Biofeedback
Volume 43, Issue 1, pp. 31–37
DOI: 10.5298/1081-5937-43.1.05

Special Issue

Neurofeedback for Traumatic Brain Injury: Current Trends

J. Lawrence Thomas, PhD, BCN,[1] and Mark L. Smith, LCSW, BCN[2]

[1]The Brain Clinic, New York, NY; [2]Neurofeedback Services of New York, New York, NY

Keywords: neurofeedback, LORETA, Z-score training, traumatic brain injury, infraslow fluctuation neurofeedback

Traumatic brain injuries constitute significant health and societal problems which can be ameliorated with some recent developments in neurofeedback. The field of neurofeedback has evolved from single channel to multiple-site training, and with LORETA Z-score training, deeper levels of the brain can reached. Neurofeedback for traumatic brain injury patients may provide improvements never before possible.

Introduction

Traumatic brain injuries (TBIs) constitute a major health problem, since there are from one to two million TBIs in this country every year, mostly from car accidents and falls (Corrigan, Selassie, & Orman, 2010; Novo-Olivas, 2014). The majority, probably 80%, are mild brain injuries (Bernad, 1988; Hoffman, Stockdale, Hicks, & Schwaninger, 1995); therefore, these would be the most likely candidates for neurofeedback treatment. But this number also might be underestimated, since many of these injuries may go unreported (Powell, Ferraro, Dikman, Temkin, & Bell, 2008). It is estimated that it costs some $60 billion dollars per year for this substantial public health problem (Corrigan et al., 2010).

Other causes of brain injury have been discussed by Thornton (2014). Concussions in football have a 72% chance of happening in every NFL football game. Of the veterans returning from Iraq, an estimated 22% have had a TBI, which totals about 308,000 soldiers. Brewer et al. (2010) estimated that there are 1.25 million emergency room (ER) visits related to brain injuries each year, but also pointed out that an estimated 56% of the TBIs are not diagnosed in the ER. Added to this, Langlois, Rutland-Brown, & Wald (2006) estimated that there were 3.8 million sports-related concussions yearly of all ages (including children's sports injuries) in the USA.

Mild traumatic brain injury (MTBI) is usually defined as having a loss of consciousness of less than 20 minutes, or a posttraumatic amnesia of less than 24 hours (meaning an altered state of consciousness, such as confusion or disorientation, and the time from the accident until there is reliable and consistent memory). These indicators of severity, however, do not predict the outcome of enduring cognitive deficits in the patient (Zasler & Katz, 2013). In this paper, MTBIs will be the primary focus. Severe cases of brain injury are usually not treated with neurofeedback, although there are exceptions (Larsen, 2009).

TBI sequelae can include problems of cognition, behavior, emotional sensitivity, attention, and many other symptoms. Patients can frequently become much more impulsive, appear to have poor judgment, have memory and word finding problems, and often are not very aware of their problems. Planning and organizing can also be significant deficits (Varney & Roberts, 1999).

Details of TBI physiology and neuropathology are numerous and complex, and are beyond the scope of this article, but can be found in other sources (Thornton, 2014; Thornton & Carmody, 2010; Zasler & Katz, 2013). One of the most extensive texts on TBI and its neuropsychology and physiology is *Concussive Brain Trauma* (Parker, 2012).

Neurofeedback Can Help Where Other Treatments Cannot

Neurofeedback is in the unique position of being able to change the physiology of brain-injured patients. Most healthcare professionals believe that once an adult has sustained brain damage, the results are permanent, while others believe that the vast majority of MTBIs resolve completely within a year or two (McCrea, 2008). Thornton (2014) has argued that those once thought to have recovered completely from a MTBI still have biological markers which reveal their impairments. Thatcher, Biver, and North (2015a) has shown that a quantitative electroencephalogram (QEEG) can reveal MTBIs with a high degree of scientific validity (Thatcher, 1999, 2011; Thatcher, Biver, McAlaster, & Salazar, 1998; Thatcher & Lubar, 2009, 2014; Thatcher et al., 2001). Prodan, Vincent,

and Dale (2014) have noted along with Thornton (2014) that there are biological markers of MTBI worth noting, especially since some professionals doubt the legitimacy of many MTBI cases. A recent article noted that the coating of platelets in mild brain injury lasts for a much longer time than in normal subjects and may constitute a new biological marker of MTBI (Prodan et al., 2014).

As this technology improves, neurofeedback could take a central place in the rehabilitation of those with MTBI. At present, the studies are limited in several ways. Many studies have a small number of subjects, or are a series of case studies. Sometimes different diagnoses are presented in a case series (Foster & Thatcher, 2015; Smith, Collura, Ferrara, & de Vries, 2014; Tinius & Tinius, 2000). Randomized, placebo controlled, double blind studies are rare in neurofeedback, and we have not found one with TBI. Admittedly, there is an inherent problem in dealing with MTBI and neurofeedback: there are probably no two brain injuries alike. Also, the treatment protocols tend to be very individualized in an attempt to match the patient's brain parameters, so the possibility of having a matched control group that can be randomized is difficult. Secondly, Hammond (2011a) has argued that the supposed "gold standard" of placebo, double blind studies that are so common in pharmacology research may not be the best way to determine effectiveness in neurofeedback studies. Indeed, many drug studies using these research constructs have only demonstrated marginal benefit when scrutinized.

Basics of QEEG and Neurofeedback Protocols

Basic EEG frequencies and QEEG. Everyone has electricity all over their body, and in the brain this electrical activity is measured in terms of its brain waves; the unit of measure is microvolts. Brain waves occur in different frequencies, understood in cycles per second, or in Hertz (Hz). All frequencies occur in all parts of the brain, but in different conditions of the brain, the distribution of the frequencies can take on specific proportions. The slowest brain wave frequency is delta, which ranges from 0.5 to 4.0 Hz, and next are theta (4–8 Hz), alpha (8–12 Hz), beta (12–30 Hz), and gamma (30–45 Hz). Be aware that different researchers define these bands in different ways.

The brain wave frequencies are measured at certain locations or sites. The system of location, the "10–20 system," specifies the site locations (19 or 21 sites). For example, Cz is at the top of the head; Fpz is in the middle of the forehead, about an inch up from the midpoint between your eyebrows. Frontal sites include Fz, F3, F4, and posterior sites include P3, P4, PZ. You can choose to train some frequencies up (or to be more active in microvolts), and some frequencies down, or to be "inhibited." Thus, one protocol could be to train Fz 12–18 Hz up and 4–7 Hz down (or "inhibit") at the same time. In order to determine precise protocols for doing neurofeedback, it is common to get a quantitative electroencephalograph (QEEG), sometimes called a "brain map." By converting the EEG data into statistics, patients can be compared to a normative group, and this can guide training of these brain waves to improve functioning—and this is neurofeedback.

The QEEG method measures all frequencies (delta, theta, alpha 1, alpha 2, beta 1, beta 2, beta 3, gamma) at each of the 19 (or 21) sites in terms of *absolute power* (microvolts) and *relative power* (percentage), the *ratios* of each frequency to every other frequency, plus all possible pairs of sites in terms of the connectivity variables (*coherence*, *asymmetry*, and *phase*). The result is some 2,500 variables. This complex brain wave data is analyzed by a computer program and compared to people the same age, and the result is the QEEG. These variables are compared to the normative database that contains the data for all ages; therefore, the patient in question is compared to people the same age. Of importance are the deviations the patient has compared to the norms with respect to all these variables, which is shown in terms of standard deviations and Z-scores. What is interesting is that the QEEG patterns are lawful and describe certain pathologies in a reliable way. Thus, attention deficit disorder, dementia, affective disorder, traumatic brain injury, and obsessive–compulsive disorder all have distinctive patterns to their QEEG. A detailed history of this process can be found in Thatcher and Lubar (2009, 2014).

Basics of neurofeedback training. Assessing the EEG will render data regarding the 19 sites of the QEEG, with many parameters available with regard to treatment. In the early days of neurofeedback, single sites were treated (e.g., training 12–15 Hz at C4), and if two sites were treated, one would follow another. Then training two sites were found to be effective (e.g., T3 and T4). As practitioners explored other parameters, two sites began to be trained in terms of the coherence between the two sites. In more recent times, several sites can be trained at once, as in Z-score training. And now, with LORETA neurofeedback, the areas beneath the surface of the cortex can be trained, and connections between networks or regions can be trained. Thus, there has been a progression over the last 20 years in neurofeedback of being able to train and improve the electrophysiology at more and more sites with more complexity.

The EEG electrodes can be placed in one or more sites, and feedback is displayed to the patient on a computer

screen so that the dysfunctional frequencies are trained down, and the "good" waves are trained up. The display can be the brain waves themselves, a computer game generated by the computer, or even a movie. For example, the patient is asked to keep the animation going, and by operant conditioning (e.g., the flickering of the movie), the patient trains his or her brain waves to be more normal. There is a fair amount of research regarding the effectiveness of neurofeedback (see Monastra, 2005; Thompson & Thompson, 2003; Yucha & Montgomery, 2008). Neurofeedback has been shown to be effective for attention deficit disorder, chronic pain, traumatic brain injury, and other brain disorders (Yucha & Montgomery, 2008). Duffy (2000), a neurologist, stated in a special issue of *Clinical Electroencephalography* devoted to neurofeedback that "if any medication had demonstrated such a wide spectrum of efficacy, it would be universally accepted and widely used" (p. v). As will be seen below, there are some limitations to the research on the effectiveness of neurofeedback with respect to TBI.

The Early Days of Neurofeedback for Mild Traumatic Brain Injury

The field of neurofeedback is remarkably new: The "early days" can be considered to be 20 to 30 years ago. The current developments include some very sophisticated methods of training brain problems that are far more complex than the early studies. However, the early studies are instructive, and can be reviewed to gain insights into the issues of neurofeedback when the research was simple. Most of the early publications were case studies, cases series, or clinical studies; few had matched control subjects were employed.

Ayers (1987) reported on doing neurofeedback with brain-injured patients and compared their progress to that of other patients doing psychotherapy alone; this could qualify as a controlled study. There was a substantial reduction of symptoms such as anger outbursts, mood problems, and anxiety. Those patients who continued with psychotherapy alone did not show improvement. Byers (1995) treated a 58-year-old woman with MTBI at two sites, with 31 sessions. His pre- and postmeasures showed improvement in several areas of functioning as well as test measures. Hoffman and his associates did studies from 1981 to 1996 (Hoffman et al., 1995; Hoffman, Stockdale, & van Egren, 1981, 1996) showing that QEEG-guided treatment of closed head injury patients produced 60% improvement in symptoms and cognitive performance after 40 neurofeedback sessions.

Other articles that have provided brief reviews of neurofeedback with traumatic brain-injured patients include Foster and Thatcher (2015), May, Benson, Balon, and Boutros (2013), Novo-Olivas (2014), and Thatcher (2000). Common criticisms include the lack of controlled studies, mixing different diagnoses, inadequate measures beyond self report, and weak neuropsychological testing. Nearly all report on QEEG improvements, but unfortunately QEEG does not correlate well to symptomatic improvement. These reviews also point out that cognitive rehabilitation and most other methods of helping the TBI patient are inadequate, and that neurofeedback has a unique opportunity to help these patients in new ways. Now let us turn to new models of neurofeedback that are complex and exciting.

Neurofeedback Approaches Relevant for Traumatic Brain Injury

Z-Score Neurofeedback

This is sometimes called *live Z-score training*. It involves using four or more sites (up to 19; the 10–20 sites) for training up to all the variables all at once. Above, I introduced the idea of Z scores, which indicate the standard deviations from the normative database when comparing the patient's variables to the norm. The variables such as absolute and/or relative power; ratios of two frequencies; connectivity variables such as coherence, asymmetry, and phase are all compared to the patient's age group. Z-score training is when all of these variables are coaxed to being normal. For example, when four sites are being trained there are 248 variables that are being trained at once, and live; these variables are all referenced to a normative database for the patient's own age group. All of these data are converted into one metric; this metric is the proportion of these Z scores that are falling within an adjustable range of scores.

If the feedback is a DVD movie, the movie flickers as the training stimulus. When the movie is dim, the brain is not cooperating, and when the movie is bright, the brain is cooperating. In this way the operant conditioning trains the brain to become more normal; the assumption is that if the brain EEG variables become more normal, the symptoms of the patient's brain dysfunction will improve. Collura (2008a, 2008b, 2014) has given some detailed explanations of the Z-score neurofeedback methodology; and Thatcher and Lubar (2014) have recently published a book on Z-score training.

LORETA Neurofeedback

LORETA stands for *low-resolution electromagnetic tomography analysis*, which takes the QEEG data and renders the

sources of the EEG deep in the brain. In this way, the underlying areas of neuropathology can be revealed in three dimensions. Thus, LORETA images show the areas beneath the surface of the cortex as well as well as cortical surfaces of the brain that are the sources of the problem(s) of the particular patient. In order to obtain data to illuminate the three-dimensional properties of the brain, a QEEG is done with all 19 channels. With these data, regression equations are utilized to help locate the sources of the problem(s) and give direct information as to how and where to train the brain with neurofeedback. In the latest developments, the QEEG data plus the NeuroGuide Symptom Check List (Applied Neuroscience, Inc., Seminole, FL) can indicate *networks* of Brodmann areas to train that reflect the cognitive problems and emotional/behavioral symptoms reported by the patients. The neurofeedback session can display the cortical surface and subsurface networks being trained in a live fashion.

Foster and Thatcher (2015) presented a LORETA case series of 11 subjects who came from the Veteran's Administration hospital with symptoms of both PTSD and MTBI. Each received individualized LORETA Z-score training, and each subject rated their symptom improvement after each session for 12 to 20 sessions. The 19-channel LORETA QEEG was done initially to select the region of training; the LORETA methodology allows the neurofeedback training to be three-dimensional in nature so areas beneath the surface of the cortex can be trained. In Thatcher's methodology (Thatcher, Biver, & North, 2015b), the Symptom Check List is reported by the patient, and this is referenced to the likely areas of the brain responsible for these symptoms, according to functional neuropsychological literature. When both the LORETA data and the Symptom Check List are "matched," this will indicate the training area(s). With each case, the training is unique to the patient. A system of rating one's goals was also employed in this study so that the patient could rate their progress in an ongoing fashion. All of the patients improved in terms of their symptom reduction and positive goal achievement.

This case series illustrates the fact that each patient is different and needs an individualized treatment approach. The method that Thatcher has devised utilizes the functional neuroanatomy knowledge of the likely localization in the brain of the reported symptoms, and combines this knowledge with actual neurophysiological data of the QEEG LORETA to create a highly specific neurofeedback training/treatment plan. This is essentially personalized medicine in training the brain to correct its own neurophysiology. A recent book by Thatcher and Lubar (2014) entitled *Z-score Neurofeedback: Clinical Applications*, details this methodology and the underlying scientific issues. Thatcher's QEEG software, NeuroGuide, allows the professional to assess and conduct a variety of neurofeedback protocols at a number of levels (see www.appliedneuroscience.com).

Doing cognitive remediation with neurofeedback. This method was introduced by Tinius and Tinius (2000), and consists of doing neurofeedback while doing cognitive remediation at the same time. In this study, 15 MTBI patients received neurofeedback while doing computerized cognitive training, and their improvements were compared to healthy controls with both groups given pre and post measures. The MTBI patients improved on 10 out of 12 neuropsychological measures and improved to the level of the healthy controls. A problem with this study is that it combined TBI and learning disabled patients.

Activation QEEG neurofeedback. Thornton (2000) has developed a model of doing neurofeedback that involves first doing several QEEG assessments under different cognitive conditions, then training the problematic cognitive functions while doing neurofeedback. As discussed above, a QEEG assessment involves collecting EEG data at 19 sites on the scalp, then converting this data by computer into how a patient's QEEG variables compare to those in his or her age group. The usual two conditions of collecting these data are with eyes open and eyes closed. Thornton and Carmody (Thornton, 1996, 2000, 2003, 2014; Thornton & Carmody, 2005, 2008, 2009, 2010) have pointed out that the brain may reveal different patterns of EEG under different cognitive task conditions, thus rendering the usual QEEG brain maps eyes-open or eyes-closed conditions at variance with the cognitive problems that may occur with patients seeking neurofeedback treatment. Thornton does a QEEG for each cognitive condition, a few examples being auditory attention, visual attention, reading, and other cognitive tasks for a total of 10 cognitive tasks. Compared to a control group (Thornton & Carmody, 2008), the group of brain-injured subjects improved with this method with effect sizes above 2.0. If this work can be replicated, it could mean a substantial contribution to the treatment of traumatic brain-injured patients.

Hemoencephalography (HEG). HEG biofeedback trains the patient to control a close correlate of the frontal lobe cerebral blood flow. An infrared camera sensor, which reads temperature (a close correlate of blood flow), is placed on the forehead and the patient learns to control the heat by

watching the display. In the case of the passive infrared hemoencephalography, the display is a movie—any DVD the patient wishes to see. If the frontal lobe blood flow and temperature remain high enough, or over the autothreshold, the patient can continue to watch the movie. When the temperature drops (believed to be related to activity in the anterior cingulate gyrus), the movie stops; by focusing on a bar graph display, the cortical activity increases, the temperature increases over the threshold, and the movie starts again. The therapist can make the task easier or more difficult. The autothreshold aspect of this system follows the temperature of the frontal lobe, which naturally fluctuates, so the movie will stop sooner or later. The patient must then focus on a part of the computer that can raise the frontal lobe temperature; when it goes over the threshold, the movie continues.

The HEG method of neurofeedback is a new kind of treatment, and there is little research as to its effectiveness, and no studies (to our knowledge) with those with traumatic brain injury. This biofeedback system was originally designed for migraine headache treatment, and has shown promising results. Carmen (2004) took 100 migraine patients that had been through many previous treatments, including many trying several medications, with little success. Positive results were usually seen in six HEG sessions, and over 90% of the patients reported significantly positive results, according to their own report. I am including this method of neurofeedback because it is specifically designed to train the frontal lobe cerebral blood flow to increase, and this has been known to be a very common area of brain injury with respect to MTBI (Thatcher, 2011).

The LENS model and TBI. The low-energy neurofeedback system, or LENS, is a method that measures the dominant frequency of one or more of the 10–20 sites, and gives the patient a tiny electrical stimulation (about one-millionth of a microvolt) at that electrode. The frequency of this very small and brief (from 0.01 seconds to 60 seconds) stimulation is offset by several Hz. Ochs believes that this "offset" jars the brain to reregulate itself. The brain seems to respond to the tiny stimulus, and appears to move towards a more healthy homeostasis, sometimes with dramatic results (Larsen, 2006), even with severe TBI.

The only controlled study of the LENS method that we know of is the Schoenberger, Shiflett, Esty, Ochs, and Matheis (2001) study. This study was done with a previous version (called Flexyx) of the LENS system. In this study, 25 sessions were given to the immediate treatment group and later to a wait-list control group, which received treatment after the first group. Positive results were found in several psychometric measures, as well as positive improvements in social and occupational outcomes. In another case report and explanation of the LENS system, Ochs (2011) reported that a TBI patient was helped substantially. Ochs also provides a detailed explanation of the method and why he thinks it works. The book, *The Healing Power of Neurofeedback* (Larsen, 2006), notes several cases of TBI helped by the LENS method and other disorders as well.

Infraslow fluctuation neurofeedback. In this new neurofeedback model (ISF), very slow frequencies are trained, at the level of 0.1 and below, to as low as 0.001 Hz. Very low frequencies have been researched for more than 40 years, but mostly in languages other than English. Using this level of physiology in neurofeedback has been hampered by technological limitations, but recently, modern electronic instrumentation has made available the use of slow frequencies for therapeutic neurofeedback. In the Smith et al. (2014) article, several cases are presented that report positive results using this new technology, but these cases are from a variety diagnostic groups and the pre and post measures consist of mostly subjective reports. As with most of the new models of neurofeedback, larger, controlled studies are needed. It is clear that the innovations in neurofeedback are outstripping the ability to do meaningful research to prove their effectiveness.

Clinical Neurofeedback for Traumatic Brain Injury

Overall Reviews

A detailed review of the positive effects of neurofeedback for those with TBI is beyond the scope of this article. The following review articles and controlled studies can serve as evidence that neurofeedback appears to be effective for TBI patients, but that rigorous research standards have not been part of this progress up to this point in time.

The May et al. (2013) article recently reviewed 22 neurofeedback TBI articles, and reported that all the studies reported benefit to the patients. However, none of these were randomized, placebo controlled, double-blind studies. The areas of improvement in these studies, taken as a whole, included improvements in attention, impulse control, and processing speed. If the studies had psychometric measures, they were usually brief assessments of cognition, such as the RBANS (repeatable battery for the assessment of neuropsychological status; Randolph, 1998), a very brief neuropsychological battery, or various means

of the patients reporting their symptoms or improvements. Some studies showed improvements in QEEG variables. While there is a growing literature of neurotherapy helping those with brain injuries, there are some inherent problems in the field with respect to doing controlled studies. It has been pointed out that randomized, double-blind, placebo controlled studies are nonexistent with regard to neurotherapy treatment for brain injuries (May et al., 2013; Novo-Olivas, 2014; Thatcher, 2000), it is also likely that this level of research design is not appropriate for this field. Meanwhile, the landscape of neurotherapy keeps expanding, and new technologies, such as LORETA, Z-score training and infraslow frequency (ISF) are being used by clinicians. Indeed, the technological innovation is outpacing the ability of the research community to prove the effectiveness of these new methods.

Neurotherapy for traumatic brain-injured patients may be valuable for improving the physiology and cognitive functioning of the brain—the extensive bibliography can attest to this. However, these are people with complex and perplexing symptoms we have in the treatment situation. It is may be that the neurotherapy practitioner is the only healthcare provider for the brain-injured patient. Becoming aware of other issues is important, and some of these are sketched out below. Further, some resources are noted after this section so that these can be used for our professional development.

The next developments. We saw how the progression of the field went from training single sites, to pairs of sites, to the connectivity variables between two sites (i.e., coherence), to many sites (Z-score training), and then to reaching into the deep areas of the brain (LORETA). Added to multiple sites is Thornton's (2014) model of doing neurofeedback while doing a cognitive task in order to improve specific cognitive abilities, and the Tinius and Tinius (2000) method of doing actual cognitive remediation while doing neurofeedback. The complexity of providing neurofeedback has increased in the number of sites, and adding cognitive tasks while doing neurofeedback. We have an interesting future in this field, one that can benefit people who have never before had such an opportunity.

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J. Lawrence Thomas

Mark L. Smith

Correspondence: J. Lawrence Thomas, PhD, BCN, Director, The Brain Clinic, 19 W 34th St., Penthouse, New York, NY 10001, email: nurosvcs@aol.com, Web: www.thebrainclinic.com.