model. That is, clinicians who follow a symptom/function-based approach might be expected to select similar protocols for a given client, and practitioners who base their protocols largely on QEEG likewise would develop similar protocols for the same client.

Method. To test this latter assumption, 13 neurofeedback practitioners having 5 to 20 years of experience using QEEG and neurofeedback were sent the same QEEG data and presenting problems of a female adult who had previously sought neurofeedback treatment. The participant's data were edited in both NeuroReport and NeuroGuide, and both edits were provided to the survey participants. The practitioners were asked to provide treatment protocols covering sites, frequencies, sequences, and so on, as well as rationales that supported their protocol selections.

Results. Ten of the 13 professionals contacted responded to the survey. Respondents were in general agreement as to which sites and frequencies to treat. However, they diverged in their sequencing of treatment sites; in whether to inhibit, reinforce, or both; in cautioning about reference contamination in the QEEG record; and in their theoretical rationales for their protocol selections.

Mark L. Johnson and Eugenia Bodenhamer-Davis are affiliated with the University of North Texas, Denton.
Address correspondence to: Mark L. Johnson, MS, Neurotherapy Lab–Assistant Clinical Coordinator, University of North Texas, 1155 Union Circle #311277, Denton, TX 76203 (E-mail: mlj0098@unt.edu).

Special thanks to Lisa Black, Rob Coben, Richard Davis, Roger DeBeus, Jay Gunkelman, Joe Horvat, Joel Lubar, Leslie Sherlin, Alicia Townsend, and Jonathan Walker for their contributions to this project.

Conclusions. Although further research will have to document the efficacy of the various protocols recommended by the experienced QEEG-based practitioners surveyed for this study, it can be assumed that these practitioners are finding some consistent success using them in their practices. Therefore, the primary implication of this study appears to be that as long as appropriate treatment sites and frequencies are addressed for a given client, competently applied neurofeedback seems to be robust enough to tolerate a relatively wide diversity in specific protocol configurations.

KEYWORDS. Neurofeedback, QEEG, protocol selection

INTRODUCTION

The evolution of neurofeedback clinical practice has involved the emergence of several different theoretical and application orientations, from the use of standard research protocols for specific disorders, such as the Peniston Protocol for alcoholism and Post-Traumatic Stress Disorder (Peniston & Kulkosky, 1989, 1990, 1991) and the frontal alpha asymmetry protocol for depression (Baehr, Rosenfeld, & Baehr, 2001), to the symptom/neurophysiological function based approach credited to Sigfried and Sue Othmer (Othmer, Othmer & Kaiser, 1999). More recently, there has been increasing advocacy and use of quantitative electroencephalography (QEEG) to help determine neurofeedback protocols (Hammond, 2006; Kaiser, 2006). However, very little is known about general clinical practices among neurofeedback practitioners who use QEEG-based treatment protocols. We were curious to know how much uniformity exists in neurofeedback protocols that are derived in part from pre-treatment QEEG data. That is, given the same client information, presenting symptoms, and QEEG record, do most experienced neurofeeedback providers using QEEG-based protocols design a protocol containing common features in terms of frequencies, sites, sequence of addressing each site, inhibit/reinforce, and so on? To date, no studies have been published addressing this question or describing the general range of clinical practices in QEEG-based neurofeedback. Nor, for that matter, has any investigation explored the level of consistency among neurofeedback providers who use a symptom/function-based approach. To address the question of similarity in QEEG-based neurofeedback protocol selection, a small survey investigation was initiated to determine how much commonality actually exists among neurofeedback practitioners experienced in using QEEG to help guide their protocol development.

METHODS

Thirteen International Society for Neurofeedback and Research members known by the authors to be experienced clinicians who use QEEG-based neurofeedback were invited to participate in this descriptive research project. The sample was nonrandom but stratified to include and contrast QEEG-based neurofeedback practitioners with 5 to 10 years of experience with practitioners with 20 + years of experience. Ten individuals responded to the survey request. Five of the respondents had more than 20 years in QEEG and neurofeedback, and the other 5 respondents had between 5 and 10 years. All were mailed a compact disc containing the QEEG data, background information and presenting problems of an anonymous female client who had previously sought neurofeedback treatment. The QEEG data was edited in both Neuroreport (NREP) and Neuroguide (NG), and both edits were provided to the survey participants. Each respondent was asked to outline a treatment protocol based on the QEEG and brief client summary data provided and to indicate the sites, sequences, and rationale that supported his or her protocol recommendations. Respondents were encouraged to use the International 10/20

system (Jasper, 1958), which allows standardized placements of electrodes when recording EEG brain wave activity (Bocker, van Avermaete, & van den Berg Lennson, 1994). All respondents received the following summary of client background and symptom information and QEEG topographical data, edited in both NG and NREP.

Client Background and Symptom Data

> *The client whose QEEG record you have has already been treated in the University of North Texas Neurotherapy Lab. She is a 57-year-old Caucasian female. Her presenting symptoms were: stress issues, depression, lack of motivation, pain, sore muscles, and sleep problems. She was on the following medications at the time of the QEEG: Fosomax-Calcium (herbal supplement) 1 pill 1 time per week for osteoarthritis. The date of the QEEG was 12/5/05 (Monday). On 12/3/05, she had had a glass of wine.*

Client QEEG Topographical Data

See Figures 1 through 7 for the client QEEG topographic data.

RESULTS

All survey respondents agreed on the specific 10 to 20 sites to be treated. However, participants diverged rather significantly when it came to sequences, rationales, and inhibits versus reinforces in their protocol recommendations. There was as much diversity in recommendations within the more experienced subgroup of respondents (all of whom are recognized leaders/pioneers in the field) as there was in the subgroup of professionals with fewer years of experience with QEEG. (All protocols recommended along with rationales are presented in table form in the appendix.) The following is a summary of each survey respondent's individual protocol recommendations and comments regarding their selection rationales.

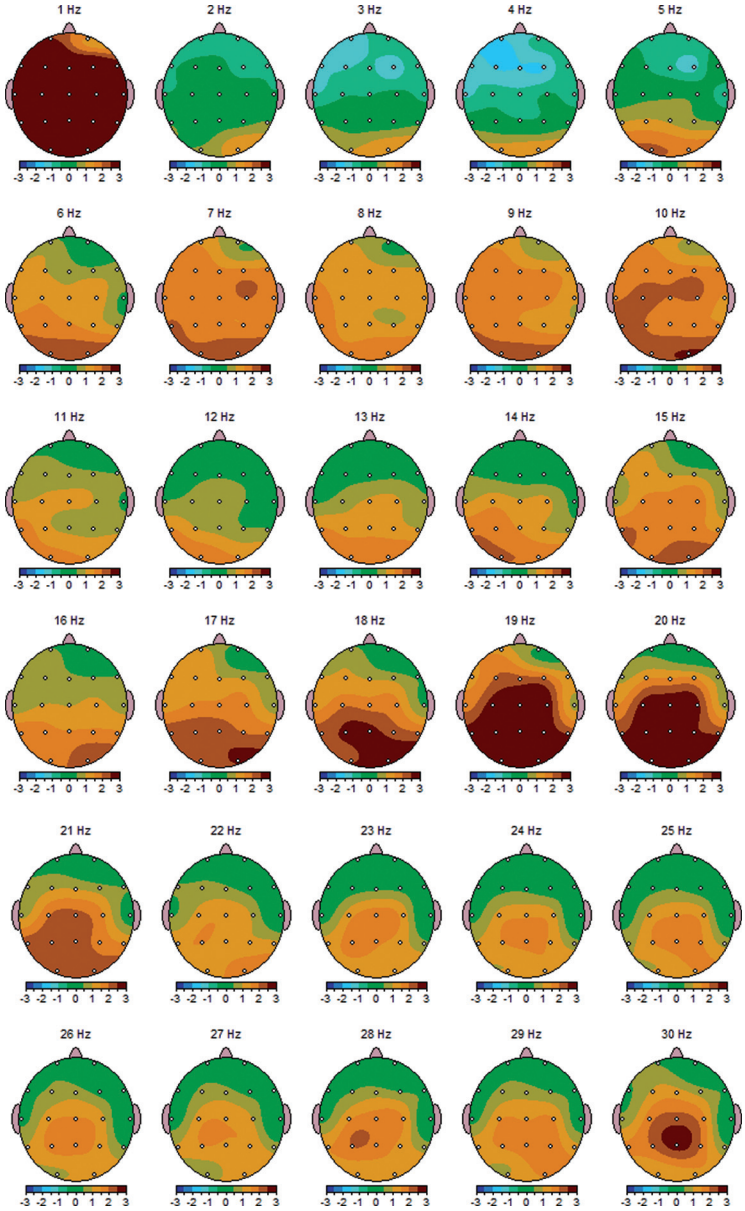
Respondents' Protocol Selections and Rationales

Respondent 1 (who has 5 years of experience) stated that she would start with FZ (a frontal vertex electrode placement site based on the international 10–20 system, Jasper, 1958) to "get frontal lobes working" first and to address possible sleep and lack of motivation concerns. She would then go to PZ (parietal vertex electrode placement site based on the international 10–20 system), which she perceived was the site of the most significant QEEG findings and likely related to stress, sleep, and possibly depression (and which may be secondary to anxiety, sleep, and stress). Respondent 1 offered the same rationale for left parietal protocols to address memory problems, auditory processing, or social difficulties that may underlie other concerns. Respondent 1 chose to address this site last because she thought it was least related to presenting problems and less significant than other findings in the QEEG topographic maps. Respondent 1 relied on the NREP maps.

Respondent 2 (who has 6 + years of experience) based his input largely on NREP Neuroelectrical Imaging (NEI) data showing a relative hypoconnectivity between F7 and P5 and some excessive power over the parietals in relative power maps (NREP and NG). Respondent 2 pointed out that the eyes-closed condition was tainted by a driven reference. This phenomenon may also be referred to as intrusion, alpha intrusion, or false frontal alpha. Several experts in the field (Rob Coben, Bill Hudspeth, and Jack Johnstone) agree with the following interpretation and nomenclature:

> It is not an artifact. It is a real signal. There is always activity at the reference electrode and the measurement is the difference between this and the active site. If the activity generated at or near the reference is greater than the activity at the "active" site, then it will show difference due to the activity at the reference. This most often occurs with alpha activity in the temporal cortex, but other frequencies, references etc. could cause similar distortions. (R. Coben, personal communication, October 16, 2008).

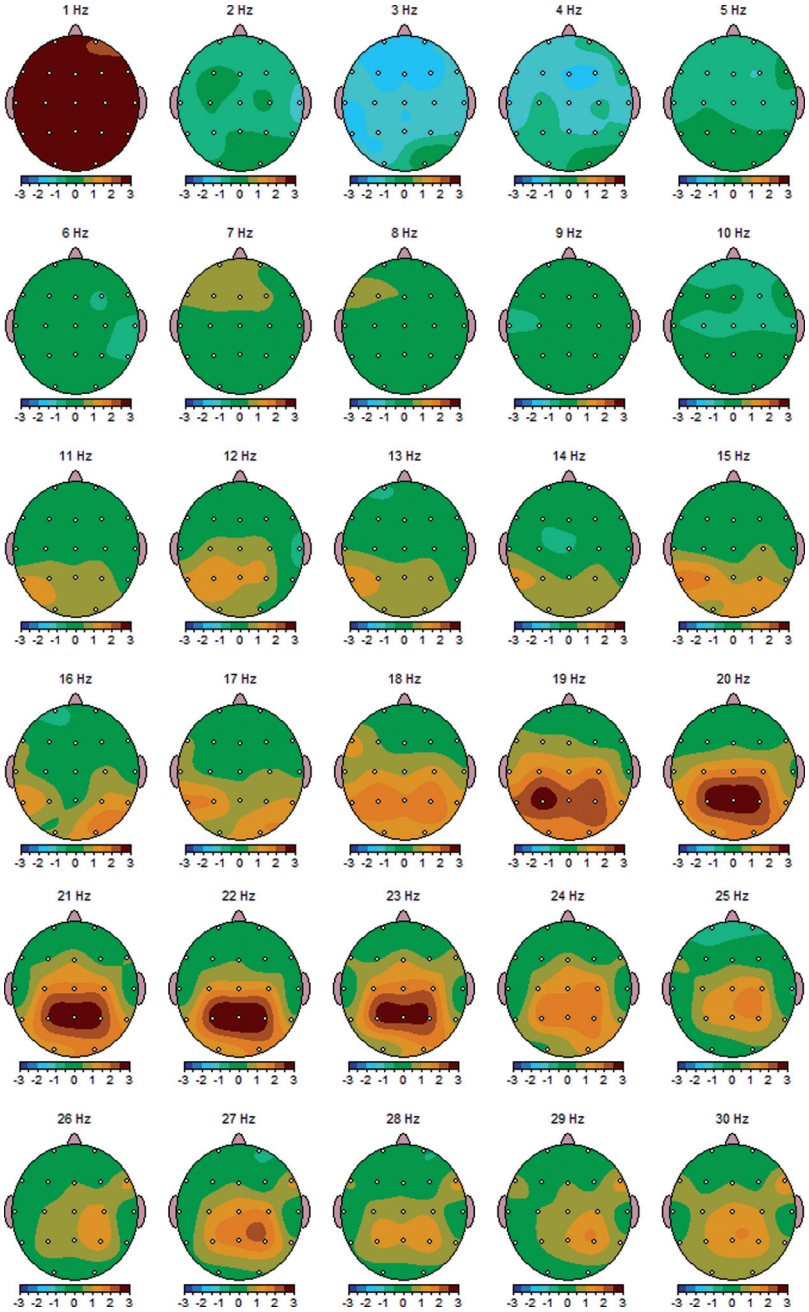
FIGURE 1. Neuroguide Z scored absolute power map eyes closed.



Respondent 2 noted that the driven reference collapsed the NEI frontally in theta and alpha and to some degree in beta. This artifact led to hypercoherence in the alpha band and threw off other readings as well. This respondent viewed the eyes-closed data (both trials) as uninterpretable and would want another recording done or remontaging at the least (but felt this would not eliminate the problem).

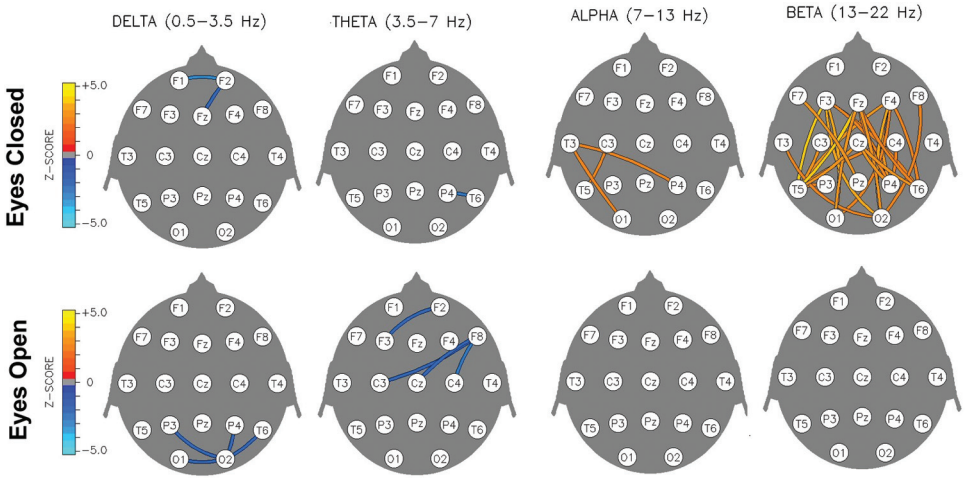
Respondent 3 (who has 20 + years of experience) recommended Sensory Motor Rhythm (SMR) training at C3 and C4 for mood stabilization, improved sleep, and perhaps to help with muscle pain. He then would move to PZ to decrease anxiety and increase relaxation, with both eyes-open and eyes-closed training. Respondent 3 suggested next working at FZ to increase alertness. This last protocol was designed to decrease depression, increase motivation, and reinforce executive functioning such as decision-making and planning strategies.

FIGURE 2. Neuroguide Z scored absolute power map eyes open.



Respondent 4 (who has 10 years of experience) would seek clinician/patient agreement as to which symptoms seemed most important to address first. He stated he would recommend starting with sleep because studies indicate that pain interferes with sleep and, in turn, sleep disturbances increase pain. He would then proceed frontally because that was where most of the imbalances were located. Respondent 4 would reward (reinforce) slow wave activity (1–7 Hz) as indicated by the maps, and he reasoned that rewarding slower wave activity should help to improve deeper sleep. Respondent 4

FIGURE 3. Neurorep coherence bandwidth map eyes closed and eyes open.

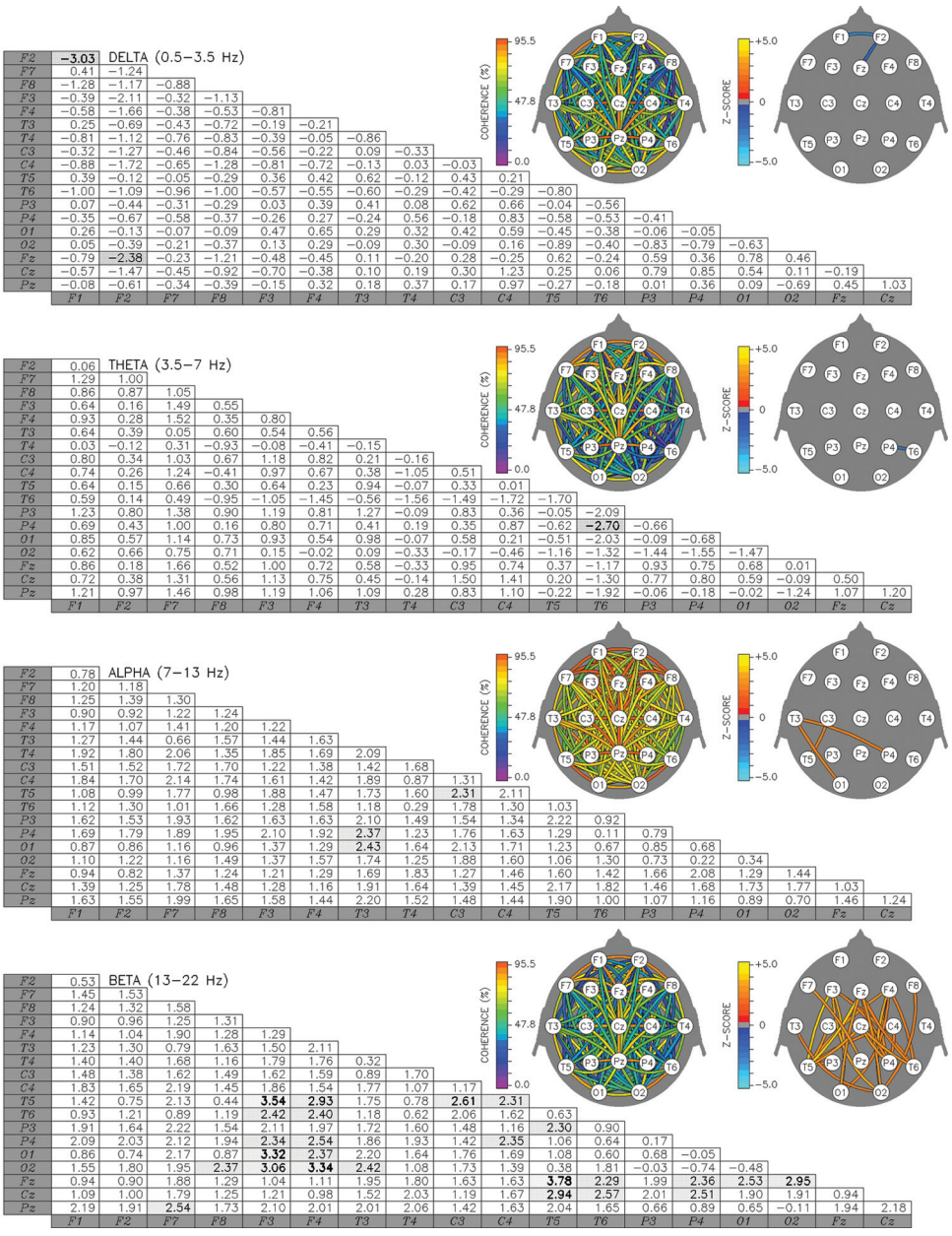


hypothesized that increasing most bandwidths frontally should help with improving pain symptoms. However, Respondent 4 narrowed his approach to coincide with QEEG findings. Respondent 4 continued to stay with the frontal lobes because it is implicated with depression. He also stated the maps showed a lack of relative power frontally in the beta bands. By activating the frontal areas, Respondent 4 hoped "to get this person going." If not, he would start looking at other options and considered this an interactive process, with all of these recommendations just seen as starting places. Respondent 4 went through the literature regarding osteoarthritis, pain, sleep, and EEG to determine what to look for with this patient's profile. He weighed the relative power results more heavily than the absolute power results. Finally, he combined the literature, symptoms, and QEEG results to come up with his protocols. If going through the above protocols produced no success, he would then train coherence based on NX-Link first, then NG and NREP.

Respondent 5 (who has 20 + years of experience) provided site-by-site rationales, which included the following: Decrease 2 to 7 Hz and increase 15 to 18 Hz at FPO2 to relieve depression. His rationale was that this is the best protocol his team has found for treating depression and is not based on the QEEG. The rest of his suggestions were all based on his Modular Activation Coherence model of brain function and neurofeedback training: decrease 1 Hz at F1 plus F2 to decrease ADHD symptoms, decrease 1 Hz at F3 plus F4 to improve motor planning on the right and motor planning on the left, decrease 1 Hz at T3 plus T4 to improve memory, decrease 1 Hz at C3 plus C4 to improve sensory motor integration on the right and handwriting and sensory motor integration on the left, decrease 1 Hz at T5 plus T6 to improve verbal understanding and emotional understanding, decrease 1 Hz at O1 plus O2 to improve visual processing, decrease 1 Hz at P3 plus P4 to improve cognitive processing of language and spatiotemporal information and math skills, decrease 21 to 30 Hz and increase 10 Hz at PZ plus P4 to decrease anxiety and irritability and to improve cognitive processing generally and cognitive processing of spatiotemporal information, and increase coherence of delta at P3/O2 to integrate cognitive processing of language and vision to the left. The reason for delaying the anxiety protocol until number 8 was that his experience has been that if the patient's cognitive difficulties are improved, treatment for anxiety will be more effective.

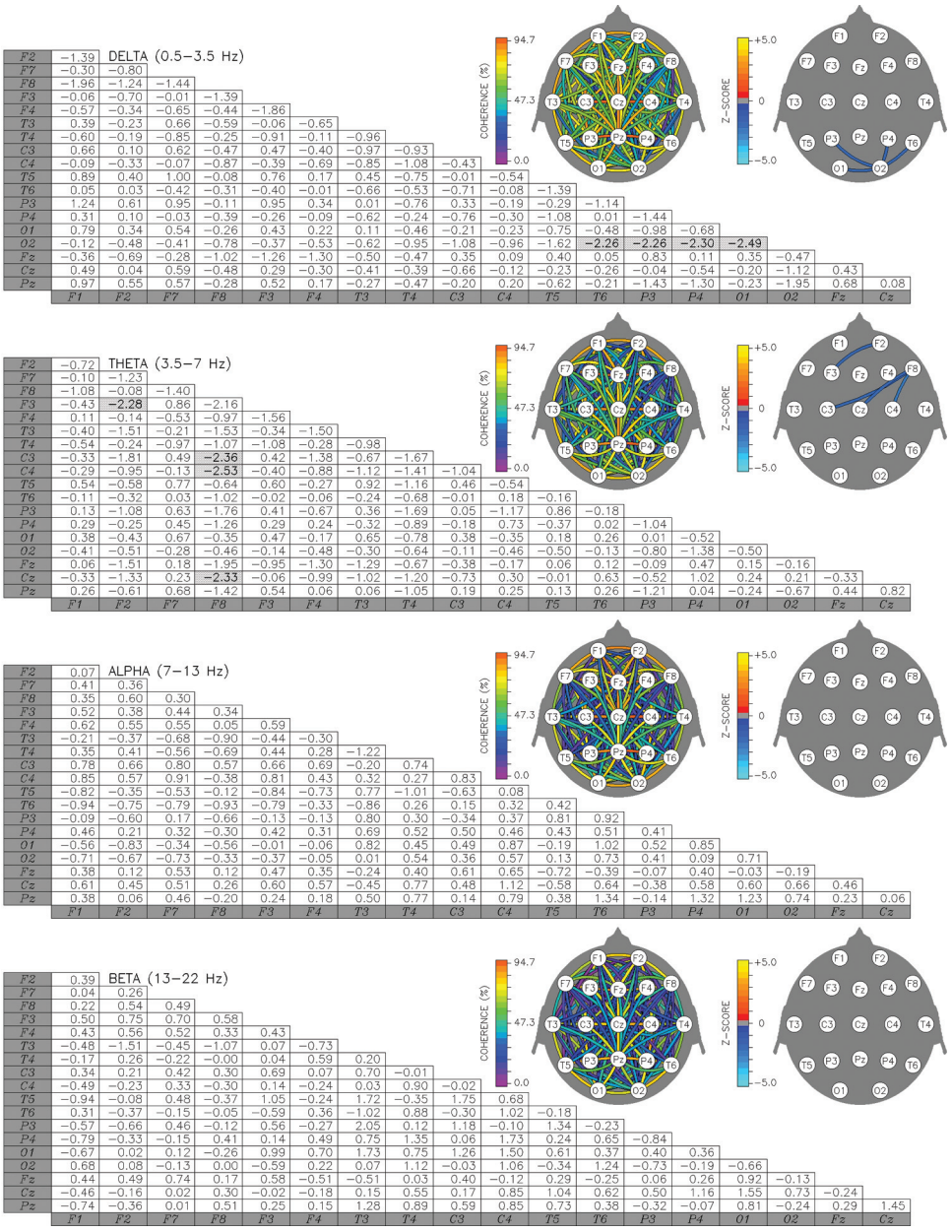
Respondent 6 (who has 8 years of experience) utilizes protocols that require

FIGURE 4. Neurorep coherence map eyes closed.



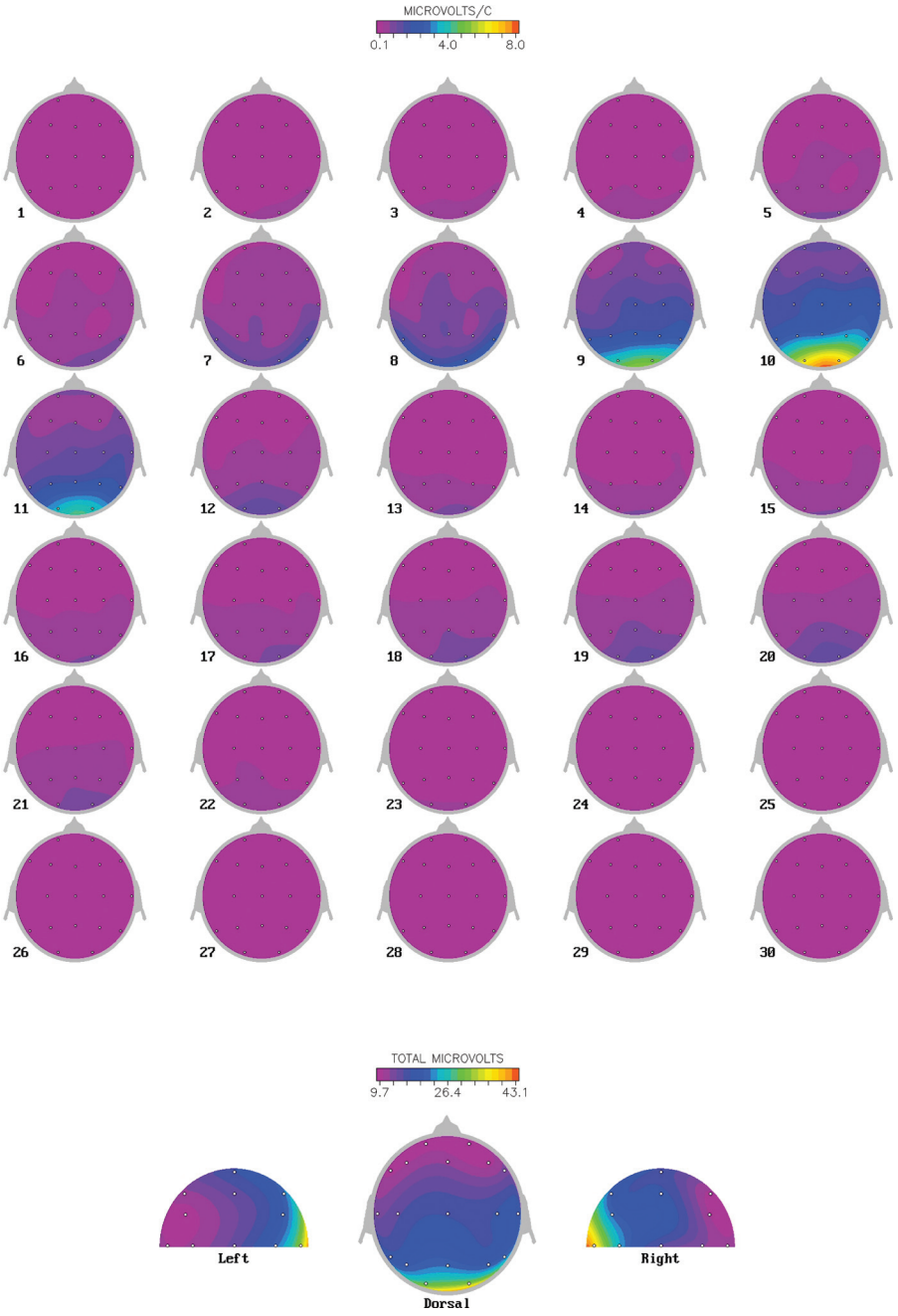
BrainMaster equipment with the "Sweet Spot" parameters (Black & Bodenhamer-Davis, 2003; McGee, 2002; or the old Neuro-Cybernetics equipment), because "each system is set-up with unique filtering and timing of feedback which optimizes treatment efficacy." The BrainMaster Sweet Spot manual describes the sweet spot method as an approach where subtle adjustments in training parameters can be set before treatment and finely adjusted during training sessions to fine-tune parameters while treatment is in progress. Respondent 6 relies heavily on raw wave data and observed the driven reference artifact of alpha activity in the QEEG.

FIGURE 5. Neurorep coherence map eyes open.



Respondent 6 would begin with eyes open training and see how her client could handle it. "The client's excesses are exacerbated in the eyes open condition, which may make it more difficult for her to begin there." But Respondent 6 stated she would be aware of this and move to eyes-closed training if she was not seeing the shifts she would expect to see. Respondent 6 stated her training would be done using bipolar hook-ups and using the BrainMaster with the Sweet Spot parameters. She considers this approach quite dynamic and treatment may include several placements and reward (reinforce) frequencies that change during the session. "The client is not left alone as the therapist is watching for changes and applying a type of desensitization during the therapeutic conversation (e.g., making sure that anxiety-producing issues are discussed in

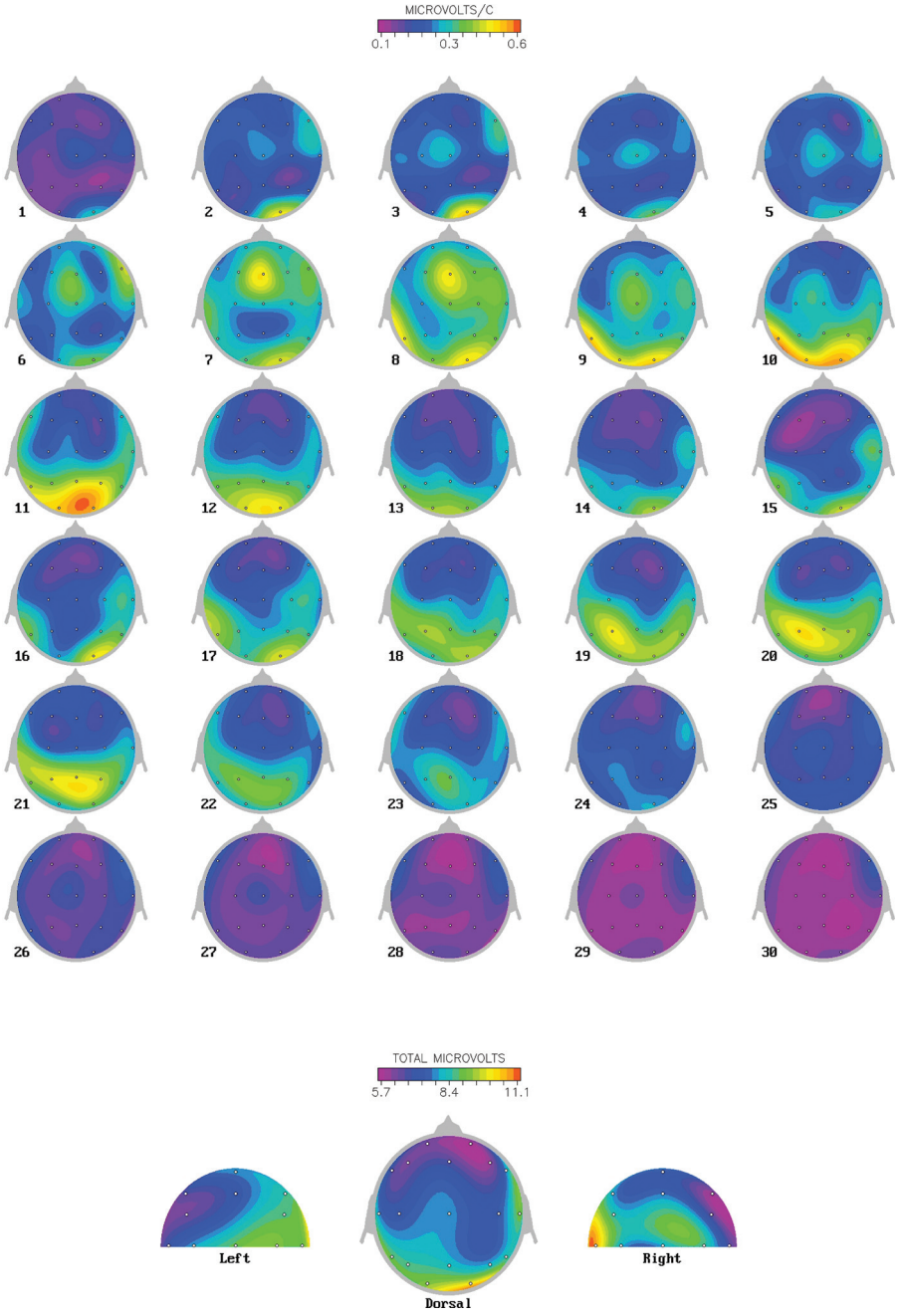
FIGURE 6. Neurorep weighted average map eyes closed.



conjunction with the feedback).'' Respondent 6 outlined the following protocols and rationales session by session:

First session. Respondent 6 would first work C5–C6 and C1–C2. She would experiment with 2–7 Hz or 2–13 Hz and 20–30 Hz or 14–30 Hz inhibits and experiment with the reward, which is likely in the 8–11 Hz range (map power, amplitude, and peak frequencies) but should be tweaked up or down to find what feels best for client and looks best in EEG (i.e., more normal). ''Sweet Spot reward range could conceivably end up

FIGURE 7. Neurorep weighted average map eyes open.



between 0–3 Hz and 15–18 Hz depending on connectivity, coherence, phase, asymmetry, and amplitude *relationships* that are not well documented in the maps. Inhibit lightly and reward generously for approximately 15 minutes at each site, but may be less—if 2–7/2–13 Hz rises sharply after 5 to 10 minutes, stop training at that site." Respondent 6 expected her client to feel better in the first session. She expected to see improved relaxation and calmness (based on subjective ratings) in conjunction with improved alertness (ratings here also) and a significant reduction in degree of pain.

"The EEG typically softens and the spectral appears even (e.g., no big peaks)." If her client experienced nothing, she would assume she was not at the right frequency or not at the right location. Thus, she would move according to the symptoms and the map. If that happened with this client at the central sulcus locations, she would pay attention to symptoms and move. (Based on the map only, she would choose a posterior placement such as P3–P4.)

Second session. If no sleep improvement was noted, Respondent 6 would stay with first-session protocol but either reward lower (e.g., sleeping too lightly) or add 3 to 9 min of left frontal (e.g., C5–AF3; e.g., waking and can't return to sleep because of busy mind) based on symptom changes. "The left frontal reward should be similar to the reward across the central sulcus, but may be up to 2 Hz higher."

Third session. If sleep improved, she would begin to target pain and utilize left frontal (e.g., C5–AF3) plus right posterior placement (e.g., C6–P4) based on symptoms and asymmetries seen in map. She would train the left frontal region for 5 to 10 min and the right posterior quadrant for 20 to 25 min utilizing Sweet Spot reward ranges and inhibit frequencies, inhibiting lightly and rewarding generously based on what looks most normalized in the brain and where the client felt best. If there was no sleep improvement, she would get more detailed assessment about what is disturbing sleep and train according to areas of the brain deemed to be helpful in remediating this concern.

Fourth session. If her client had an excellent response to prior training, she would stay close to prior sites and frequencies. If not, she would move to AF3–AF4 sites in combination with CP5–CP4 based on symptoms and power and amplitude maps. She would use the inhibit ranges that felt and looked best (e.g., more normal) during the first session and reward the frontal locations slightly lower (e.g., 1/2; to 2 Hz lower) than the Sweet Spot at the central sulcus, and at posterior locations, 1–4 Hz lower than Sweet Spot. She would expect to make adjustments during the sessions and would not expect the reward frequency to be the same during this session as it was in the prior sessions, though she would expect it to be close. ("It typically drops slightly.") She would train frontal sites for about 10 min and posterior sites for about 20 min but pay attention to overtraining effects and stop as soon as they are seen.

Fifth session. The site she would train would depend on goal attainment. She would expect sleep and pain to be improved significantly. If not, she would move to right hemisphere placements based on NREP NEI hypoconnectivity (e.g., C6–AF4 and C6–P4). She would train the frontal region for about 10 min and the posterior region for about 20 min. She would evaluate her client's ability to handle stress, mood, and motivation and begin to target these symptoms while maintaining improvements from prior sessions. For example, she might train using a protocol that was helpful for sleep at one session and then target new symptoms using a different protocol at the next session, and then return to the protocol that was helpful for pain reduction. Respondent 6 wouldn't want her client to lose any gains; she "would continue to monitor changes, understanding that resolution of symptoms correlates to brain changes that may affect overall brain balance. Hence, new symptoms could crop up that need to be targeted as areas of the brain learn new ways of functioning."

The first five sessions would be critical in the next protocols chosen. Assessment of both brain waves and symptoms would be continuously taking place so that decisions could be made and training tweaked for optimal goal attainment. She did not expect to be working with the same map at this point. "So, Sweet Spot reward frequencies may be very different than during the first session (typically lower)." Respondent 6 used both NG and NREP to determine her protocols.

Respondent 7 (who had 20 + years of experience) developed some of his own "triangulated" electrode site placements based on the 10–10 system but used 10–20 site references to decrease confusion. For example, LFT referred to "left frontal triangle,"

a point equidistant from F7, FP1, and F3. LPT referred to "left posterior triangle," a point equidistant from T5, P3, and 01. Respondent 7 believed that activating all three sites enables them to be treated simultaneously. Respondent 7 would reduce 5–15 Hz at O1 & O2 (2 channel), reduce alpha coherence at LFT – RPT, reduce 17–21 Hz at F3 & RPT (2 channel), reduce alpha coherence at RFT – LFT, reduce 13–21 Hz at F4 & LPT (2 channel), reduce alpha coherence at F3 – CZ/C4, reduce alpha coherence at T5 – CZ/C4. He would do the entire sequence (1 through 7) completely, repeat the sequence five times, and then remap before he would continue to work.

Respondent 7 said the aspects of training that were most important to him were to choose the highest *z* scores for potential treatment sites. Beyond that, he just wanted to alternate coherence and amplitude training and "move around the brain" in treatment rather than focus on a particular location. He believed that "this produces greater and faster overall changes on/in the brain, although slower change at a particular site." Respondent 7 said he and a longtime colleague debated whether to train power (amplitude) first or whether to train coherence first. He eventually came to believe that overall activation might be improved by moving around rather than focusing on a particular location for several consecutive sessions. So the three aspects of his choices were (a) highest *z* scores provide potential sites, (b) alternate amplitude and coherence/phase, and (c) provide a sequence that moves around the brain rather than focus on one location.

Respondent 8 (who has 8 years of experience) generally uses primarily slow and fast wave inhibit protocols, seldom reinforces. If QEEG and symptoms indicate, he likes to work frontal first, moving next to the motor strip/central areas, and then into the posterior areas of the brain. In this case, though, he felt it was necessary to try to help bring about calming by reducing the parietal high beta, and then he would move into his usual course of protocol sequencing. Respondent 8 uses a 20–30 Hz inhibit as a "safety valve" on all inhibit protocols and decides during the session how strong he needs the inhibit to be based on the amount of excess high beta present.

After reducing the parietal high beta, he would move to the frontal lobes to start getting the executive function as well as inhibitory function working in the frontal lobes (inhibit 2–8 Hz and 20–30 Hz at FZ). Respondent 8 expected after this frontal training to see sleep improve, stability start, focus improve, and OCD symptomology decrease. Moving next to CZ, he would expect more calming while continuing to improve the above symptomology in the frontal area. Next, he would move to F8 continuing to decrease excess high and low activity, expecting more calming, improved impulse control, and decreased irritability. Training down this same activity at O2 would help with possible visual processing problem. If client is overaroused after the frontal training, Respondent 8 would use an SMR Protocol (2–7 HZ inhibit, 12–15 HZ reinforce, and 20–30 HZ inhibit) at P4 to help calm the individual. If client is fine, he would move on to reducing alpha.

To train down excess alpha, Respondent 8 would start at CZ hoping to continue calming and reducing attention/focus issues if present, or at least increasing clarity. Moving next to the posterior temporals, he targeted social integration issues such as limits and boundaries issues and poor social perceptions. Moving next to the Occipitals, the intent was to reduce the eyes open alpha with what may be a visual processing problem. Respondent 8 bases his protocols on the NREP results.

Respondent 9 (20 + years of experience) stated that his goal was to train Fz referenced to the linked ears, training 6–9 Hz suppression with eyes open, with reinforcement of SMR at Cz-linked ears used for stabilization, to counterbalance any overactivation due to the Fz suppression training. He also indicated the presence of a focal slowed spectral alpha peak at Fz in the 7–8 Hz range, separate from the "healthy" faster 10 plus Hz posterior alpha rhythm that attenuates with eye opening. "The symptoms suggest the anterior location due to the general complaint of 'depression and stress,' and more

specifically they point to the locus of the anterior cingulate with 'motivational' and the 'perseveration' associated with chronic pain issues.''

Respondent 9 stated that removal of the slowed alpha at Fz through the NF training may ''overactivate'' the brain function, so the SMR training may be used in counterbalance with the Fz training as needed for stability.

The raw EEG provided all Respondent 9 needed, and he stated that the data he worked off of are easily seen in either of the commercial quantitative software packages mentioned (NG and NREP).

Respondent 10 (5–10 years of experience) stated that he would recommend sessions of two or three times per week evenly spaced as much as possible. He also usually attempts to have the session last 30 min. ''If the person fatigues after 25 minutes, I might push them to 27 minutes and end. Likewise if they are really in the 'zone' I might go until 40 or 45 minutes based on availability.'' He defined progress as client report of improved complaints *and* change in EEG. ''Change in EEG may show learning but if the client doesn't feel better it is of no value. Change in complaints without EEG learning usually lasts no longer than a few sessions.''

Respondent 10 always begins with an eyes open protocol to help the client identify and understand the process of neurofeedback. He starts with the first protocol mentioned (inhibit 6–9 Hz and augment 12–18 Hz at FZ referenced to linked ears) for several reasons. ''It should be one of the easiest protocols for the client to learn, therefore, providing her with some initial success with training and also it is likely that she is able to feel some improvement.'' He based this protocol on the QEEG results; thinks the client will respond quickly to it; and correlated this protocol to the complaints of lack of motivation, pain, and sleep problems. ''This protocol should be utilized at least 5 sessions and continued if the client reports progress. It should be discontinued when progress has plateaued or if there is no report of progress after 5 sessions.''

Respondent 10 stated his second protocol (inhibit 1–7 Hz and augment 9–11 Hz and inhibit 15–32 Hz at 0Z referenced to linked ears) is more challenging because of the increased number of requirements. He added, ''The eyes closed condition shows more aberration and stronger deviation than the eyes open condition.'' He thinks this protocol should address the complaints of pain, lack of motivation, sleep problems, and stress.

Respondent 10's third protocol (inhibit 1–7 Hz and augment 9–11 Hz and inhibit 15–32 Hz at 0Z referenced to AFZ) is identical to the second with the exception of the reference. This protocol is designed to shift the anterior to posterior asymmetry and the potential elevated hypercoherence, ''which appears mostly like a reference or noise problem.'' He stated this protocol should not be used more than three to five sessions without a new evaluation. He reasoned, ''I think the findings of hypercoherence are reference contamination, but the asymmetry may need to be addressed.''

He thinks his final protocol (inhibit 17–32 Hz at 0Z referenced to linked ears) is likely to be the most challenging to learn. ''It tends to be difficult for the client to learn how to 'try not to try' and to actually reduce parietal and occipital beta.'' ''This protocol would be aimed at complaints of stress, sleep and pain with possible implications in depression.'' Respondent 10 used both NREP and NG.

Comparison of Protocols/Rationales

The QEEG-based practitioners surveyed for this study closely agreed on certain aspects of their protocol recommendations and diverged significantly on other aspects. The following outcome information is summarized in Tables A1 to A5.

Site Commonalities and Differences

There was complete agreement among all respondents on treating the frontal lobe, though specific site recommendations varied, collectively including Fp1, Fp2, AF3 (between FP1 and F3), AF4 (between FP2 and F4), FZ, F3, F4, F7, and F8. Seven

out of 10 respondents would also treat sites on the sensory motor strip, with sites named varying among CZ, C1, C2, C3, C4, and C6. Seven out of 10 respondents targeted the parietal lobe and would treat PZ, P3 and P4. Five out of 10 respondents would treat the temporal lobe and would treat T3, T4, T5, and T6. Only 4 of the 10 respondents recommended treating the occipital lobes; 2 would treat at 01 and 02, 1 would treat at 0Z and 02, and 1 would treat at OZ and POZ. Comparison of subgroups within this survey sample (those having 5–10 years of experience and those with 20 + years of experience; see Table A2) showed that the two subgroups did not differ significantly in their site recommendations. Respondent comparisons of site commonalities and differences can be found in Tables A3 and A4.

Bandwidth Commonalities and Differences

The majority of respondents were in consistent agreement as to which brain wave frequency bandwidths to train, though sites varied among the brain regions in question. There were also slight discrepancies as to whether to inhibit or reinforce the agreed-upon bandwidth activity. Respondent comparisons of bandwidth commonalities and differences can be found in Table A5.

Sequence Commonalities and Differences

Half of the 10 survey respondents would begin by treating the frontal lobe (one also included parietal), whereas 4 of 10 respondents would start with the sensory motor strip. Two respondents would start with the parietal lobe (one also included frontal), and 1 respondent would begin in the occipital region. So there was a preference among the majority of these neurofeedback providers to begin treatment in the frontal lobes and/or sensory motor strip. The majority of the 20 + year subgroup (3 of 5) would begin treatment frontally. Two of the five 5- to 10-year experience subgroup would start on the sensory motor strip, whereas one of the five 20 + year subgroup started at this location. Other differences in sequence between these two subgroups were unremarkable (see Table A6).

Rationale Commonalities and Differences

There was almost total agreement (with one exception) that the elevated 1 Hz delta activity, seen in the QEEG should not be addressed in treatment because it (activity at 1 Hz that does not go beyond 1Hz) is suspected by many to be an artifact.

Three of the respondents (Respondent 10) from the 20 + year experience subgroup and 2 from the 5- to 10-year experience subgroup (Respondents 2 and 6) identified a reference-driven artifact. As Respondent 2 noted, "The eyes closed condition is tainted by a driven reference artifact that collapses the NEI frontally in theta and alpha and to some degree in beta. This artifact leads to hypercoherence in the alpha band and is throwing off other readings as well." Respondent comparisons of additional input in the QEEG record can be found in Table A7.

Training Commonalities and Differences

Four of the 10 respondents would do an "all inhibits" protocol with this case, whereas 5 of 10 respondents would use a mix of inhibits and reinforcers. Only one respondent would use all reinforces.

Several respondents shared unique input regarding their training methods. One respondent (Respondent 2) noted that he controls for artifact by suppressing 1–3 Hz. Another (Respondent 8) inhibits 20–30 Hz as a "safety valve" for all inhibit protocols. In addition, some of the respondents specified the number of sessions they would complete at each site with this case. This information generated additional questions from the surveyors.

Each respondent was asked several brief follow-up questions: (a) How many minutes do your clients generally train? (b) In general, what is the average number of sessions you do per site clients? (c) What determinant/method(s) do you use to know when to move to the next site (i.e., number of sessions, percentage of amplitude change,

report of symptom change, etc.)? (d) On average, what is the total number of sessions per client? and (e) How many sessions will be done on an unresponsive site? Respondent comparisons of training commonalities and differences can be found in Tables A8 and A9. The following is a summary of the average ranges reported by the survey respondents.

The range for usual training time per session was 15 to 40 min, with 20 to 30 min appearing to be the average. Fifteen-min training was stipulated for new clients in the early stages of neurofeedback treatment, whereas a client showing motivation to continue a successful session might be encouraged to continue to up to 40 min.

The range for the average number of sessions per site was as little as one session for respondents whose approach was either to move around the head or to use the Sweet Spot method (which also involves adjusting and moving frequencies "on the fly," and as high as 20 sessions for respondents who continue to see and hear reports of gains at a particular site).

Respondents were in relative agreement on three main factors used to determine when to move from one training site to the next. The determinants were (a) the client's learning curve and ability to demonstrate "EEG learning," (b) changes in the EEG (i.e., decrease in amplitudes and/or decreases in the variability of the waveform), and (c) symptom/behavior change or the lack thereof. Only two respondents replied to the question about how long to remain on an unresponsive site. Respondent 6 said 3 to 5 sessions, and Respondent 1 said 15 to 20 sessions. The average number of total treatment sessions per client ranged from 20 to 60, with considerations given for the client's learning curve as well as his level of complexity/severity of symptoms.

DISCUSSION AND CONCLUSIONS

The recommendations provided by this small sample of QEEG-based neurofeedback practitioners contained some areas of consensus but also considerable differences in how they would proceed with neurofeedback treatment, even though most provided clear theoretical or research rationales as bases for their recommendations. It was apparent that some respondents were clearly relying on clinical experience, published data, and theoretical conceptualizations in addition to the objective QEEG findings as they formulated treatment strategies, which may be speculated as reflecting common practice among QEEG-guided practitioners in the field. This study was not designed to determine the clinical validity of the various protocol approaches reported. Because it can be assumed that the practitioners responding to this survey have been doing neurofeedback with some success for 20 or more years using the QEEG-based methods they described in their responses, a tentative conclusion that could be derived from this study is that if appropriate treatment sites and frequency ranges are targeted, there is more than one way to do effective neurofeedback, even when using QEEG as a basis for protocol selection. These findings may provide impetus to the field for further research into the mechanisms that contribute to good clinical outcomes when varying neurofeedback approaches are used.

A limitation of this study is that it was administered to a relatively small, nonrandom sample of experienced neurofeedback providers known to use QEEG. No conclusions can be drawn about the actual effectiveness of any of the protocols described by respondents, as this was beyond the scope of the study and none of the protocols recommended was used in the survey participant's case.

A future study might seek to compare protocols used in very similar cases to evaluate the efficacy of different QEEG-based treatment approaches. In addition, a similar study might be administered to neurofeedback providers experienced in using symptom/neurophysiological function-based or other approaches that do not include QEEG in protocol selection decisions.

REFERENCES

Baehr, E., Rosenfeld, J. P., & Baehr, R. (2001). Clinical use of an alpha asymmetry neurofeedback protocol in the treatment of mood disorders: Follow-up study one to five years post therapy. *Journal of Neurofeedback*, *4*, 11–18.

Black, L. M., & Bodenhamer-Davis, E. (2003, October). Bi-polar client: Neurophysiological, clinical and treatment effects. Paper presented at the Biofeedback Society of Texas annual conference, Arlington, TX.

Bocker, K. B. E., van Avermaete, J. A. G., & van den Berg-Lennson, M. M. C. (1994). The International 10–20 System revisited: Cartesian and spherical coordinates. *Brain Topography*, *6*, 231–236.

Budzynski, T. H. (1999). From EEG to neurofeedback. In J. R. Evans (Ed.), *Introduction to quantitative EEG and neurofeedback* (pp. 66–76). San Diego, CA: Academic.

Demos, J. N. (2005). *Getting started in neurofeedback*. New York: W. W. Norton & Company, Inc.

Hammond, D. C. (2006). What is neurofeedback? *Journal of Neurotherapy*, *10*(4), 25–36.

Jasper, H. H. (1958). The 10–20 System of the International Federation. *Electroencephalography and Clinical Neurophysiology*, *10*, 370–375.

Kaiser, D. A. (2006). What is quantitative EEG? *Journal of Neurotherapy*, *10*(4), 37–52.

McGee, S. (2002, October). Neurofeedback for bi-polar disorder: A sweet spot. Paper presented at 8th Annual Clinical Interchange Conference, Los Angeles, CA.

Othmer, S., Othmer, S., & Kaiser, D. (1999). EEG biofeedback: An emerging model for its global efficacy. In J. R. Evans (Ed.), *Introduction to quantitative EEG and neurofeedback* (pp. 244–304). San Diego, CA: Academic.

Peniston, E., & Kulkowsky, P. (1989). Brainwave training and B-endorphin levels in alcoholics. *Alcoholism: Clinical and Experimental Research*, *13*, 271–279.

Peniston, E., & Kulkowsky, P. (1990). Alcoholic personality and alpha-theta brainwave training. *Medical Psychotherapy: An International Journal*, *3*, 37–55.

Peniston, E., & Kulkowsky, P. (1991). Alpha-theta brainwave neurofeedback therapy for Vietnam veterans with combat related post-traumatic stress disorder. *Medical Psychotherapy: An International Journal*, *4*, 47–60.

APPENDIX

TABLE A1. Protocol sites and sequences.

Seq	R1	R2	R3	R4	R5	R6	R7	R8	R9	R10
1st	FZ ↓ 6–9 Hz ↓ 20–30 Hz	F7 & P5 (P3/T5) ↑ 8–15 Hz ↓ 18–24 Hz	C3 & C4 SMR (12–15Hz)	C3 and/or C3-FZ seq ↑ 15–18 Hz C4 and/or C4-PZ seq ↑ 12–15 Hz	Fp02 ↓ 2–7 Hz ↑ 15–18 Hz	C5-C6 & C1-C2 Experiment ↓ 2–7 ↓ 2–13 ↓ 20–30 ↓ 14–30 ↑ 8–11 Sweet Spot Ranges: 0– 3 or 15–18	01 & 02(2) ↓ 5–15 Hz	PZ ↓ 18–25 If Needed: Same at P3 & P4	FZ ↓ 6–9 Hz CZ SMR	FZ (EO) ↓ 6–9 Hz ↑ 12–18 Hz
2nd	PZ ↓ 18–30 Hz	Re-map after 15–20	PZ ↓ 18–30 Hz ↑ 10–12 Hz	F3–F4 seq and/or FZ ↑ 1–7 Hz EC	F1 & F2 ↓ 1 Hz	Sleep impr. = Contin- + C5-AF3	↓ Co Alpha @ LFT RPT	FZ ↓ 2–8 ↓ 20–30		OZ (EC) Ref-ears ↓ 1–7 ↑ 9–11 ↓ 15–32
3rd	P3 ↓ 18–30 Hz		FZ ↓ 6–11 Hz ↑ 14–18 Hz	C3/C4 and/or CZ ↑ 1–7 Hz EC	F3 & F4 ↓ 1 Hz	C5-AF3 & C6-P4 use Sweet Spot Ranges	F3 & RPT ↓ 17–21(2)	CZ ↓ 2–8 ↓ 20–30		OZ (EC) Ref-AFZ ↓ 1–7 ↑ 9–11 ↓ 15–32
4th	T5 ↓ 7–10 Hz ↓ 17–30 Hz		F3 ↓ 6–11 Hz ↑ 14–18 Hz	Fp1–Fp2-s ↑ 12–15 Hz F3–F4 seq ↑ 12–18 Hz	T3 & T4 ↓ 1 Hz	AF3-AF4 & CP5-CP4 Adj. to SSRanges	↓ Co Alpha @ RFT-LFT	F8 ↓ 2–8 ↓ 20–30		POZ (EO) ↓ 17–32 Ref-ears

TABLE A1 (continued)

Seq	R1	R2	R3	R4	R5	R6	R7	R8	R9	R10
5th					C3 & C4 ↓ 1 Hz	C6-AF4 & C6-P4	F4 & LPT(2) ↓ 13–21	02 ↓ 2–8 ↓ 20–30		
6th					T5 & T6 ↓ 1 Hz		↓ Co Alpha @ F3-CZ/C4	If arousal: P4 ↓ 2–7 ↑ 12–15 ↓ 20–30		
7th					01 & 02 ↓ 1 Hz	↓ Co Alpha @	T5-CZ/C4	CZ ↓ 8–13 ↓ 20–30		
8th					P3 & P4 ↓ 1 Hz			T5 ↓ 8–13 ↓ 20–30		
9th					PZ & P4 ↓ 21–30 Hz ↑ 10 Hz			T6 ↓ 8–13 ↓ 20–30		
10th					P3/02 ↑ delta coherence			0Z ↓ 8–13 ↓ 20–30		

TABLE A2. Subgroup protocol sites and sequences.

	5–10 Years' Experience					20 + Years' Experience				
Seq	R1	R4	R6	R8	R10	R2	R3	R5	R7	R9
1st	FZ ↓6–9 Hz ↓20– 30 Hz	C3 and/or C3–FZ seq ↑15–18 Hz C4 and/or C4-PZ seq ↑12–15 Hz 0–3 or 15– 18	C5–C6 & C1–C2 Experiment ↓2–7 ↓2–13 ↓20–30 ↓14–30 ↑8–11 Sweet Spot Ranges:	PZ ↓18–25 if Needed: Same at P3 & P4	FZ (EO) ↓6–9 Hz ↑12– 18 Hz	F7 & P5 (P5=**P3**/**T5** ↑8–15 Hz ↓18–24 Hz	C3 & C4 SMR(12 15 Hz)	F_p02 ↓2–7 Hz ↑15–18 Hz	01 & 02(2) ↓5–15 Hz	FZ ↓6– 9 Hz CZ SMR
2nd	PZ ↓18 30 Hz	F3–F4 seq and/or FZ ↑1–7 Hz EC	Sleep impr. =Continue +C5-AF3	FZ ↓2–8 ↓20–30	OZ (EC) Refears ↓1–7 ↑9–11 ↓15–32	Re-map after 15–20	PZ ↓18–30 Hz ↑10–12 Hz	F1 & F2 ↓1 Hz	↑Co Alpha @LFT-RPT	
3rd	P3 ↓18 30 Hz	C3/C4 and/or CZ ↑1–7 Hz EC	C5-AF3 & C6-P4 use Sweet Spot Ranges	CZ ↓2–8 ↓20–30	OZ (EC) Ref-AFZ ↓1–7 ↑9–11 ↓15–32		FZ ↓6–11 Hz ↑14–18 Hz	F3 & F4 ↓1 Hz	F3 & RPT ↓17–21(2)	
4th	T5 ↓7–10 Hz ↓17–30 Hz	Fp1–Fp2-s ↑12–15 Hz F3–F4 seq ↑12–18 Hz	AF3–AF4 & CP5–CP4 Adj. to SSRanges	F8 ↓2–8 ↓20–30	POZ (EO) ↓17–32 Refears		F3 ↓6–11 Hz ↓14–18 Hz	T3 & T4 ↓1 Hz	↓Co ↓Co @RFT-LFT	

TABLE A2 (continued)

	5–10 Years' Experience					20 + Years' Experience				
Seq	R1	R4	R6	R8	R10	R2	R3	R5	R7	R9
5th			C6-AF4 & C6-P4	02 ↓2–8 ↓20–30				C3 & C4 ↓1 Hz	F4 & LPT(2) ↓13–21	
6th				If arousal: P4 ↓2–7 ↑12–15 ↓20–30				T5 & T6 ↓1 Hz	↓Co Alpha @F3-CZ/C4	
7th				CZ ↓8–13 ↓20–30				01 & 02 ↓1 Hz	↓Co Alpha @T5-CZ/C4	
8th				T5 ↓8–13 ↓20–30				P3 & P4 ↓1 Hz		
9th				T6 ↓8–13 ↓20–30				↓21–30 Hz ↓10 Hz PZ & P4		
10th				0Z ↓8–13 ↓12–30				P3/02 ↑ delta coherence		

TABLE A3. Respondent comparisons of site commonalities and differences.

Respondent	Frontal Lobe	Sensory Motor	Parietal Lobe	Temporal Lobe	Occipital Lobe
1	FZ		PZ P3	T5	
2	F7		P3	T5	
3	FZ F3	C3/C4	PZ		
4	F3–F4-s and/or FZ (EC) Fp1–Fp2-s F3–F4-s	C3 and/or C3/FZ C4 and/or C4/PZ C3/C4 and/or CZ (EC)	(PZ)		
5	Fp02 F1 & F2 F3 & F4	C3 & C4	P3 & P4 PZ & P4 P3 (& 02)	T3 & T4 T5 & T6	01 & 02
6	AF3–AF4 (& CP5–CP4)	C5–C6 & C1–C2 C5-AF3 (and C6-P4) C6-AF4 (and C6-P4)	P4		
7	F3 & RPT F4	(F3)-CZ/C4 (T5)-CZ/C4		T5	01 & 02
8	FZ, F8	CZ	PZ P3 & P4	T5, T6	02, 0Z
9	FZ	CZ			
10	FZ				OZ, POZ
Level of agreement	10/10	7/10	7/10	5/10	4/10

TABLE A4. Subgroup comparisons of site commonalities and differences.

Respondent	Frontal Lobe	Sensory Motor	Parietal Lobe	Temporal Lobe	Occipital Lobe
1	FZ		PZ P3	T5	
4	F3–F4-s and/or FZ (EC) Fp1–Fp2-s F3–F4-s	C3 and/or C3/FZ C4 and/or C4/PZ C3/C4 and/or CZ (EC)	(PZ)		
6	AF3–AF4 (& CP5-CP4)	C5–C6 & C1–C2 C5-AF3 (and C6-P4) C6-AF4 (and C6-P4)	P4		
8	FZ, F8	CZ	PZ P3 & P4	T5, T6	02, 0Z
10	FZ				OZ, PZ
Subgroup (5–10 years exp.) level of agreement	5/5	3/5	4/5	2/5	2/5
2	F7		P3	T5	
3	FZ F3	C3/C4	PZ		
5	Fp02 F1 & F2 F3 & F4	C3 & C4	P3 & P4 PZ & P4 P3 (& 02)	T3 & T4 T5 & T6	01 & 02
7	F3 & RPT F4	(F3)-CZ/C4 (T5)-CZ/C4		T5	01 & 02
9	FZ	CZ			
Subgroup (20+ years exp.) level of agreement	5/5	4/5	3/5	3/5	2/5
Combined level of agreement	10/10	7/10	7/10	5/10	4/10

TABLE A5. Bandwidth Agreement.

Respondent	Frontal	Central	Parietal	Temporal	Occipital
1	6–9↓		PZ & P3 18–30↓	T5: 7–10↓ 17–30↓	
2	F7: 8–15↑ 18–24↓		P5/P3: 8–15↑ 18–24↓	T3: 8–15↑ 18–24↓	
3	6–11↓ 14–18↑	C3 C4-12–15	PZ: 18–30↓ 10–12↑		
4	F3-F4 or FZ 1–7↑	C3 & C4 12–15 or 15–18			
5	F1 F2 F3 F4 1↓ Fp02: 2–7↓ 15–18↑	C3 C4: 1↓	P3 P4: 1↓ PZ P4: 21–30↓ 10↑	T3 T4 T5 T6: 1↓	O1 O2: 1↓ P3-02 Delta- coherence
6	n/a	n/a	n/a	n/a	n/a
7	F7 Fp1 F3 Alpha Co↓	CZ/C4 Alpha Co↓ −F3 & −T5	P4 (RPT) 17–21↓	T6 (RPT) 17–21↓	01 02 5–15↓
8	2–8↓	2–8↓ 8–13↓	18–25↓	T5 T6 8–13↓	02: 2–8↓ 0Z: 8–13↓
9	6-9↓	SMR			
10	6–9↓ 12–18↑				0Z: 1-7↓EC 9–11↑EC 15–32↓EC 17–32↓ (EO)
Level of bandwidth agreement	7/9 Delta and theta Ranges	5/6 Alpha and Lo Beta Ranges	5/6 High Beta Range	3/4 Alpha range	3/4 Delta-theta range

TABLE A6. Respondent and subgroup comparisons of sequence commonalities and differences.

Respondent	Start w/Frontal Lobe	Start w/Sensory Motor	Start w/Parietal Lobe	Start w/Temporal Lobe	Start w/ Occipital Lobe
1	X				
2	X		X		
3		X			
4		X			
5	X				
6		X			
7					X
8			X		
9	X	X			
10	X				
Level of agreement	5/10	4/10	2/10	0/10	1/10
Subgroup					
1	X				
4		X			
6		X			
8			X		
10	X				
Subgroup (5–10 years exp.) level of agreement	2/5	2/5	1/5	0/5	0/5
2	X		X		
3		X			
5	X				
7					X
9	X				
Subgroup (20+ years exp.) level of agreement	3/5	1/5	1/5	0/5	1/5
Combined level of agreement	5/10	3/10	2/10	0/10	1/10

TABLE A7. Respondent comparisons of additional input.

Respondent	Identified Elevated 1 Hz Activity as Artifact	Identified Driven Reference Artifact	Control for Artifact by Suppressing 1–3 Hz	Inhibit 20–20 Hz as a "Safety Valve" for all Inhibit Protocols	Up Train Slow Waves	1 Session Per Site to Move Around the Brain
1	X					
2	X	X	X			
3	X					
4	X				X	
5						
6	X	X				
7	X					X
8	X			X		
9	X					
10	X	X				
Level of agreement	9/10	3/10	1/10	1/10	1/10	1/10

TABLE A8. Respondent comparisons of training practices.

Respondent	All Inhibits	All Enhances	Mixed
1	X		
2			X
3			X
4		X	
5			X
6			X
7	X		
8	X		
9	X		
10			X
Agreement	4/10	1/10	5/10

TABLE A9. Respondent comparisons of training practices.

Respondent	Training Time Per Session	Average Number of Sessions per Site	Determinant to Move to Next Site	Average No. of Total Sessions	Max No. of Sessions on Unresponsive Site
1	15–20 min Increase to 30–40	Coherence 3–7 Amplitude 10–30	Shift in activity or behavior they can maintain, or no change	Minimum of 30, up to 100+ for more severe. Average = 50–60	15–20
2	15–20 min	20	Follow-up evaluation including QEEG		
3					
4	30 min (may start w/20)	10 as an estimate	EEG learning; Symptom improvement; EEG changes		
5					
6	15–45 min	1–20	Behavioral goals EEG changes	20–25 = average person 4–60 (complexity)	3–5
7					
8	30 min	Site 1: 12–15 Site 2: 6–8 Site 3: 4–6 Continue: 4–6	Site EEG changes (reduction of variability and decrease of amplitude, and symptom changes	40	
9	30–45 (1 per day) 3–4 per week; reduce to 1–2 per week; then fewer	Depends on client's learning curve	Depends on client's learning curve	15–20 = Clinical impact; 20–30 = stable results for attention deficit hyperactivity disorder	
10	30 min 25–40 (pending tired or alert)	5–15	Complaint reduction & EEG learning	Depends on Client	
Agreement range	15–40	1–20	Behavior change/not EEG change Learning curve	20–60	3–20

Note. QEEG = quantitative electroencephalography.