



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

Neurofeedback Therapy for a Mild Head Injury

Alvah P. Byers

Published online: 18 Oct 2008.

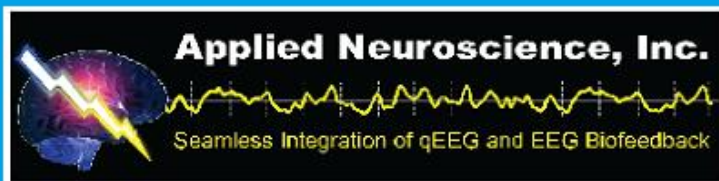
To cite this article: Alvah P. Byers (1995) Neurofeedback Therapy for a Mild Head Injury, Journal of Neurotherapy: Investigations in Neuromodulation, Neurofeedback and Applied Neuroscience, 1:1, 22-37, DOI: [10.1300/J184v01n01_04](https://doi.org/10.1300/J184v01n01_04)

To link to this article: http://dx.doi.org/10.1300/J184v01n01_04

PLEASE SCROLL DOWN FOR ARTICLE

© International Society for Neurofeedback and Research (ISNR), all rights reserved. This article (the “Article”) may be accessed online from ISNR at no charge. The Article may be viewed online, stored in electronic or physical form, or archived for research, teaching, and private study purposes. The Article may be archived in public libraries or university libraries at the direction of said public library or university library. Any other reproduction of the Article for redistribution, sale, resale, loan, sublicensing, systematic supply, or other distribution, including both physical and electronic reproduction for such purposes, is expressly forbidden. Preparing or reproducing derivative works of this article is expressly forbidden. ISNR makes no representation or warranty as to the accuracy or completeness of any content in the Article. From 1995 to 2013 the *Journal of Neurotherapy* was the official publication of ISNR (www.isnr.org); on April 27, 2016 ISNR acquired the journal from Taylor & Francis Group, LLC. In 2014, ISNR established its official open-access journal *NeuroRegulation* (ISSN: 2373-0587; www.neuroregulation.org).

THIS OPEN-ACCESS CONTENT MADE POSSIBLE BY THESE GENEROUS SPONSORS



Neurofeedback Therapy for a Mild Head Injury

Alvah P. Byers

The purpose of this study was to evaluate Neurofeedback Therapy (NFT) for a Mild Head Injury (MHI). The subject was a 58-year-old female who fell and struck her head in 1988. The NFT began in 1994 and was preceded and followed by neuropsychiatric and neuropsychological evaluations as well as quantified electroencephalograms (QEEG). The patient completed a symptom checklist and the Minnesota Multiphasic Personality Inventory-2 (MMPI-2) before and after NFT. Treatment consisted of 31 sessions of NFT. The NFT was designed to enhance the sensorimotor rhythm (SMR) of 12-15 Hertz (Hz) and the beta (15-18 Hz) frequency bands of the electroencephalogram (EEG) while at the same time suppressing the theta (4-7 Hz) frequency band. Twelve sessions were used for SMR NFT and 19 sessions for beta NFT. The comparison of the pre- and post-measures as well as the process measures suggests NFT is a promising intervention for the rehabilitation of patients with MHI. Questions regarding these findings are explored together with suggestions for further research.

Alyce Green and Elmer Green (1975) said, "It may be possible to bring under some degree of voluntary control any physiological process that can be continuously monitored, amplified and displayed." Neurofeedback Therapy (NFT) is an application of this basic biofeedback principle. Simply stated, NFT provides a human subject relatively accurate and relatively immediate information by a visual and/or an auditory signal that a given electroencephalograph (EEG) frequency band at a given amplitude has been produced. The subject is trained to either enhance or suppress selected EEG frequency bands for the purpose of normalizing the disordered EEG pattern.

This is a report of the use of NFT in the treatment of a Mild Head Injury (MHI). A review of the literature reveals NFT has been used for the remediation of neuropathophysiology that appear to be similar to a MHI. Some of those pioneering efforts are relevant to this paper.

Sterman and his associates (Sterman & Friar, 1972; Sterman, McDonald & Stone, 1974) found by training epileptics to enhance the human EEG sensorimotor rhythm (SMR) of 12-15 Hertz (Hz) their seizure management could be improved. Lubar and Bahler (1976) found similar results when they trained epileptics to enhance the SMR frequency and sup-

press the theta (4-7 Hz) slow wave frequency. Lubar summarized the development of his application of NFT to attention deficit/hyperactive disorder (ADHD) and attention deficit disorder (ADD) children in his research recognition award paper (Lubar, 1991). He reported ADD and ADHD children demonstrate an abnormally slow wave activity in the 4-8 Hz range in their EEG, with a concurrent deficiency in the faster beta (16-20 Hz) range, especially in the central-frontal locations (Cz and Fz). He indicates a theta to beta variation of 3.1 or more is abnormal. On the basis of his work, it appears that SMR training leads to reduced hyperactivity while the beta training leads to enhanced attention and concentration. Tansey (1983, 1984, 1985a, 1985b, 1990) reported that children with learning disorders and hyperactivity were helped to overcome the learning disorder and reduce the hyperactivity as a result of training to enhance 14 Hz activity near the Cz location (supplementary motor area). He reported reduced 7 Hz activity. Ayers (1987, 1991) has reported beneficial results for closed head injured patients with NFT in which the patient is trained to enhance beta (15-18 Hz) activity at T3-C3 or T4-C4 while suppressing the slower theta (4-7 Hz) activity. She reported reduction of many of the symptoms of closed head injuries in a relatively short period of time.

The NFT protocols used by the authors summarized above were attempts to train the patient to enhance the EEG frequency bands variously described but in the range of 12-20 Hz while at the same time suppressing a slower frequency band variously described, but in the range of 4-8 Hz. The above authors consider the relatively high amplitudes in the slow wave EEG frequencies and relatively low amplitudes in the faster EEG frequencies "abnormal." It is believed a permanent "normalization" of the EEG pattern apparently takes place as a result of NFT. A wide variation in normal individual EEG records makes it difficult to determine a normal QEEG record. One approach to identifying a normal QEEG is the reference data base of Thatcher (1989).

The diagnosis of a MHI using the EEG record is the domain of medical neurology, however, in the context of this report it is instructive in a general way to be aware of the EEG record that is reported to be consistent with a MHI. Duffy, Iyer, and Surwillo (1989, page 252) indicate, "Diffuse slowing of the EEG is a nonspecific finding that may be seen after concussion."

Ayers (1987) stated the EEG of a closed head injured patient will show:

" . . . petit mal variant activity in the 3-5 hertz range in the cortical area where damage occurred. There will be a generalized slowing of the EEG, most predominate at the site of injury. The amplitude of frequencies in the 4-7 hertz range will be considerably higher at the site of injury. In addition, polyphasic spikes may be seen at the site of injury" (p. 2).

The clinical presentation of a MHI is marked by a multiplicity of symptoms with variable degrees of severity, and a variety of patterns. The DSM-IV (American Psychiatric Association, 1994, pages 133-138 and 148) indicates that the essential feature for the diagnosis of cognitive deficits (dementia) caused by head trauma is memory loss and at least one other cognitive impairment that leads to impairment in social functioning. In addition, a variety of other symptoms are often seen, including depression, irritability, attentional

problems, anxiety, increased aggression, and other personality changes.

Neuropsychological testing can reveal a wide range of cognition deficits among MHI patients. The deficits will depend on the severity of the trauma, as well as the angle of the blow or whether or not the skull was even impacted as in a whiplash. Commonly, one can expect reduced response time or impaired cognitive efficiency even though a given examination task can be accomplished in time. Problems with complex mental processes, information retrieval (dysnomia), light and/or sound sensitivity are often detected. Problems dealing with a high level of social stimulation, such as family gatherings, are commonly seen. Often a patient can do fairly well in a quiet and focused neuropsychological examination, but not so with workplace or daily living tasks when in the midst of distractions (Lezak, 1995).

Many patients recover from a MHI in 6 months to a year by natural history alone (Levin et al., 1987). Still others do not recover (Rimel, Giodani, Barth, Boil & Jane, 1981; Kwentus, Hart, Peck & Kronstein, 1985; Uzzel, Langfitt & Dolinskas, 1987). The percentage of MHI patients who recover varies according to the method of the study and the criteria used (Thatcher, Walker, Gerson & Geisler, 1989).

Currently there are a number of treatment approaches for MHI patients. Interventions such as Compensatory Strategy Training (CST), cognitive retraining, medication, occupational therapy, physical therapy, optometry and ophthalmology, psychotherapy, behavioral therapy, and family therapy all offer interventions designed to promote recovery from MHI. These interventions, though helpful, do not seem to resolve the MHI symptoms according to Ayers (1987) who stated, ". . . there has been a dramatic lack of effective treatment resolution of the post concussion syndrome" (p. 2).

Two hypotheses to be tested in this study were: (1) A patient presenting the symptoms of a MHI, while at the same time showing excessive slow wave (theta) activity, can be trained to suppress the excessive slow wave

activity and enhance the faster frequencies (beta); and (2) Such training will reduce or eliminate the cognitive deficits suffered as a result of the MHI.

Method

Subject

The subject in this study is a 58-year-old, divorced, Caucasian grandmother living alone. She earned A and B grades while graduating from high school. She later earned an Associates degree from a junior college where she continued to carry A and B grades. At the time of her injury she was employed in a sales position requiring her to do a good deal of driving. Her job required that she remember a large number of variables that were a part of the sales task. In her leisure time, she kept, rode, and trained horses, and went boating.

On March 31, 1988, this patient fell on a slippery asphalt parking lot while making a call on customers. She had no memory of striking her head, however, she reported being stunned. After the fall she went to "sleep" in her car for about an hour. She stated she then went home and spent the next three or four days "sleeping" off and on.

Following her recovery from the acute phase of the injuries she attempted to return to work. She found she could not attend to several ideas or tasks at the same time as she had before her injury. She could not recall the many minute variables that factored into each sale. While driving she would forget her location and destination. At times she retreated from the office to her car to do her desk work because the background noise in the office confused her. However, while in her car, she would find that she "spaced out" and still was unable to complete her work. She was fired from her job due to inefficiency. She had never before lost a job. She later tried several job placements, but lost each one for the same reasons. She attributed her symptoms to stress in response to the physical injuries and constant pain in her neck, shoulder, and knee. In addition, she was diagnosed in 1990 by her family physician as having an Epstein-Barr

Virus (E-BV) infection. He based his diagnosis on elevated antibodies to the E-BV, together with her clinical symptoms.

The patient was treated with a focus on what was believed to be her medical problems as a result of her fall and the diagnosis of E-BV infection. In fact, an orthopedic surgeon, on July 24, 1992, rendered an opinion that the patient did not suffer any limitations due to her fall in 1988. He thought she was a "symptom magnifier."

On August 20, 1992, a treating orthopedic physician referred the patient to me for assistance in pain management. She was taking Prozac 20 mg in the evening. In that first contact with her, she presented pain in her neck, shoulder, and knee, blurred vision, tinnitus and numbness in her hands and feet. She complained of sleeplessness, with her mind racing over financial matters. The insurance company she had invested in for retirement had gone bankrupt. This was a major source of stress. She believed all of her "mental problems" were due to the E-BV infection, chronic pain (related to her physical injuries), and the stress resulting from her severe financial losses. Her family physician and orthopedic physician supported this belief.

The initial referral was for biofeedback-assisted pain management. No psychological screening or testing was allowed by the referring orthopedic physician and insurance company. In response to this referral, this writer conducted psychological interventions aimed at attitude restructuring for a more positive outlook and biofeedback-assisted relaxation therapy for stress reduction. The patient learned to voluntarily warm her hands to criteria (95.5[∞] F). She also responded to positive attitude restructuring. During this treatment period, this writer, through neuropsychologically-focused inquiries, developed the suspicion that the patient had suffered a MHI and suggested this to the patient and her referring physician. He elected to ignore the MHI hypothesis.

On July 8, 1993, eleven months later, a treating psychiatrist, to whom she had been

referred by the orthopedic physician, referred the patient back to this writer for a one-session clinical interview to rule out post concussion syndrome (PCS). A neuropsychological interview was conducted. A pattern of possible cognitive deficits was confirmed. She now revealed the symptoms reported above, which suggested short-term memory or retrieval problems, impairment in complex mental processes, concentration and attention problems, especially in context of distraction, receptive aphasia, disorientation to location and directions, and failure to recognize current friends until some time lapse. Dyscalculia, and agnosia were also suspected. These impairments made it impossible to hold her job and caused marked difficulty in everyday living. A complete neuropsychological evaluation was recommended to rule out a MHI.

On October 15, 1993, an independent neuropsychiatric evaluation was conducted at the request of the insurance claims adjuster. The neuropsychiatrist rendered a diagnosis of "post concussion syndrome caused by the fall in 1988" (A.C. Roberts, personal communication, October 15, 1993). In addition to the physical symptoms noted by the other physicians, his report went on to suggest, "The diagnosis of E-BV infection needed to be reevaluated."

Subsequently an orthopedic surgeon rendered an independent medical evaluation on November 8, 1993. His impressions confirmed her thoracic and lumbar spine strain injuries as well as contusion of the left shoulder and sprain of the left knee. He did not mention the E-BV infection and attributed her cognitive deficits, especially memory problems, to a PCS. He agreed that further studies were needed to rule out a MHI.

The patient was subsequently referred by the treating psychiatrist to another psychologist for an independent neuropsychological evaluation. He confirmed the diagnosis of a PCS. He concluded his evaluation with:

"These findings and an examination of the patient's life history lead to a clear diagnosis of organic brain syndrome due to a traumatic brain injury she suffered in her fall in March of

1988. The visual-perceptual and executive impairments...are a pattern typical of a coup-contrecoup type closed head injury. The impact of the fall was occipital and appears to have resulted in contrecoup injury to the frontal lobes" (R. A. Kooken, personal communication based on a complete neuropsychological evaluation, February 25, 1994).

The treating psychiatrist armed with this neuropsychological evaluation referred the patient back to this author for Compensatory Strategy Training (CST). On April 20, 1994, the patient arrived for her first session. I informed the psychiatrist that in addition to the 12 once-a-week sessions for CST, I recommended daily NFT and would initiate it at no additional cost.

Clinically, the patient began CST and NFT with a more open willingness to discuss her symptoms. The previous medical diagnosis and her denial of cognitive symptoms for fear that someone would think she was crazy was no longer keeping her silent regarding her symptoms. She stated briefly after the car has stopped the car seems to continue to move; there are problems with balance on her boat; difficulty parking the boat; poor memory, especially while reading, such that reading was no longer enjoyable (she would lose her place between sips of coffee and not be able to remember the content of what she was reading); depression; disorientation to location, such as after shopping finding it difficult to find her way home; and living in a "fog."

Procedure

Before treatment the patient signed a consent for treatment form and the agency disclosure forms. Pre-treatment and post-treatment measures in this study consisted of a quantitative electroencephalogram (QEEG), the MMPI-2, neuropsychological testing, neuropsychiatric interviews and the patient's completion of a symptom checklist. Assessment of the patient's progress was accomplished by daily chart notes and NFT session records.

A pre- and post-QEEG was conducted in eyes closed relaxed (ECR) condition with 19 active locations on the scalp according to the

international 10-20 location system (Jasper, 1958). The Neurosearch, 24-channel computerized EEG (Lexicor Medical Technology, Inc., Boulder, Colorado) was used to produce the QEEG record. The QEEG was taken using the Electro-cap (Electro-cap International, Inc., Eaton, Ohio). Each lead was checked separately. Impedance was judged acceptable when each electrode impedance registered below 5 k ohms. Amplifier gain was set at 32000. High pass filter was set at .5 Hz, with low pass filter set at 32 Hz. Sampling rate was at 128 samples per second per channel. Each QEEG record was edited to eliminate artifact contamination. Frequency analysis was performed using a Fast Fourier Transform (FFT). The QEEG frequency bands chosen were theta, 3.5-7.0 Hz, alpha, 7.0-13 Hz, and beta, 13-22 Hz (Thatcher, 1989).

The Biolex EEG computerized biofeedback software was used with the Neurosearch 24 described above. The same settings, analysis, and electrode impedance criteria were utilized during the NFT sessions. However, in the NFT sessions the frequency bands used were: theta, 4.0-7.0 Hz; alpha, 8.0-12.0 Hz; SMR, 12.0-15.0 Hz, and beta, 15.0-18.0 Hz.

The patient experienced several losses of relatives and friends in the month of March. Moreover, during this month her father died after six weeks of severe suffering. To cope with this, her family physician started her on Prozac 20 mg each evening beginning March 30, 1994. The evening dosage was sedating for her, which helped her to sleep. This is a known, but relatively unusual response to Prozac. After four weeks, she began on her own to reduce the dosage to every other day. She discontinued regular dosages of Prozac by mid-May, taking one 20 mg tablet "every now and then, about 3 to 4 times a month" at night to help her sleep. This was her pattern throughout the time she was in NFT. Therefore the pre-treatment QEEG was conducted on April 28, 1994 while she was still taking Prozac regularly. The post-treatment QEEG was conducted August 10, 1994 when she was taking Prozac as needed.

Twelve CST sessions were conducted on a

once-a-week basis in which, among other compensations, she was instructed to place checklists in appropriate places in the home and car. These checklists consisted of actions to be carried out before starting or leaving that area or task. She carried a memory pad and a map of the city in her purse. She made a list of all places and errands in chronological order before leaving the house, then carried out each errand one at a time. A grocery list was used while shopping for groceries. She wore her keys on a wrist band.

The training goal in the first 12 NFT sessions was to enhance the SMR frequency band of 12-15 Hz, while simultaneously suppressing the theta frequency band (4-7 Hz) in the ECR condition following the work of Tansey (1984, 1985a and 1985b). The Cz scalp location was used as the active lead with linked ear reference (both ears are used as a reference to the active lead) with a ground on the forehead. The Cz location was also selected based on the work of Tansey (1990). During the SMR NFT, the patient was instructed to use Tansey's imagery and think of herself as a heavy hollow rock and keep the feedback sound on. The patient could hear the feedback tone whenever the SMR amplitude equaled or exceeded the threshold setting, while at the same time she kept the theta band below a pre-set level. If the theta band exceeded the theta pre-set level, even if she simultaneously exceeded the SMR threshold, the reinforcing auditory feedback signal was denied. The SMR threshold and theta level settings were adjusted periodically to insure the patient heard a feedback tone about 70 to 85 percent of the time. The tone was denied when her theta exceeded its threshold setting about 20 to 30 percent of the time. This was done to shape the enhancement of the SMR frequency and suppress the theta frequency. The 12 SMR NFT sessions were discontinued when it appeared the patient was not changing SMR or theta levels.

The SMR training was followed by 19 NFT sessions in which the task was to enhance the beta frequency band (15-18 Hz) used by Ayers (1987, 1991) while simultaneously suppressing the theta band (4-7 Hz). The beta NFT ses-

sions were conducted on the left side of the head as the assumed side of the injury based on the descriptions of the fall and injuries to the left side of the body. The pre-treatment QEEG indicated the highest theta amplitudes recorded in the left mid-temporal region. Because of these indicators, the bipolar T3-C3 montage was used for training (Ayers, personal communication, February 1993).

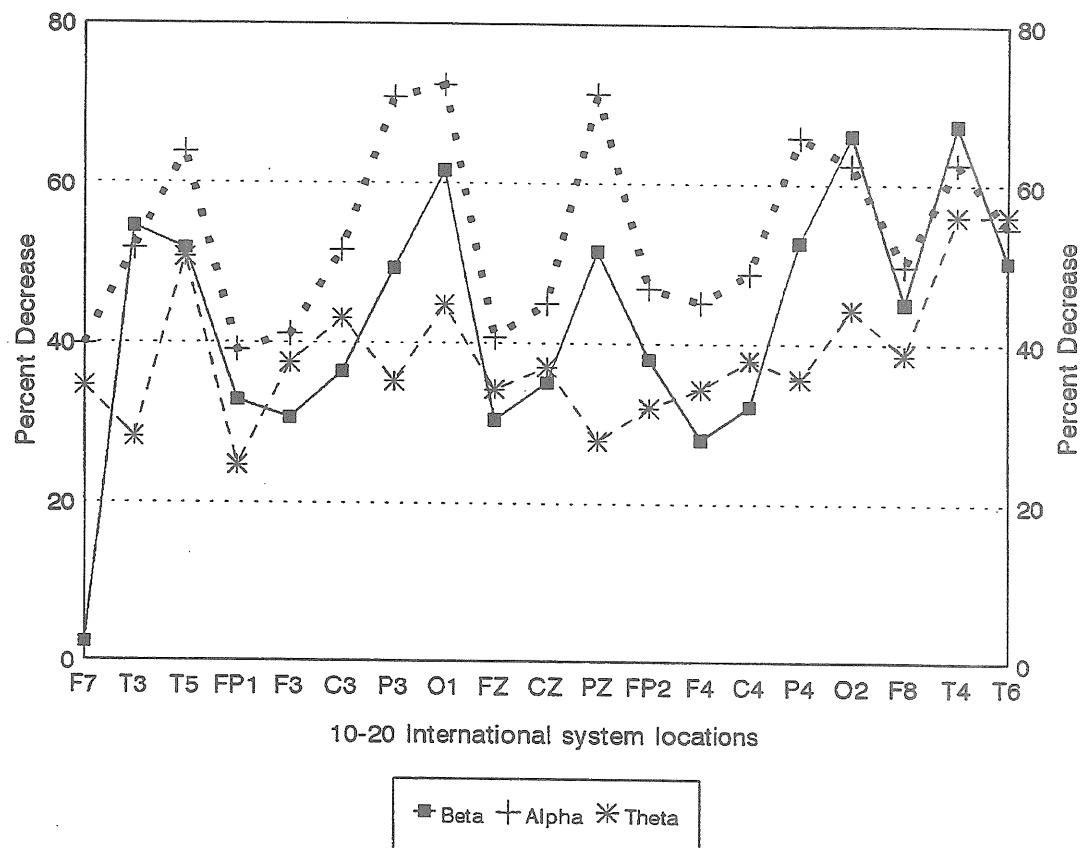
The beta training was conducted with eyes open watching the monitor. When the patient exceeded her pre-set beta frequency band threshold setting, she could see the bar graph on the monitor for beta move above the threshold line and at the same time hear a pleasant reinforcing audio signal. When her theta band (not visible to the patient to avoid distraction)

exceeded the theta pre-set level, the audio feedback signal was denied. The patient was instructed to learn to discover the mental set or strategy that would keep the bar above the threshold setting while maintaining the audio signal. The same shaping technique was applied in the beta NFT sessions as in the SMR NFT sessions.

Results

The QEEG taken in ECR condition at the end of the 31 NFT sessions was compared with that taken before NFT. The ECR QEEG showed a rather general reduction in amplitude, peak-to-peak, across the 19 leads. The percentage of amplitude change in each of the frequency bands at each location can be seen

Figure 1
Percentage of decrease in QEEG power, peak to peak, during ECR condition at 19 locations for each frequency band after neurofeedback therapy.



in Figure 1. The percentage of change can be seen as greater in the alpha band at O1 followed by P3 and Pz, respectively. The largest percentage of decrease in the beta band was at T4 and O2 followed by O1 and T3. The greatest percentage of change in the theta band was at T6 followed by T4 and T5, respectively. The reader can notice the changes recorded were all in the direction of a percentage decrease in amplitude with the only exception at F7 where a just noticeable percentage of increase is recorded in the beta frequency. It is possible, in spite of careful editing of the QEEG data, that some artifact contamination is still present in this data. The comparative percent amplitude changes at each location needs to be interpreted with caution.

Measures of a trend between the pre- and post-QEEG can confirm or disconfirm whether

the changes between pre and post measures are or are not due to chance alone. Figure 2 shows the closest fit straight line to the average microvolts peak-to-peak for each frequency band across 12 SMR NFT sessions. The closest fit straight line in the delta frequency band shows a positive slope suggesting a gradual increment in delta amplitude across the 12 SMR NFT sessions. This increase in delta across training sessions may well be artifact contamination. There is no ready explanation for it since the clinical procedure was consistent, especially with regard to cable movement and impedance levels. The patient appeared to become increasingly quiet throughout the training. Editing of the QEEG as practiced by this writer is as consistent as possible. The relatively flat trend lines of the other frequencies suggest no progressive change, positive or neg-

Figure 2
Closest fit straight line to the average microvolts for each frequency band across 12 SMR NFT sessions.

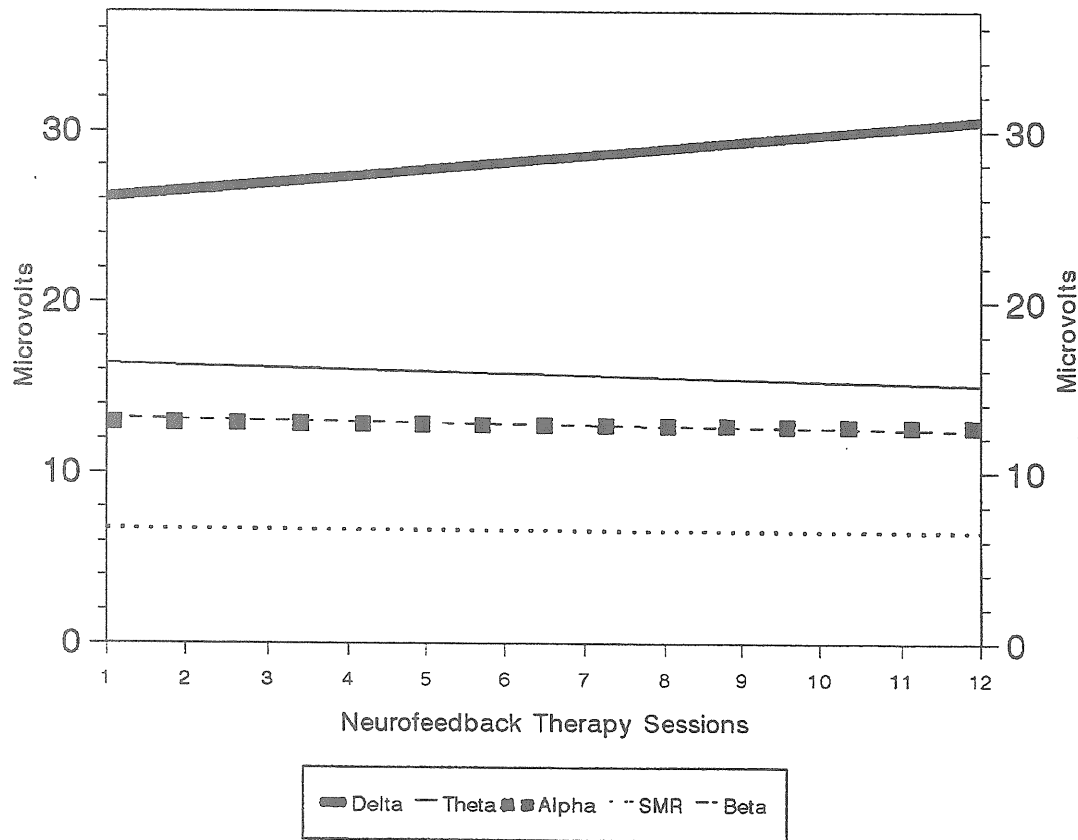
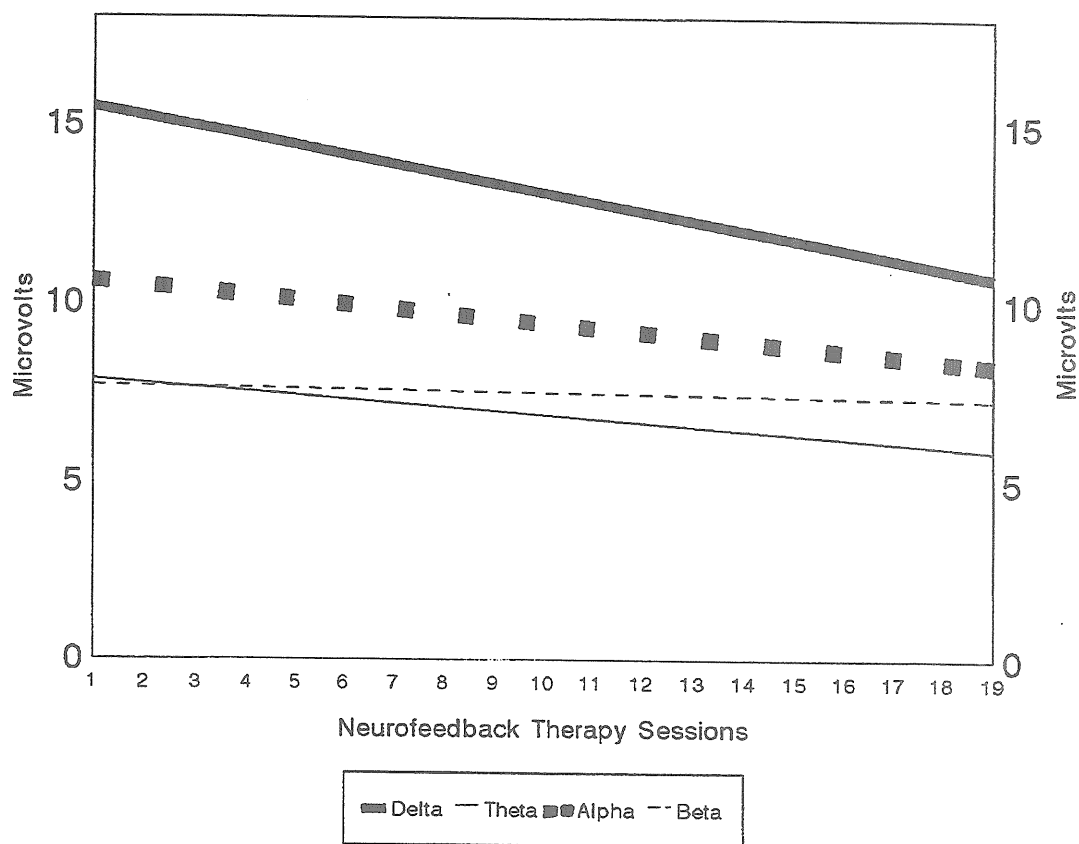


Figure 3
Closest fit straight line to the average micorvolts for each frequency band across 19 beta NFT sessions



ative, across the 12 sessions as measured at the Cz location.

The closest fit straight line to the average microvolts peak-to-peak for each frequency band across 19 beta NFT sessions is shown in Figure 3. Figure 3 shows that each of the monitored frequency bands has a negative slope. A negative slope to the trend line suggests a progressively declining average power for each frequency band across the 19 beta NFT sessions as measured at the T3-C3 location. The reader can notice the comparatively small degree of negative slope to the trend line for the beta frequency band compared to the relatively greater degree of slope to the trend line for delta, theta, and alpha frequency bands. This trend line supports the argument that the post QEEG changes are not due to chance alone.

The neuropsychiatric clinical evaluation, before NFT on October 15, 1993, nearly six years after the injury, confirmed the diagnosis of MHI, as previously stated. Subsequent to the completion of NFT, the same neuropsychiatrist conducted a second relatively briefer clinical evaluation on November 1, 1994. The neuropsychiatrist concluded from his experience as a national authority and author in the field of head injury: "The gains that she made were quite substantial and exceeded those occurring as a result of using a set of lists and other reminders" (A. C. Roberts, personal communication, November 1, 1994).

The same neuropsychologist conducted both the first (February 2, 1994) pre-treatment and, six months later (August 25, 1994), the second post-treatment neuropsychological

Table 1
Neuropsychological Test Results Before and After Neurofeedback Therapy

Name of Test	Test Scores	
	Before NFT	After NFT
1. Booklet Category Test	91 errors	83 errors ^a
2. Verbal Fluency Test	38 productions	45 productions ^a
3. Behavioral Dyscontrol Scale	13	15 ^a
4. Brown-Peterson Test		
10 second delay	13 correct	16 correct ^a
20 second delay	13 correct	27 correct ^a
5. Luria Wood Learning Test		
First trial	7 words	7 words
Number of trials to learn:		
All of 10 words	8	9 (did not learn all 10)
30 minute delay	5	3
6. Beck Depression Inventory	13	12
7. Trail Making Test Part B	66 secs.	71 secs.
8. Wisconsin Card Sort Test		
Categories completed	4	6 ^a
Correct responses	71	69
Errors	57	13 ^a
Perseverative Errors	26	6 ^a
Nonperseverative Errors	31	7 ^a
Percent Perseverative Errors	20.3	7.3 ^a
Failure to maintain set	1	1
9. Shipley Institute of Living Scale		
Vocabulary T Score	55	62 ^a
Abstract	52	60 ^a
Conceptual Quotient	70	84 ^a
Estimated WAIS-R 1.0	100	110 ^a

^a Indicates improvement.

evaluation. In the second testing he used only the instruments in which the patient had performed poorly in the first testing. His findings are summarized in Table 1. He concluded his second testing with:

Re-examination with the Shipley Institute of Living Scale revealed a significant positive change in the patient's ability to utilize fluid intellectual capacities. Her Part II score on this measure was significantly higher in the re-examination, a finding which increased her

conceptual quotient score, a measure of dissociation between crystallized and fluid intellectual capacity. This dissociation is thought to be a sensitive indicator of the presence of organic brain syndrome, as crystallized skills are insensitive to the effects of acquired brain injury, while fluid intellectual skills are often profoundly affected.

Working memory performances revealed that the patient is better able to hold information in memory while performing other men-

tal operations. Her 10-second delayed recall performance on the Brown-Peterson Test represents a significant improvement, although at a 20-second delay her performance continues to be impaired.

Although her performance on the Wisconsin Card Sorting Test was likely to have been influenced by practice effect, the magnitude of her improvement would suggest an increase in her cognitive flexibility and problem-solving ability. A slight improvement in performance on the Booklet Category Test supports this. Further evidence of improvement in executive functioning was seen in her performance on the Behavioral Dyscontrol Scale (from a score of 13 to a score of 15). Patient's verbal fluency has improved, as has

her insight into her own situation. There was evidence in re-examination that she was better able to recognize and spontaneously correct errors.

Evidence for improvement in declarative memory function was not observed. Patient did not improve in her performance on the Luria Word Learning Test, a ten unrelated word list. She was not able to learn the list in nine trials.

There has been no significant change in patient's mild depression since the initial examination. Her depression is not so severe as to cause disruption in cognitive functioning, in my opinion.

The patient's report of her own function-

Figure 4
Symptom ratings before and after neurofeedback therapy according to the scale: none, 0; minimal, 2; mild, 4; moderate, 6; severe, 8; very severe, 10.

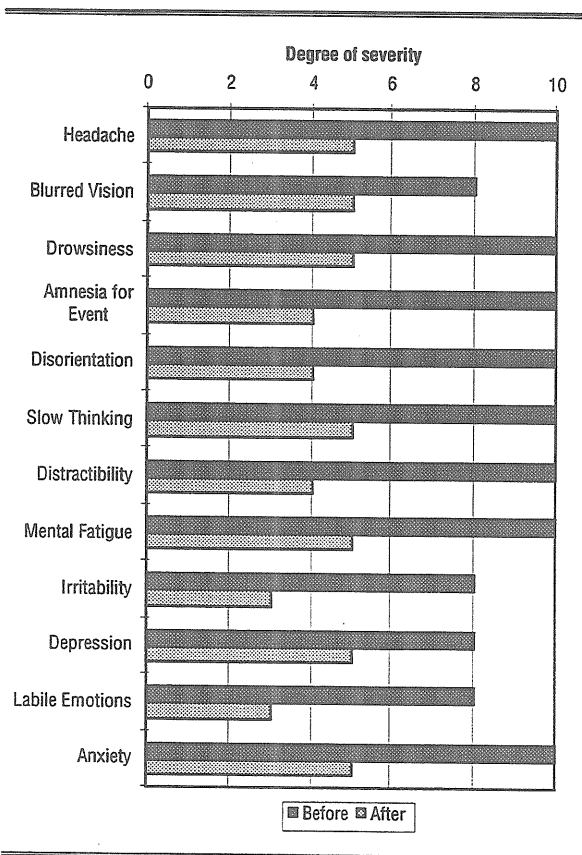
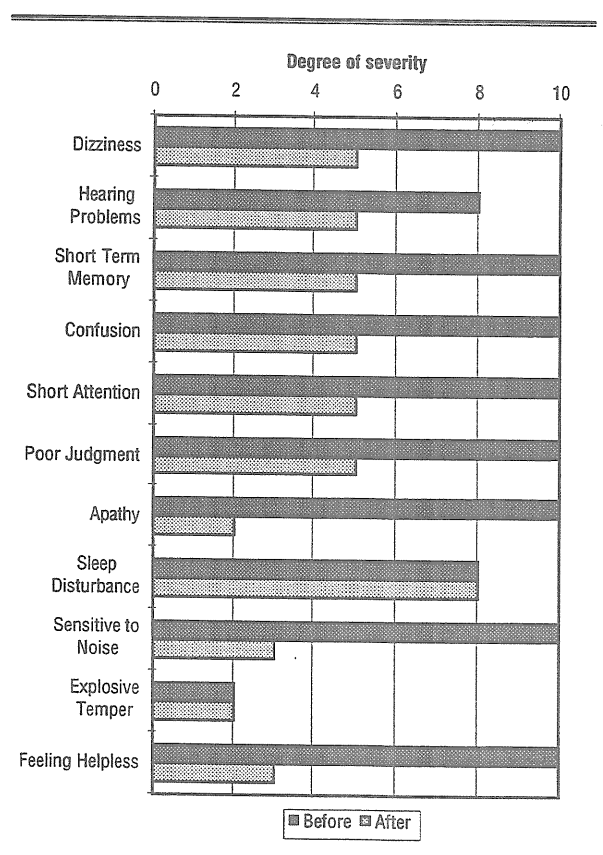


Figure 4a
Symptom ratings before and after neurofeedback therapy according to the scale: none, 0; minimal, 2; mild, 4; moderate, 6; severe, 8; very severe, 10.



ing suggests that improvements in working memory function, coupled with increased cognitive flexibility, are responsible for positive changes in her ability to conduct her everyday life in an effective manner (R. A. Kookan, personal communication, December 6, 1994).

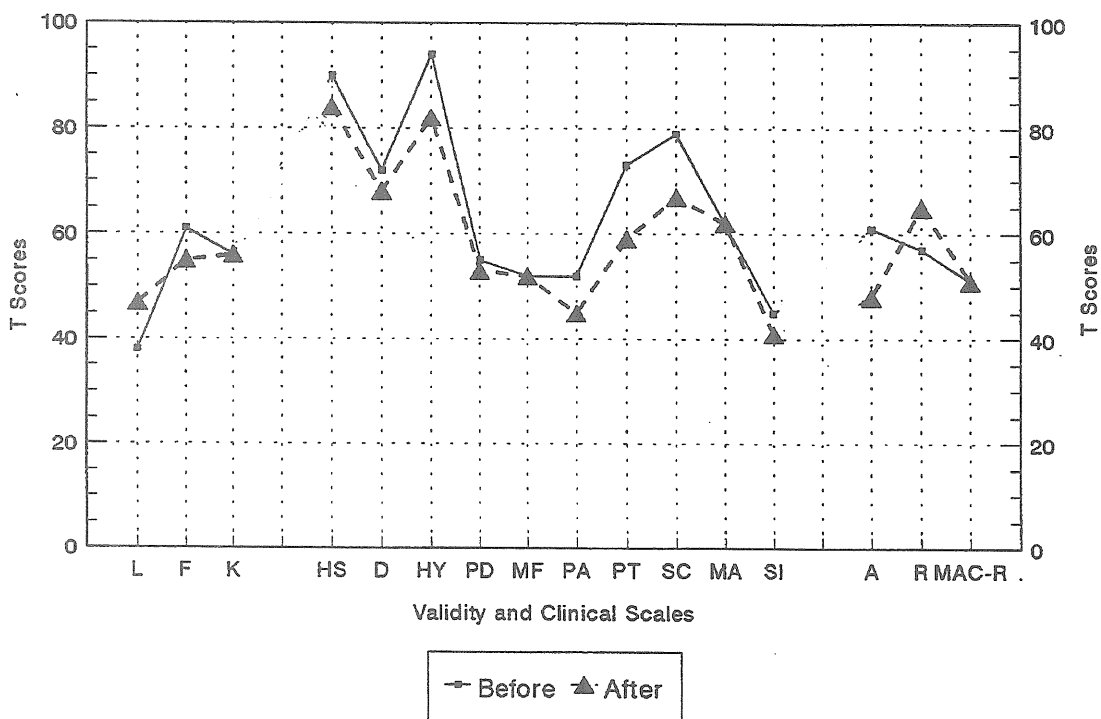
The patient was seen daily. Chart notes recorded patient improvement beginning with the 3rd SMR NFT session in which she indicated her memory was better. In the 6th NFT session, she reported improved memory and that the "fog" was lifting. In the 9th NFT session, she claimed her "taste" was more accurate, especially for strawberries, and she was less prone to disorientation for directions. In the 10th NFT session, she stated she was less depressed and reading was improving. In the 11th NFT session, she indicated she still forgets some things like leaving the sprinkler on. In the 12th NFT session, she reported looking forward to getting up in the morning to see what

additional signs of recovery she will notice. Before NFT she stated she was reluctant to get up in the morning for fear of what "stupid thing she would do today." At the 13th NFT session (the first beta training session), she reported visiting a friend in the hospital, leaving the patient's room, going to the hospital cafeteria and returning to the patient's room without getting lost. Additionally, for the first time in six years, she could play cards with her grandchildren. In the 14th NFT session, she reported what might be labeled a relapse. She thought it was due to stress. The fog and fatigue had momentarily returned. In the 16th NFT session, she reported she no longer felt like a rat in a maze that can't find its way out. In the 19th NFT session, she reported dreaming of forgotten childhood traumatic memories and of deceased loved ones. This was resolved through one psychotherapy session. She still feels as if the car is moving after it comes to a stop. She also reported that she

Figure 5

Minnesota Multiphasic Personality Inventory-2, T-scores before and after Neurofeedback therapy.

Source: Minnesota Multiphasic Personality Inventory-2. Copyright by the Regents of the University of Minnesota 1942, 1943 (renewed 1970), 1989. This profile form 1989. All rights reserved. "MMPI-2" and "Minnesota Multiphasic Personality Inventory-2" are trademarks owned by the University of Minnesota.



had read a two-inch thick English translation of a Russian novel in three days and enjoyed it.

The patient completed a symptom checklist for MHI both before and after NFT. Inspection of Figures 4 and 4a reveals subjectively significant changes in her reported symptoms. She still reports sleep disturbance as a very severe symptom. Visual inspection reveals at least a 50% or more improvement in all of the other symptoms checked.

The patient responded to the MMPI-2 before and after treatment in such a way as to earn two relatively similar profiles. Figure 5 indicates both profiles are similar to what this writer frequently sees among chronic pain patients. This patient is still in therapy for her orthopedic injuries. The patient's scores on Hs, Hy, A, Pt and Sc scales improved. Indeed, clinically, her level and quality of personality functioning were somewhat improved with respect to anxiety and coping better with reality. In any case, her overall quality of personality adjustment as measured by the MMPI-2 appears to have remained fairly constant throughout NFT.

Discussion

The hypothesis that a patient presenting the symptoms of a MHI, while at the same time showing excessive slow wave (theta) activity, can be trained to suppress the excessive slow wave activity and enhance the faster frequencies (beta) was not clearly demonstrated. The hypothesis that such training will reduce or eliminate the cognitive deficits suffered as a result of the MHI was demonstrated in that many of the patient's symptoms were reduced during and following the NFT.

Clearly, the patient improved across the treatment period. This improvement is unlikely attributable to chance or the passage of time since the injury was six years prior to treatment. The neuropsychiatrist identified substantial gains not attributable to CST alone. Moreover, the neuropsychologist indicated several measurable positive changes in the second evaluation compared to the first. He also indicated that some cognitive functions did not change.

The patient's self-reported improvement was clearly more than this writer would have expected from the concurrent psychotherapy and CST. The recovery reported here is similar to that reported by Ayers (1987, 1991).

The overall reduction of EEG amplitude reported across all frequencies was most pronounced in the alpha band in the post-treatment QEEG compared to the pre-treatment QEEG. Indeed, reduced artifact contamination may have affected these reductions. It was expected that the theta band would have exhibited the greatest diminution.

Exactly what factors contributed to the positive outcome reported cannot be proven from this single case study. There is no doubt that the neurofeedback therapist's positive attitude was a motivational factor. The patient's cognitive symptoms had been misunderstood for years. Certainly, she was gratified that someone could finally explain the cause for her symptoms without her being considered crazy. However, in view of unremarkable outcomes with standard psychotherapeutic approaches to a MHI, it is unlikely the patient and therapist attitudes (placebo) in this case can account for the outcomes.

Is it possible that the changes in this patient's QEEG recordings and the concurrent reduction of symptoms could have been achieved by 31 sessions of other self-regulation training such as digital temperature control, electromyograph reductions, or control of electrodermal response? This writer does not know of any studies comparing the relative efficacy of NFT compared to any other protocol for biofeedback-assisted self-regulation therapy for MHI for 31 or more sessions.

The effect of Prozac, though perhaps clinically important, may have influenced the pre-treatment QEEG to only a mild degree. Lucas (1992) reported from the work of Saletu, B. and Grunberger, J. (1985) Fluoxetine dosages of 30, 60, and 75 mg in normal volunteers showed mild EEG changes as compared to placebo:

“. . . the most prominent being an increase in alpha activity and decreased fast beta activi-

ty. Such findings are suggestive of an improvement in vigilance, although, compared to classical tricyclic antidepressants, the lack of sedative property may be more related to a lack of anticholinergic activity."

Tarn, M., Edwards, J. G. and Sedgwick, E. M. (1993) reported similar findings in their study of Fluoxetine and Amitriptyline. A single 60 mg evening dose of Fluoxetine taken for four weeks failed to result in significant changes in amplitude or frequency across all frequency bands.

These studies suggest, on the one hand, the alpha amplitude reduction reported here (Figure 1) might have been slightly more than if treatment had started from a baseline without Prozac. Moreover, the reduction in amplitude of beta reported here (Figure 1) might have been even less than a pre-treatment baseline without Prozac. On the other hand, since she continued to use her 20 mg dosage of Prozac on an as-needed basis throughout the treatment period, the effects of Prozac may well have been scattered across treatment sessions.

The E-BV infection complicated the diagnosis. When the family physician was provided with the additional reports, as summarized above, he reconsidered his diagnosis of E-BV infection as the underlying cause of her cognitive deficits. Without the benefit of the latter reports he considered the diagnosis of E-BV infection to be a "...prudently-derived cause of her symptoms." In his revised diagnosis he also stated, "...a high percentage of the normal population have elevated antibodies to the E-BV and do not have Chronic Fatigue Syndrome." He concluded with: "Retrospectively, and with the more current understanding of the significance of the traumatic closed-head injuries and the long-term sequelae, it makes most sense that the patient's symptoms were secondary to the injury" (M. T. Rendler, personal communication, June 17, 1994). This conclusion lends support to the contention that it was a MHI that caused the cognitive deficits, which were treated with NFT in this patient, rather than the E-BV infection. Still, it is also possible that both the MHI and the E-BV were treated.

There is strong evidence that the measured EEG changes were not due to chance alone. In support of this is the closest fit straight line across the 19 beta NFT sessions for all frequencies (Figure 3). This is evidence of a progressively reduced amplitude in QEEG measures across NFT sessions. This is not likely to be due to chance alone. Moreover, the degree of negative slope for delta, alpha, and theta frequency bands is more obvious than the degree of negative slope for beta. The training was focused on suppression of the slower band waves. This is consistent with the first hypothesis. Finally, it can be assumed that EEG changes took place concurrently with the patient's reported progressive symptom relief beginning with the 3rd NFT session.

The lack of reduced amplitudes recorded across the SMR NFT sessions (Figure 2) may suggest that the SMR sessions made little or no contribution to the treatment outcome. Clinical improvement, however, was being reported by the patient during the period of time in which the patient was being treated with the SMR NFT protocol. This reported progressive change may indicate that EEG changes were actually taking place during the SMR training but were not being recorded at the Cz location used in the SMR NFT. Differential changes in EEG amplitude have been shown to take place in areas of the brain not concurrently recorded at the location chosen for NFT (Byers, 1992, and Fahrion, Walters, Coyne, and Allen, 1992).

The MMPI-2 profile relative consistency from pre- to post-treatment assessment supports the argument that the MMPI-2 is not designed to measure neuropsychological functioning. The relative stability of the patient's personality functioning across the treatment time adds further support to the argument that the NFT offered was focused on MHI symptoms.

Taken together, the data reported herein suggests that the QEEG changes recorded are at least closely related to the patient's concurrently reported symptom relief and that the NFT was very likely the major contributing factor to both QEEG changes and concurrent

symptom relief. Moreover, the QEEG amplitude changes from pre-treatment baseline to post-treatment baseline, together with the reported improvement, suggest that the QEEG record of this patient was normalized.

Further research needs to be done since one cannot generalize from this single case study. It may be that the SMR training was not needed and treatment could have started with the beta training protocol or the SMR training was prematurely terminated. Perhaps continued NFT training would have resulted in significantly reduced theta to beta ratios. Perhaps there are protocols that are specific for a given type of MHI. Additional single case studies of NFT can identify new research issues, but cannot settle the issue of the efficacy of this NFT protocol for the larger population of MHI patients. Studies are needed using NFT with a large number of experimental and control subjects. Such studies need to control for sex, age, education, ethnicity, EEG frequency bands used for training, scalp locations, whether eyes need to be open or closed during NFT, and the neurological history, including the etiology of the injury under study. The type of equipment used will also need to be considered. The FDA standards may help to equalize the effects of equipment on outcomes.

This study could be refined by selecting a patient not on medication and not suffering any other medical problems such as E-BV. Furthermore, as already indicated, SMR and beta feedback training sessions might be extended. SMR eyes-open may also make a difference.

How soon after an MHI should NFT be offered to the patient? Some might say NFT should not be tried until after a reasonable period of time has elapsed to allow for spontaneous remission to take place by natural history. At this time we have no controlled studies to indicate when we should routinely offer NFT to the MHI patients. In the interest of reducing patient suffering and economic loss, it may be desirable to conduct research to discover the earliest time after injury to offer NFT. Such knowledge might help to avoid the secondary gains that can occur by a delayed recov-

ery. It might be of interest to pursue NFT for athletes who routinely suffer repeated MHI, such as boxers, football players, hockey players, and baseball players.

References

- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- Ayers, M. E. (1987, December), *Electro-encephalic neurofeedback and closed head injury of 250 individuals*. A paper presented at the National Head Injury Foundation Annual Conference.
- Ayers, M. E. (1991). *A controlled study of EEG neurofeedback training and clinical psychotherapy for right hemisphere closed head injury*. A paper presented at the National Head Injury Foundation, Los Angeles, California.
- Butcher, J. N. (1989). *Minnesota Multiphasic Personality Inventory-2, User's guide, the Minnesota report: Adult clinical system*. Minneapolis: National Computer Systems.
- Butcher, J. N., Dahlstrom, W. G., Graham, J. R., Tellegen, A., & Kaemmer, B. (1989). *Minnesota Multiphasic Personality Inventory-2 (MMPI-2), Manual for administration and scoring*. Minneapolis: University of Minnesota Press.
- Byers, A. P. (1992). The normalization of a personality through neurofeedback therapy. *Subtle Energies*, 3, 1, 1-17.
- Duffy, F. H., Iyer, V. G., and Surwillo, W. W. (1989). *Clinical electroencephalography and topographic brain mapping: Technology and practice*, Springer-Verlag, New York, Berlin.
- Fahrion, S. L., Walters, D. E., Coyne, L., Allen, T. (1992). Alterations in EEG amplitude, personality factors and brain electrical mapping after alpha theta brain wave training: A controlled case study of an alcoholic in recovery. *Alcoholism: Clinical and Experimental Research*, 16, 547-552.
- Green, A. M., & Green, E. E. (1975). Biofeedback: Research and therapy. In Jacobson, N. O., Ed. *New ways to health*.

Natur Och Kultur, Stockholm.

- Jasper, H. H. (1958). The ten-twenty electrode system of the International Federation. *Electroencephalography and Clinical Neurophysiology*, *10*, 371-375.
- Kwentus, J. A., Hart, R. P., Peck, E. T., & Kronstein, S. (1985). Psychiatric complications of closed head trauma. *Psychosomatics*, *26*, 8-15.
- Levin, H. S., Mattis, S., Raff, R. M., Eisenberg, H. M., Marshall, L. F., Tabaddor, K., High, W. M., and Frankowski, R. F. (1987). Neurobehavioral outcome following minor head injury: A three-center study. *Journal of Neurology and Psychiatry*, *66*, 234-243.
- Lexicor Medical Technology (1990). *Neurosearch-24 User's Manual*, Boulder, Colorado.
- Lezak, M. D. (1995). *Neuropsychological Assessment* (3rd ed.). New York, Oxford University Press.
- Lubar, J. F. (1991). Discourse on the development of EEG diagnostics and biofeedback for attention-deficit/hyperactivity disorders. *Biofeedback and Self-Regulation*, *16*, 3, 201-225.
- Lubar, J. F., & Bahler, W. W. (1976). Biofeedback management of epileptic seizures following EEG biofeedback training of the sensorimotor rhythm. *Biofeedback and Self-Regulation*, *1*, 1, 77-104.
- Lucas, R. A. (1992). The human pharmacology of fluoxetine. *International Journal of Obesity*, *16*, (suppl.), S49-S54.
- Rimel, R., Giodani, B., Barth, J., Boil, T., & Jane, J. (1981). Disability caused by minor head injury. *Neurosurgery*, *9*, 221-223.
- Saletu, B. & Grunberger, J. (1985). Classification and determination of cerebral bioavailability of fluoxetine: pharmacokinetic, pharmaco-EEG, and psychometric analyses. *Journal of Clinical Psychiatry*, *46*, 45-52.
- Sterman, M. B., & Friar, L. (1972). Suppression of seizures in an epileptic following sensorimotor EEG feedback training. *Electroencephalography and Clinical Neurophysiology*, *33*, 89-95.
- Sterman, M. B., McDonald, L. R., & Stone, R. K. (1974). Biofeedback training of the sensorimotor electroencephalographic rhythm in man: Effects on epilepsy. *Epilepsia*, *15*, 395-416.
- Tansey, M. A. (1984). EEG sensorimotor rhythm biofeedback training: Some effects on the neurological precursors of learning disabilities. *International Journal of Psychophysiology*, *1*, 163-177.
- Tansey, M. A., (1985a). The response of a case of petit mal epilepsy to EEG sensorimotor rhythm biofeedback training. *International Journal of Psychophysiology*, *3*, 81-84.
- Tansey, M. A. (1985b). Brain wave signatures: An index reflective of the brain's functional neuroanatomy: Further findings on the effect of EEG sensorimotor rhythm biofeedback training on the neurological precursors of learning disabilities. *International Journal of Psychophysiology*, *3*, 85-95.
- Tansey, M. A. (1990). Righting the rhythms of reason: EEG biofeedback training as a therapeutic modality in a clinical office setting. *Medical Psychotherapy*, *3*, 57-68.
- Tarn, M., Edwards, J. G. & Sedgwick, E. M. (1993). Fluoxetine, amitriptyline and the electroencephalogram. *Journal of Affective Disorders*, *29*, 7-10.
- Thatcher, R. W., Walker, R. A., Gerson, I., & Geisler, F. H. (1989). EEG discriminant analyses of mild head trauma. *Electroencephalography and Clinical Neurophysiology*, *73*, 94-106.
- Uzzel, B. P., Langfitt, T. W., and Dolinskas, C. A. (1987). Influence of injury severity on quality of survival after head injury. *Surgical Neurology*, *27*, 419-429.