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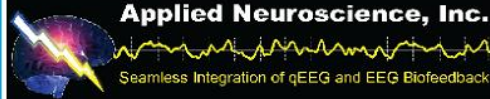
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ADVERSE REACTIONS AND POTENTIAL IATROGENIC EFFECTS IN NEUROFEEDBACK TRAINING

In the early days of the field of psychotherapy, it was naively assumed that patients either improved or remained unchanged. Operating on this assumption, research prior to the 1960's commonly only included change and no change groups. With time, however, more than fifty studies (Bergin & Lambert, 1978; Lambert, & Bergin, 1994) have now documented that an average of about 10% of patients entering psychotherapy and couples therapy show deterioration effects. This body of research has identified certain characteristics of patients (e.g., being more severely disturbed or schizophrenic; having unrealistic expectations) that seem to make them more vulnerable to having a negative outcome from therapy. The research has also pinpointed certain therapist styles (e.g., lack of individualization of treatment by authoritarian therapists wherein everyone receives the same treatment; lack of empathy; harsh and excessive confrontation from a therapist or members of a therapy group) that seem to be associated with increased risk of iatrogenic effects.

In the field of neurofeedback, which is still somewhat young in its development, some clinicians say that they have never heard of an adverse reaction. However, an early study (Lubar & Shouse, 1976) with an A-B-A design found that when theta (4-7 Hz) was being inhibited and SMR reinforced, improvements were seen in hyperactivity. But, when the contingencies were reversed and theta was reinforced, there was a reversal of the positive changes. Similarly, in a double-blind, A-B-A crossover study of medically refractory epileptic patients (Whitsett, Lubar, Holder, Pamplin, & Shabsin, 1982) when sleep EEG's were evaluated it was found that during initial training to reduce theta activity and enhance SMR, there was an 18% decrease in paroxysmal activity from a baseline of 72%. But, following a reversal of reinforcement contingencies there was an increase of 29%, and after reinstating therapeutic contingencies there was a drop of over 60%. The authors concluded that the reversal phase "appears to have been detrimental to the patients" (p. 203). Thus, we do have a limited amount of controlled research documenting the potential for negative effects if inappropriate training is done.

I have personally observed transient negative effects. One of the more common ones has been individuals feeling somewhat anxious and experiencing difficulty sleeping following training to increase beta activity. In such cases, I have learned that reducing the frequency band of up-training, simply focusing on inhibiting slow activity rather than simultaneously increasing beta as well, or doing one to two sessions of SMR training at C3 and C4 has quickly alleviated the short-lived symptoms. These experiences have led me to believe that it is quite important to obtain subjective feedback from patients, as well as from parents or spouses, about what they are experiencing as training progresses.

I have felt concern about the use of alpha-theta training without individualized assessment. It is true that the average quantitative EEG (QEEG) profile for alcoholics (John, Pritchep, Fridman, & Easton, 1988) shows an excess of beta, and deficiencies, particularly frontally and centrally, in the theta and alpha frequency bands. However, there is also research (Schubiner, Tzelepis, Milberger, Lockhart, Kelley, & Schoener, 2000) which finds that there is a significant subgroup (24%) of alcoholics who qualify for a diagnosis of ADHD. If posterior alpha-theta training were done with an alcoholic who also had problems with ADHD and who did not have an excess of beta activity, might this exacerbate the ADHD? Alcoholics also frequently suffer with comorbid depression, which many studies (Alper, 1995; Brenner et al., 1986; Itil, 1983; John et al., 1988; Knott & Lapierre, 1987; Monakhov & Perris,

1980; Nystrom, Matousek, & Hallstrom, 1986; Pollock & Schneider, 1990; Nieber & Schlegel, 1992) have found may be associated with an alpha or theta excess. Is it possible that training to enhance alpha or theta might increase depression in such a person? This should be even more a cause for added concern because recent research has documented that post-sobriety depression significantly increases the risk of relapse in alcoholics (Curran, Flynn, Kirchner, & Booth, 2000). We don't know the answers to these questions yet, but I think it is wise for thoughtful clinicians to consider such issues and for us to ask ourselves tough questions. Given such possibilities, in an alcoholic with excess beta (which especially in the right frontal area may be associated with insomnia, anxiety, anger, and depression), might it be wise to consider inhibiting this excessive activity, or to inhibit it while also doing a limited amount of alpha-theta training if there are no comorbidities or EEG activity that would contraindicate such training?

In examining the raw EEG data and also the quantitative EEG data on my patients, I have seen many cases where "standardized" or canned protocols could well have produced iatrogenic effects. Recently a man in his early twenties came to my office who had previously been diagnosed with ADD, had taken Ritalin for some years, and who fully met all the DSM-IV diagnostic criteria for diagnoses of both ADD and ADHD. A popular, standardized protocol with research (Lubar, 1995) supporting its overall or frequent effectiveness would be to use a bipolar electrode placement at Fz and Cz, increasing 16-22 Hz while inhibiting 6-10 Hz. However, the NYU, Nx Link database (John et al., 1988) revealed not only excesses within the alpha frequency band, but also some beta excess in the range of 1.22-1.30 Z-scores at Fz, Cz, and C4. This led me to inquire about symptoms of obsessive-compulsive disorder, which revealed that the patient should receive a dual diagnosis. He strongly met the criteria for OCD both in the interview and with objective evaluation. I anticipate that if I had reinforced beta activity over the area of the cingulate or in the right hemisphere, this patient may well have experienced an exacerbation of OCD and anxiety symptoms. Instead, when beta in the range over 20+ Hz was inhibited, OCD symptoms rapidly declined, and soon our neurofeedback work shifted to also inhibiting his excess alpha.

Among clinicians who use QEEGs to evaluate patients, if the therapist does not use methods to insure vigilance during data gathering and has not been carefully trained in artifacting, the subsequent topographic maps on which they base their treatment plan may be seriously flawed, resulting in very misguided protocols. For example, if we are not very

attuned to signs of drowsiness (Santamaria & Chiappa, 1987) the improperly “artifactual” data may show temporal, central, and/or frontal alpha or theta that is not really present—except in drowsiness. The clinician may also mistake frontocentral beta in the range of 25-30 Hz as a beta excess or evidence of possible over activity of the cingulate associated with OCD, when in fact Santamaria and Chiappa (1987) discovered that one-third of subjects experience this in association with drowsiness. If thus mistaken, a clinician could conceivably reduce frontocentral beta, resulting in iatrogenic cognitive inefficiency. Thus, I echo calls in the contributions below by Margaret Ayers and John Nash for carefully examining raw data.

In evaluating patients with excess alpha or theta activity, I encounter a large number of cases where beta activity is not deficient, but normal, and a moderate number of cases where beta activity is excessive. This has led me to seriously question the advisability of reinforcing an increase in beta activity in such cases—something that Margaret Ayers discusses below. Just as I believe in the need to individually tailor psychotherapy following careful assessment, so I have become a believer in the need to individualize neurofeedback to the unique brain wave characteristics of the patient. I do not believe that “one size fits all,” and as the psychotherapy literature on iatrogenic effects informs us, we should be very cautious when someone claims to have found a single approach, or “the one true light.”

At this point in the evolution of neurotherapy there are no research-based reports apart from what I have cited documenting transient or more significant negative effects. Therefore, I encourage clinicians to report on individual cases where there have been adverse effects. I likewise call upon researchers to include measures to evaluate and monitor potential transient adverse reactions, and to also not only examine whether or not patients change, but whether there are any deterioration effects.

In the absence of research on this important topic, and in an effort to sensitize ourselves to it, I have asked some very seasoned practitioners to discuss their clinical experience and to share their opinions on this topic.

Steve Stockdale, PhD
Daniel Hoffman, MD
Margaret E. Ayers, MA
John Nash, PhD

REFERENCES

- Alper, K. (1995). Quantitative EEG and evoked potentials in adult psychiatry. Chapter in J. Pankseep (Ed.), *Advances in biological psychiatry, Vol. 1* (pp. 65-112). Greenwich, CT: JAI Press.
- Bergin, A. E., & Lambert, M. J. (1978). The evaluation of therapeutic outcomes. Chapter in S. L. Garfield & A. E. Bergin (Eds.), *Handbook of psychotherapy & behavior change* (2nd Edition) (pp. 139-189). New York: Wiley.
- Brenner, R. P., Ulrich, R. F., Spiker, D. G., Sclabassi, R. J., Reynolds, C. F., Marin, R. S., & Boller, F. (1986). Computerized EEG spectral analysis in elderly normal demented and depressed subjects. *Electroencephalography & Clinical Neurophysiology*, *64*, 483-492.
- Curran, G. M., Flynn, H. A., Kirchner, J., & Booth, B. M. (2000). Depression after alcohol treatment as a risk factor for relapse among male veterans. *Journal of Substance Abuse Treatment*, *19*(3), 259-265.
- Itil, T. M. (1983). The discovery of antidepressant drugs by computer-analyzed human cerebral bioelectrical potentials (CEEG). *Progress in Neurobiology*, *20*, 185-249.
- John, E. R., Pritchep, L., Fridman, J., & Easton, P. (1988). Neurometrics: Computer assisted differential diagnosis of brain dysfunctions. *Science*, *293*, 162-169.
- Knott, V. J., & Lapierre, Y. D. (1987). Computerized EEG correlates of depression and antidepressant treatment. *Progress in Neuropsychopharmacology in Biological Psychiatry*, *11*, 213-221.
- Lambert, M. J., & Bergin, A. E. (1994). The effectiveness of psychotherapy. Chapter in A. E. Bergin & S. L. Garfield (Eds.), *Handbook of psychotherapy & behavior change* (4th Edition) (pp. 143-189). New York: Wiley.
- Lubar, J. F. (1995). Neurofeedback for the management of attention-deficit/hyperactivity disorder. Chapter in M. S. Schwartz (Ed.), *Biofeedback: A practitioner's guide* (pp. 493-522). New York: Guilford Press.
- Lubar, J. F., & Shouse, M. N. (1976). EEG and behavioral changes in a hyperkinetic child concurrent with training of the sensorimotor rhythm (SMR): A preliminary report. *Biofeedback & Self-Regulation*, *1*(3), 293-306.
- Monakhov, K., & Perris, C. (1980). Neurophysiological correlates of depressive symptomatology. *Neuropsychobiology*, *6*, 268-279.
- Nieber, D., & Schlegel, S. (1992). Relationships between psychomotor retardation and EEG power spectrum in major depression. *Biological Psychiatry*, *25*, 20-23.
- Nystrom, C., Matousek, M., & Hallstrom, T. (1986). Relationships between EEG and clinical characteristics in major depressive disorder. *Acta Psychiatrica Scandinavica*, *73*, 390-394.
- Pollock, V. E., & Schneider, L. S. (1990). Quantitative, waking EEG research on depression. *Biological Psychiatry*, *27*, 757-780.
- Santamaria, J., & Chiappa, K. H. (1987). *The EEG of drowsiness*. New York: Demos Publications.
- Schubiner, H., Tzelepis, A., Milberger, S., Lockhart, N., Kruger, M., Kelley, B. J., & Schoener, E. P. (2000). Prevalence of attention-deficit/hyperactivity disorder and conduct disorder among substance abusers. *Journal of Clinical Psychiatry*, *61*(4), 244-251.

Whitsett, S.F., Lubar, J.F., Holder, G.S., Pamplin, W.E., & Shabsin, H.S. (1982). A double-blind investigation of the relationship between seizure activity and the sleep EEG following EEG biofeedback training. *Biofeedback & Self Regulation*, 7, 193-209.

QUESTION: “Have you seen any negative effects associated with EEG neurofeedback?”

RESPONSE: Steven Stockdale, PhD, The Neuro-Health Center, 2132 North Nevada Ave., Colorado Springs, CO 80907 (E-mail: stockdale@pcisys.net), and Daniel A. Hoffman, MD, The Neuro-Therapy Clinic, P.C., 8200 E. Belleview, Suite 600-E, Englewood, CO 80111 (E-mail: dhoffman48@aol.com).

Having done neurofeedback for about 12 years, both in Denver and Colorado Springs, we have identified certain adverse reactions from EEG neurofeedback. However, we have also found them to be both rare and transient. They do not seem to be permanent when corrected with EEG neurofeedback treatment. Some of the problems we have seen are:

1. Theoretically, there is always concern of potentially inducing seizures from neurofeedback training. We have not experienced this personally, but it has been raised as a concern by some neuroscientists.
2. At times we have found that alpha-theta training can make people too “spacey.” Usually reading a magazine for a few minutes corrects this (i.e., Beta).
3. With one patient we found that training to increase alpha increased his depression.
4. On several occasions, we have seen training to increase SMR cause agitation in the patient, instead of settling them down.
5. When treating rapid cycling bipolar disorders, we have seen that it is very easy to over-train a patient, either increasing their manic/agitation symptoms, or increasing their depression. One needs to go very slowly, checking in with the patient every 5-10 minutes and reverse treatment if you over-shoot.
6. Sometimes, training to increase theta can elicit unwanted traumatic memories.

In summary, these are some of the adverse reactions we have seen from neurofeedback training. However, again, these reactions have been rare and not permanent when addressed and treated with EEG neurofeedback.

RESPONSE: Margaret E. Ayers, MA, Neuropathways EEG Imaging, Inc., Beverly Hills, CA 90210 (E-mail: neuropat@ix.netcom.com).

Iatrogenic effects of EEG feedback are a subject that must be discussed in order to strengthen the field. EEG feedback based on protocols rather than neurobiological principles can result in harm. Humans are not Betty Crocker recipe protocols.

Each month I get individuals from other EEG feedback clinics doing protocols adverse to clients. For example, the first common problem that I see is that anyone labeled ADD or ADHD has usually only been diagnosed using a brain map or QEEG which is secondary or reconstructed data and never shows the primary raw EEG data. Many medical problems have the same symptomatology as ADD. ADD is diagnosed by behavioral indices such as the Test of Variables of Attention, or parent and classroom reports of behavior. If one does not have the raw, primary EEG data, it is not possible to see the ADD pattern in the EEG and to determine for sure that this pattern exists. Head trauma, anoxia, hypoxia, birth difficulties, depression, or absence seizures all have behavioral symptoms of ADD. On one occasion, I sent individuals with three different diagnoses to a psychologist friend to diagnose. One had ADD, one had head trauma, and the third had genetic unipolar depression. They all came back with a diagnosis of ADD.

When labeled ADD without the clinician having raw EEG data, a common protocol is to train the brain at the CZ electrode site. If the client actually has head trauma, cerebral palsy, absence seizures, or hypoxia, he or she will get worse when training either 12-15 Hz or beta at this site. The symptoms become worse because of a neurological principle that any training over the corpus collosum acts as a reverberator or kindles a seizure response in someone with epilepsy. This is the reason that the corpus collosum is severed clinically in some severe forms of epilepsy to prevent kindling of seizure activity.

When QEEG brain maps are used to diagnose ADD, subtypes of ADD are mentioned. When one can see the primary EEG data, it is clear that rather than ADD subtypes, other medical problems are present such as anoxia at birth, absence seizures, head trauma, or other problems. We

must examine the raw EEG, whether analog or digital real time, not averaged or derived EEG data.

Secondly, we must look at existing medical research on each problem. For example, if someone were in a spindle coma, we would not train him or her to produce 12-15 Hz activity because that is exactly what is causing him or her to remain in the coma. In 1976, Sterman, Goodman and myself inhibited excessive 12-15 Hz activity in a group of quadriplegics. Their spastic paralysis improved and they were able to feed themselves, which they could not do prior to the feedback. We see excessive 12-15 Hz activity in most individuals with severe spasticity from stroke, head trauma, or cerebral palsy. Obviously in those cases, we would not enhance 12-15 Hz activity, but rather we would inhibit it. Producing more 12-15 Hz activity is not appropriate in these problems and may make them worse.

If we train individuals to produce alpha in the frontal lobes to make them feel better, we are also ignoring an important neurological principle. The brain produces a DC shift from the frontal to the occipital cortex across the surface. It starts with dominant beta in the front and slows down with the DC shift to alpha in the occipital region. Therefore, you would inhibit excessively high beta in the frontal area rather than producing alpha. If you produce alpha in the frontal area of these individuals, you will get disorientation and a histrionic personality along with disorganization. In more than 20 years of neurofeedback training, I have inhibited high beta in anxious or agitated patients and inhibited theta on the right frontal area in genetic unipolar depressive patients with great success. This success is due to following sound neurological principles.

Lastly, if we train individuals to enhance beta or the crossover alpha-beta frequency of 12-15 Hz, the theta amplitude will also increase. Enhancement increased the amplitude of all of the frequencies in the raw EEG. If an individual has severe brain damage, tendencies to be violent, temporal lobe or complex partial epilepsy, beta or 12-15 Hz enhancement can make them worse. If you look at thousands of EEG's as I have, you will see what beta enhancement does.

The brain is primarily inhibitory, not an arousal system as most protocols assume. When damaged, the brain will produce higher amplitude beta to try to inhibit the abnormal amount of theta amplitude increase. The brain is always trying to maintain homeostasis, so we should not oppose this adaptive, normal inhibitory process. We need to inhibit abnormal EEG activity so the balance can return. For more than twenty-five years, I have been inhibiting abnormal EEG slow wave activity or

abnormal high beta activity with tremendous success and with no deleterious side effects.

It is critical to see the raw EEG, to use knowledge of basic neurological principles, and to understand that the brain is primarily inhibitory.

RESPONSE: John Nash, PhD, Past President, SNR, 3300 Edinborough Way, Suite 110, Edina, MN 55435 (E-mail: bmainsc@qeeg.com).

A number of potential ill effects are possible from attempts to apply neurotherapy with clinical patients. With proper care and training, all of these can be avoided rather easily. Clinicians who would use this technology and patients who would attempt to get help with it should be aware of the following considerations:

1. The possibility exists that EMG artifact is poorly controlled during what is intended to be beta up training. Electromyographic activity spans a very broad frequency spectrum and it or its harmonics regularly contaminate the beta spectrum. Headaches can result from training the patient to clench muscles of the jaw or forehead, falsely believing this to be elevated beta. I have seen this happen in a couple of patients who have appeared at my clinic following previous “beta” training, which I believe ended up simply increasing their tonic levels of muscle tension on the temporalis and/or frontalis muscles. Beta and muscle tension should be monitored in a carefully designed display. I prefer both beta amplitude and high band 25-32 Hz be displayed next to each other, so the patient can be trained to reduce the higher frequency activity while simultaneously bursting beta above a designated threshold.
2. False “beta” from EMG artifact will also create apparently abnormal beta coherence, since EMG is random with respect to actual beta activity. I have seen “frontal beta discoherence” (low beta coherence) mistakenly identified when in fact large amounts of poorly controlled frontal and frontotemporal EMG existed. This can usually be seen clearly in the raw data and in “beta” relative and absolute power topographic and statistical maps.
3. The possibility exists of a layperson getting enthralled with theta-imagery and training persistently into theta. This could result in depression or dissociation. I have heard rumors that this has occurred when a layperson obtained full access to a neuro-

- therapy device and utilized it without supervision or professional advice.
4. The possibility exists of up-training frontal alpha and creating ADD-like effects. I would expect the same potential for any eyes-open alpha up training.
 5. There is the possibility of well-meaning therapists causing patients to become discouraged from further training by targeting the wrong parameters. This might happen if symptoms were used to deduce protocols and then frontal training was used when a parietal or occipital problem was the root cause. While there is some evidence that theta-down, beta-up training frontally improves EEG parameters widely across the cortex, this may not be true in all cases. I like to use what I call “functional” QEEG (FQEEG). By this I mean utilizing not only eyes closed database norms, but also looking at alpha blocking and theta/delta patterns as they change from eyes closed to eyes open, reading, listening, mental math and drawing. This gives me nice pictures of regional failures of activation during particular activities, which usually correspond well to the patient’s reports of difficulties with those tasks.
 6. The possibility clearly exists for a person with inadequate training in psychotherapy (e.g., cognitive behavior therapy, interpersonal therapy) or peripheral biofeedback choosing neurotherapy simply because that is the tool they have. This could easily result in unproven or unnecessarily long treatments being administered (e.g., if extensive alpha training were given for tension headaches, when home audiotaped progressive relaxation and a few sessions of EMG feedback might well work faster). Similarly, one would be very concerned if treatments with well-proven effectiveness (cognitive behavior therapy [CBT], and/or interpersonal therapy, and/or medications) were ignored in the treatment of depression in favor of neurotherapy as a primary treatment. A reactive or even single major depressive episode typically responds to 6-20 skillful cognitive therapy sessions. This has been well known and demonstrated in research (DeRubeis, Gelfand, Tang, & Simons, 1999), which concluded that, “antidepressant medication should not be considered, on the basis of empirical evidence, to be superior to cognitive behavior therapy for the acute treatment of severely depressed outpatients” (p. 1007). So 30-40 sessions of neurotherapy instead of CBT would be inappropriate except in a patient with recalcitrant, treatment-resistant depression, or recur-

rent depression, where I would certainly consider adding neurotherapy to the treatment plan. Proper warnings of the relatively experimental/investigational nature of neurotherapy for depression would naturally be given to the patient. Further, I would always use functional QEEG (FQEEG) to assess the actual patterns existing in the particular patient, rather than relying on belief structures that are not firmly substantiated by data (e.g., that depression is always related to excess left frontal alpha). In point of fact the person may have recurrent depressions from a genetic predisposition, helplessness resulting from poorly treated ADHD, from an old traumatic brain injury, from global excess theta or from many other different underlying causes. These various causes have different QEEG patterns and one should consider the possibility that they may require rather different neurotherapy treatment approaches. In the absence of large studies demonstrating effectiveness on specific subtypes of depression, I rely on targeting the specific departures from normal range identified by FQEEG.

7. The possibility exists for emotional reactions to occur that the neurotherapist is not trained to deal with, particularly if the neurotherapist is not a licensed mental health care provider or working under their direct supervision. It is my belief that neurotherapists should not be offering treatment to patients with DSM-IV diagnoses unless they are licensed by their state to provide mental health diagnosis and care in conventional ways, or unless a licensed healthcare provider who is also expert in neurotherapy directly supervises them. This latter condition is necessary so that one has some chance of estimating the source of treatment effects, side effects, and the necessary mixture of treatment approaches (e.g., neurotherapy, cognitive behavior therapy, medications).
8. The possibility of over training in some direction exists; for example, training to a point of too little slow wave activity and too much beta activity, which could result in an overdriven or anxious state. When I see short tempers, excessive ego-involvement, and irritability, I cannot help but wonder if excessive training in one particular direction may play a role. One's enthusiasm for effects must be carefully tempered by accurate phenomenological observations of the person. Affect, thought content, rapidity of thought and speech and a host of non-verbal signals—"behavioral manifestations of internal responses," as one of my trainers

called them—should be carefully monitored during and for a period of time after training. Five sessions of coherence or synchrony training may produce nice results, but this does not mean that twenty sessions of the same thing will do even better. One should establish measurable goals before treatment is begun that will serve as signals to terminate a particular training (e.g., normalization of a continuous performance task).

9. I also believe that there is the possibility of decompensation in a borderline patient or other unstable person undergoing neurofeedback training. This could occur with EEG-driven technologies. In fact, I saw this occur while watching a “demonstration” some time ago. One should never take a person any further into pain (e.g., memories, regrets, conflicts) than one has established the ability *beforehand* to quickly lead them to their own resources of comfort and control. One might train with autogenic therapy, SMR, or open focus alpha while simultaneously asking the person to recall powerful moments of success, comfort and safety that they have experienced *or imagined experiencing*. One might then establish a quick, cued access to such states, and could then engage deep interior work, alpha-theta assisted, with fair safety from emotional harm. By alpha-theta assisted, I mean left occipital alpha-theta up training, as Peniston (Peniston & Kulkosky, 1990) described, or any other technique designed to help the person “suspend” in the hypnogogic reverie state between waking and sleeping. Edward Maupin (1965) and Gill and Brenman (1959) termed this “regression in the service of the ego.” As with any neurotherapy application where relatively little published outcome data exists, one should obtain a formal informed consent.
10. It should be possible when using neurotherapy with a bipolar patient to facilitate either too much excitement or too much calm. I have worked with only a couple of bipolar patients. One showed a short-lived hypomanic state during left posterior alpha training. This was easily and quickly reversed with 12-15 Hz central up training. I speculate that the enhanced alpha might have brought too many cortical resources into an allocatable condition too quickly.

In closing I want to say that I have used various types of neurotherapy with about a thousand patients, most of who had significant difficulties and the majority of who had multiple previous unsuccessful attempts to

get help from medications and conventional psychotherapy. I have large numbers of very happy former patients who have gained significant control over their symptoms. I have had no reports of persistent negative side effects beyond fatigue following sessions and occasional feelings of eyestrain or mild headache, usually occurring in patients with traumatic brain injury. Focusing the patient on keeping very relaxed, loose jaw and relaxed eyes during the training minimizes these effects. The fatigue and strain invariably passes by the next day and appears to be the result of the hard work during the session. If one is well trained and thoughtful about the application of this technology, one will “first do no harm.”

REFERENCES

- DeRubeis, R. J., Gelfand, L. A., Tang, T. Z., & Simons, A. D. (1999). Medications versus cognitive behavior therapy for severely depressed outpatients: Mega-analysis of four randomized comparisons. *American Journal of Psychiatry, 156*(7), 1007-1013.
- Gill, M. M., & Brenman, M. (1959). *Hypnosis and related states: Psychoanalytic studies of regression*. New York: International Universities Press.
- Maupin, E. W. (1965). Individual differences in response to a Zen meditation exercise. *Journal of Consulting Psychology, 29*, 139-145.
- Peniston, E. G., & Kulkosky, P. J. (1990). Alcoholic personality and alpha-theta brain-wave training. *Medical Psychotherapy, 3*, 37-55.