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EEG Biofeedback in the Treatment of Lyme Disease:

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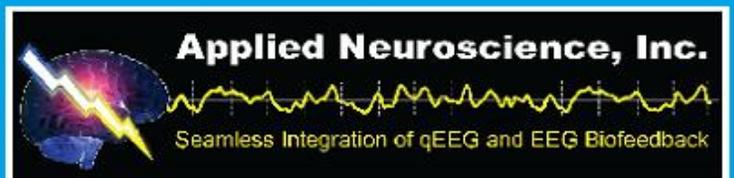
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EEG Biofeedback in the Treatment of Lyme Disease: A Case Study

Russell C. Packard, M.D. and Lesley P. Ham, M.A.

EEG biofeedback (EBF) was evaluated in a 44-year old male with advanced Lyme disease and associated cognitive dysfunction. Treatment consisted of 40 sessions of EBF designed to suppress theta and enhance beta. Following treatment, the patient reported a 50% improvement overall in cognitive functioning. A modest improvement in theta, the theta/beta ratio, and the P3 evoked potential were also observed. Results indicate a possible role for EBF in advanced stage Lyme disease.

Introduction

EEG biofeedback therapy (EBF) or neurofeedback has been effective in treating a variety of disorders. The areas receiving the most research include attention deficit disorder (Lubar, 1991; Mann, Lubar, Zimmerman, Miller, & Muenchen, 1991), epilepsy (Lubar & Bahler, 1976; Sterman, McDonald, & Stone, 1974), learning disabilities (Lubar, Gross, Shively, & Mann, 1990; Tansey, 1985), head injury (Ayers, 1987, 1991; Byers, 1995; Hoffman, Stockdale, Hicks, & Schwaninger, 1995), and alcohol/drug addictions (Penniston, 1989; Walters, 1994). Recently, other clinicians have begun investigating the usefulness of EBF in a variety of conditions associated with cognitive dysfunction, such as Alzheimer's disease (Budzynski, personal communication), dementia (Budzynski, personal communication), and stroke (Ayers, 1995). In light of these initial positive findings, we decided to treat a patient with Lyme disease and associated cognitive dysfunction with EBF. The following case report thoroughly describes the patient's symptoms, cognitive assessments, the EBF procedure, and objective and subjective results.

Lyme Disease

Lyme Disease (LD) or Lyme Arthritis is the most common tick borne illness in the United States (Ciesielski et al., 1988). Specifically, LD is an inflammatory disorder often

recognized by early skin lesions (Berkow, 1987). Initial symptoms may be followed weeks to months later by neurologic cardiac and/or joint abnormalities (Malawista & Steere, 1986). For some patients, LD is inconsequential; for others it is disabling (Barbour, 1989). The first phase, generally lasting a few weeks, involves a skin lesion (beginning as a red macule) that often expands into several smaller lesions (Berkow, 1987). Common symptoms associated with this phase include fatigue, chills and fever, headache, stiff neck, backache, and sore throat (Steere, Bartenhagen, & Craft, 1983a). In most cases, symptoms are intermittent and changing.

The latter phases consist of neurologic, cardiac, and arthritic abnormalities. Neurologic difficulties develop in approximately 15% of patients within weeks to months of the initial phase (Berkow, 1987). Symptoms may include meningitis, encephalitis, cerebellar ataxia, cranial neuritis, chorea, and/or motor and sensory radiculoneuritis, but they usually resolve (Malawista & Steere, 1986; Steere, Pachner, & Malawista, 1983b). Myocardial abnormalities, generally consisting of atrioventricular block, occur in about 8% (Barbour, 1989). Arthritis develops in about half of patients within weeks to months of onset of LD (Berkow, 1987). Intermittent swelling and pain in several joints, primarily the knees, may recur for several years (Barbour, 1989). Some

patients develop unremitting joint involvement and neurologic abnormalities (Kohler, 1986). Later stage neurologic complications include neuropsychiatric disease, central nervous system disease, and fatigue syndromes (Broderick, 1987; Kohler, 1986).

The treatment of choice for LD, particularly in its early stages, is antibiotic therapy. Antibiotics, however, may fail in patients with chronic LD (Steer et al., 1983b). For symptomatic relief, anti-inflammatory medications are often used. Unfortunately, for later stage neurologic problems, minimal treatments are available.

Cognitive Assessments

Brain mapping may provide an effective means of evaluating cognitive changes, particularly if MRI and CT scans are normal. EEG brain mapping is a digital technique resulting in color-coded maps of EEG activity, which may aid in identifying subtle areas of regional slowing that have missed identification by traditional EEG assessment (Meador, 1992). It consists of several quantitative EEG techniques, including EEG frequency analysis, topographic display, statistical comparisons to a normative database, and other similar computer-based calculations based on EEG or evoked potentials.

Evoked potentials, electrical fields produced by a regular pattern of neural activity in response to an external stimulus (Meador, 1992), are typically mapped by visual or auditory methods. Visual EPs can be produced by a change in flash (such as a strobe lamp), a change in pattern (such as rotating a checkerboard, bar graphs, or stripes), or both (Epstein, 1992). Auditory EPs, elicited by acoustic stimulation, produce up to 30 components (Goodin, 1992). We have become particularly interested in the event-related or long-latency potentials (N1, P1, N2, P2, P3), most notably the endogenous P3 component.

P3 is a positive wave produced about 300 to 400 msec after attending to an infrequent (unexpected) task relevant stimulus (Goodin, 1992). Some of its primary corre-

lates include cognitive functions such as memory, information delivery, and decision making (Pratap-Chand, Sinniah, & Salem, 1988). Analysis of P3 involves examination of latency, amplitude, and topography. Latency refers to the time, usually measured in milliseconds, between the onset of the stimulus and any particular point of the wave form (significantly increased latencies indicate abnormality); amplitude refers to the distance from the peak of one wave form component to the trough related to that component (low amplitude measures suggest abnormality); and topography refers to the image of a wave form component (the image should, for the most part, be symmetrical). Research indicates that P3 is responsive to cognitive changes associated with dementia (Polich, 1991), schizophrenia (Pritchard, 1986), and traumatic brain injury (Olbrich, Nau, & Zerbin, 1986; Papanicolaou, Levin, & Eisenberg, 1984; Pratap-Chand et al., 1988).

In light of these findings, we became interested in evaluating cognitive changes in mild traumatic brain injury (TBI) with P3 analysis. In a recent study, 50 patients sustaining mild TBI and reporting difficulties with concentration and/or memory, and 20 age-matched controls were evaluated with P3 (Packard & Ham, 1994-95). Results indicated that 68% had some type of abnormality in one or more areas (latency, amplitude, topography). Following this, we recently have begun investigating whether the successful treatment of EBF in many of these patients will result in improved P3 (Packard & Ham, in progress). Similarly, for the present case study, we decided to examine the effects of EBF, if successful, on P3.

Method

Subject

The patient, HG, is a married, Caucasian male, age 44, with two adolescent sons. He has a college degree in electronics and was employed with a large company as a computer specialist. He first presented for treatment in October of 1986

complaining of tremor in the left arm associated with a "strange feeling" and tingling sensation. In the two months prior to initial evaluation, the sensation was accompanied by heaviness and pain. In addition, he reported a two month history of severe headaches. All assessments (EEG, EMG nerve conduction, MRI, CT, and visual and brainstem evoked responses) were normal. To treat headaches and tremor, Inderal-LA was prescribed with some reported benefit.

HG returned in February of 1987, complaining of intermittent numbness in the right arm, difficulty concentrating, and difficulty getting words out. He also noticed some forgetfulness and difficulties with depth perception. Repeat visual evoked responses, MRI, and EEG were normal. A neuropsychological battery (WAIS-R, Wide Range Achievement Test-R, Bender Visual-Motor Gestalt Test, Weschler Memory Scale, and Halstead-Reitan Neuropsychologic Test Battery) was conducted the following month. All scores fell within normal ranges, with the exception of the Digit Symbol subtest on the WAIS-R (measure of visual memory and visual-motor integration) and the spelling subtest on the ERAT-R (the patient stated he was always a poor speller). Overall, the neuropsychologist found no evidence of diminished cognitive functioning.

Difficulty with symptoms continued intermittently with no sure indication of diagnosis until August of 1988. At this time, elevated antibody levels to Lyme disease antigens were found. Correspondingly, the patient remembered receiving a tick bite in 1985. In June of 1989, his condition began to deteriorate, with noticeable decreases in energy level, diminished hearing, and gradual worsening of headaches, joint pain, and cognitive difficulties. He began having continuous headaches and joint pain, significantly aggravated by humidity. In addition, HG noticed feelings of discouragement and depression, with his family observing episodes of emotional lability. The days he was able to work progressively decreased (in August of 1990 he worked seven days in

three months). In January of 1991, he was medically retired from the company for which he had worked for 16 years. Increasing cognitive difficulties were observed, such as getting lost in his neighborhood. Not surprisingly, cognitive evoked potentials conducted in 1991 revealed a delayed P3. There were also difficulties with swallowing and esophageal spasms. Finally, HG was declared 100% disabled per Social Security. He began to require almost continuous antibiotics, but these occasionally had to be discontinued due to fungal infections. Arthritic symptoms, headache, and cognitive functions would all become dramatically worse when he was off antibiotics.

By March of 1994, it appeared that physical and emotional symptoms had reached a plateau. Cognitive symptoms, however, seemed to be more of a problem. An increase in short-term memory problems and increased difficulty processing information were reported. Similarly, EEG and quantitative EEG revealed a mild diffuse disturbance of cerebral activity. Cognitive evoked potentials showed a delayed P3 similar to a 1991 study. Visual evoked responses were slightly prolonged. At this point, we decided to try EBF for the patient's cognitive difficulties. Although we knew of no research on EBF for LD, we decided to try this therapy for two reasons: 1) the patient's cognitive problems were fairly similar to cognitive difficulties following head injury, and EBF has been used effectively in many cases to treat head injury (Ayers, 1987, 1991; Byers, 1995; Hoffman et al., 1995), and 2) we felt that the patient was showing slow but progressive declines in cognitive functioning, and some type of therapy needed to be initiated. In short, EBF seemed to be the best option for improving this patient's situation.

Procedure

Therapy was begun in March of 1995 after an initial session to discuss EBF and give the patient a demonstration. See Table 1 for a chart of symptoms reported by the patient throughout the course of treatment. Sessions lasted approximately 75 minutes,

Table 1
Symptoms Endorsed by HG as Problematic

Date:	2/21	4/4	4/25	6/6	7/7	8/16	9/11
Problems Organizing	3	2	2	1	1	0	0
Headache	3	3	3	3	3	1	1
Blurred Vision	3	3	3	3	3	3	3
Disorientation	3	3	3	1	1	0	0
Slow Thinking/Processing	3	3	3	3	3	3	3
Labile Emotions	3	2	2	1	2	0	1
Tinnitus	3	3	3	3	3	3	3
Short-term Memory Difficulties	3	3	2	3	2	2	2
Confusion	3	2	1	1	1	0	0
Shortened Attention Span	3	2	2	3	1	2	0
Sleep Disturbance	3	3	3	3	3	3	3
Sensitivity to Noise and Light	3	3	3	3	3	3	3
Difficulty Concentrating	3	3	2	1	1	0	0
Trouble Finding Words/ Word Reversal	3	2	3	1	1	2	0
Forgetfulness	3	3	3	3	2	1	1

with 30-45 minutes of training time and the other 30-45 minutes for hook-up, discussing symptoms and training results, and providing suggestions for using techniques in daily life. The patient received a total of 40 sessions, twice weekly. Near the end of therapy, sessions were gradually phased out. Following session 40, a follow-up brain mapping study was conducted to assess changes in the P3 component.

Training primarily consisted of learning to increase beta and decrease theta. We decided to use shaping techniques to achieve the ideal response. For the first 18 sessions, the patient was trained to increase a wide beta band (13-20 Hz) and decrease 4 to 7 Hz theta (remained constant). For sessions 19 through 24, he was trained to produce beta at 15 to 20 Hz. For the final ses-

sions (25 through 40), beta enhancement was set at 15-18 Hz. The Cz placement was used for sessions 1 through 22. This was changed to Fz following session 22 in an attempt to achieve more significant changes. Cz and Fz were selected because these were the areas revealing the greatest amount of slowing on quantitative EEG.

During sessions, a variety of techniques were used to enhance beta and inhibit theta. These techniques consisted of visual (watching the theta or beta bar graph), auditory (listening to tones associated with enhanced beta), or "enhanced thinking" exercises (mental math, reading, etc.). The pre-set beta frequency band threshold for enhancing 15- to 18-Hz beta was accompanied by a reinforcing audio signal; if theta exceeded a pre-set level, however, the signal immedi-

ately stopped. In all exercises, the patient was instructed to learn the mental scheme or strategy that would keep the bar above the threshold setting while maintaining audio signal.

Apparatus

Baseline and follow-up brain mapping studies were conducted on a Bio-Logic Brain Atlas III using the International 10-20 system of electrode placement. The patient was in a relaxed, awake state, and the study was recorded approximately 10 minutes with eyes open and closed. After being analyzed by visual inspection, the recording was then digitized, edited to reduce artifact by the neurologist/psychiatrist (first author), segmented, and subjected to quantitative analysis. Thirty seconds of artifact free EEG were stored as fast Fourier transforms of power (amplitude). This was displayed in color graduated topographic maps. Impedance was judged acceptable when below 2.5 k ohms. The qEEG bands were: delta—0 to 4 Hz, theta—4 to 8 Hz, alpha—8 to 13 Hz, beta—13 to 20 Hz.

The extended cognitive evoked potential study was performed utilizing an "oddball paradigm." In this paradigm, the patient is instructed to count infrequent, rare tones in a series of frequent tones. Response to rare tones typically shows a positive wave form approximately 300 milliseconds following the stimulus (the P3). Findings are compared to stored data for normal subjects of similar age, and Z-score transformation is carried out to determine any significant statistical difference from normal.

The Biorex EEG computerized 4-channel biofeedback system version 2.0 (Lexicor Medical Technology), was used for all EBF sessions. A monopolar placement at either Cz or Fz was selected (as previously described), with a forehead ground and linked ear reference. Impedance was judged acceptable when below 5 k ohms. Amplifier gain was set at 32,000. Sampling rate was at 128 samples per second per channel. The frequency bands chosen were: delta—0 to 4

Hz, theta—4 to 7 Hz, alpha—8 to 12 Hz, SMR—12 to 15 Hz (last several sessions), beta—15 to 18 Hz (last several sessions), and EMG—25 to 32 Hz.

Results

Subjective

During the first session, HG reported that the symptoms of greatest concern included blanks in memory, "day dreaming" a good part of the time, forgetfulness, difficulty concentrating, difficulty finding words, headache, labile emotions, blurred vision, tinnitus, and sleep disturbance. He became aware of the effect of concentration on beta and theta by session 2. He began to detect some ability to concentrate and control beta/theta by session 5. At session 8, he stated EBF was helping somewhat, but he was unsure exactly how or why. Improvements appeared to wax and wane (better results for a few sessions, followed by a bad day) throughout much of treatment. After 20 sessions (half of therapy), he stated that his primary improvements were increased ability to focus, less disorientation and confusion, less instances of word reversal, less difficulty with labile emotions, and decreased frustration with self and others (namely family members). This was confirmed by the patient's wife, who attended session 22 with him. Around session 30, HG began reporting increased difficulties with headache. He stated that he had sustained a constant headache, at least moderate to severe in intensity, for the past three weeks. Although cognitive functioning was better, he felt the headache was affecting his ability to concentrate at optimal level. At this point, it was decided to give him a trial of Zolofit (in addition to Inderal, which had previously been prescribed) for headache management. (It is noteworthy that HG did not show depressive symptoms such as depressed mood, suicidal ideation, guilt, or sleep disturbances). After beginning Zolofit, he reported decreased difficulty with headaches and even better mental clarity and energy (the patient attributed this to a

decrease in headaches). Overall, he reported cognitive symptoms were 50% better since receiving treatment with EBF.

Objective

As stated, HG did show some improvement, although not linear, throughout treatment (see Figures 1-3 for theta, beta, and ratio values).

Due to beta shaping from 13 to 20 Hz to 15 to 18 Hz (described previously), it was impossible to compare beta values from early sessions to later sessions. Theta, however, remained constant (4 to 7 Hz) throughout treatment and is comparable across sessions. Similarly, for the final sessions (25 through 40) in which beta remained constant, changes in beta and the theta/beta ratio can be evaluated. Since several different values were taken throughout treatment (5 to 6 training exercises were conducted), the amount of theta, beta, and the ratio were averaged to obtain one overall value for each. Results show no significant change for beta. Theta and the theta/beta ratio did show modest improvement from the beginning to the end of treatment. For example, theta ranges from a high of 13.7 μv (sessions 8 and 9) to a low of 8.7 and 8.6 μv (sessions 38 and 40), and the theta/beta ratio (from sessions 25 through 40 where beta is 15 to 18 Hz) ranges from a high of 2.7 (session 26) to a low of 1.9 (session 38). In the sessions near the end of treatment in which theta was uncharacteristically elevated (32, 34, 39), the patient reported either severe headache or joint pain.

The first brain mapping study (5/15/91) revealed a normal EEG and quantitative EEG. Evoked potentials revealed a P3 which was moderately delayed at 352 ms and was of low amplitude (data not available). The second brain mapping study (3/10/94) showed a mild diffuse disturbance of cerebral activity (slowing) with both EEGs. The P3 showed a latency of 361 ms and an amplitude of 3.4 μv over P3 with an irregular, low amplitude topography. The P3 conducted in 9/14/95 (after EBF) revealed a

latency of 350 ms with an amplitude of 4.98 with a fairly symmetric topography. Although this is still somewhat abnormal for the patient's age, it represents an improvement from previous studies.

Discussion

The hypothesis that EEG biofeedback will reduce cognitive deficits associated with LD was partially confirmed. Subjective findings indicated a 50% improvement overall in cognitive function following therapy. Objective findings signify that the patient was able to decrease slow wave activity (theta), but was unable to significantly enhance fast wave activity (beta). This led to only a modest decrease in the theta/beta ratio. It is noteworthy that improvements in theta and the ratio were not linear. There were many episodes in which improvement waxed and waned over several sessions. However, the few sessions near the end of treatment in which theta was elevated were attributed (by HG) to severe headaches or joint pain. The other objective measure, cognitive evoked potentials, indicated that the P3 conducted at the end of therapy was improved from a prior test, but was still not normal for the patient's age.

Although gains made by the patient are believed to be primarily related to EBF, other factors may have influenced the outcome. In clinical practice, it is difficult to have pure EBF training sessions. Adjustments must occasionally be made in other treatment areas, which could unduly impact results. For example, some suggestions (preliminary cognitive-behavior therapy) for enhancing cognitive skills were provided in sessions, such as writing notes, recitation of things to remember, and practicing cognitive exercises. Although we feel this may have strengthened the overall outcome, it is very unlikely that it was the primary factor, since HG had been engaging in some of these behaviors prior to initiating EBF. Secondly, HG was placed on the antidepressant Zoloft (Sertraline Hydrochloride) on 7/24/95 (session 35), with subsequent improvements in energy level and

Figure 1
Changes in beta, theta, and ratio throughout sessions 1 through 18 (beta = 13 to 20 Hz)

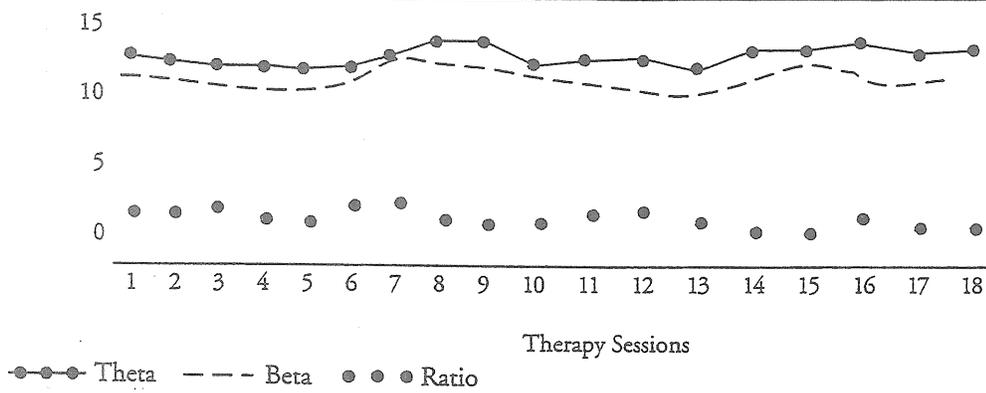


Figure 2
Changes in beta, theta, and ratio from sessions 19 through 24 (beta = 15 to 20 Hz)

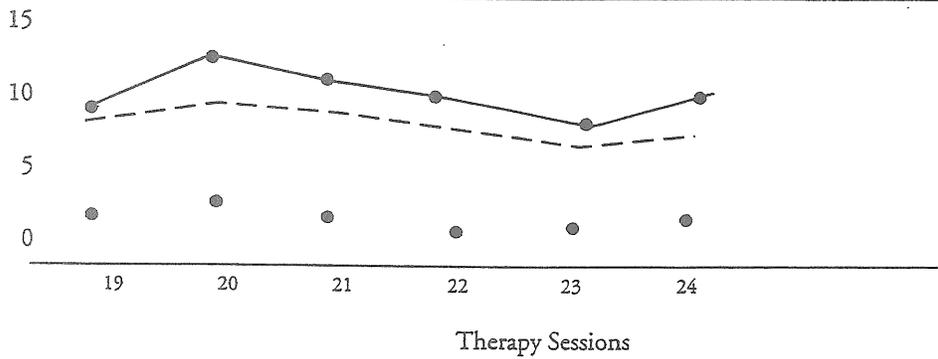
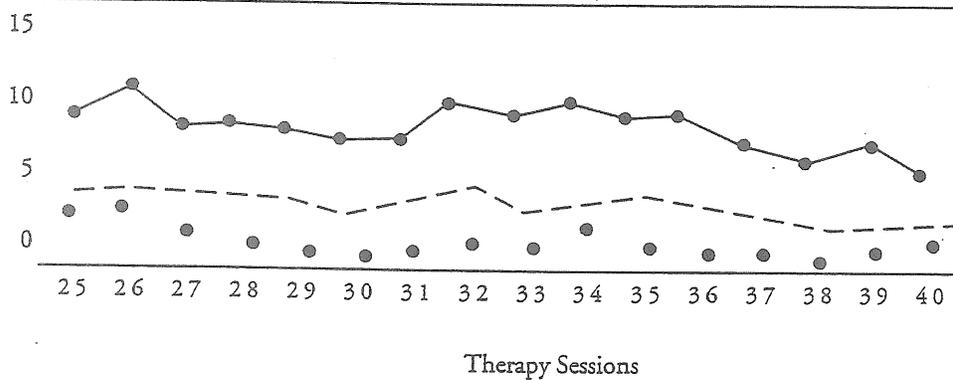


Figure 3
Changes in beta, theta, and ratio throughout sessions 25 through 40 (beta = 13 to 18 Hz)



headaches. While the addition of Zoloft undoubtedly had a positive influence on certain areas (pain and energy level), we do not feel that it significantly affected cognitive skills, since substantial gains were occurring prior to this time and the patient did not show significant depressive features during treatment. Finally, in many investigations the time factor (i.e. improvement occurring throughout treatment simply due to the passage of time) is the principle caveat. In this study, the time factor was not an issue, since the patient had cognitive symptoms since 1987 (eight years). In fact, time passage could provide more credence to EBF, since the patient appeared to be slowly declining before treatment.

One characteristic which may have particularly benefited HG was his motivation. He attended all treatment sessions, had a positive attitude about his situation and improvement, and practiced cognitive exercises (worked on his computer, solved mathematical problems, and read various articles) without prompting by the therapist. The effect of motivation on EBF across a variety of disorders could be explored in future investigations.

Unfortunately, this case study was conducted retrospectively. Therefore, many interesting areas could not be explored due to the completion of treatment. For instance, we were unable to have comparison neuropsychological tests conducted after treatment. Secondly, beta scores at the end of treatment were not directly comparable to beta scores in initial sessions, since beta range was shaped from 15 to 18 Hz. Thirdly, other frequencies (SMR, alpha) were not consistently recorded, resulting in a lack of firm conclusions about areas other than beta/theta. Fortunately, delta was often recorded, indicating a gradual decrease over therapy similar to theta. Finally, as previously discussed, EBF could not be conducted in isolation due to the clinical nature of this study.

In sum, study results indicate that EBF may represent a possible treatment alterna-

tive for individuals with LD and cognitive symptoms in advanced stages. Findings, however, are obviously preliminary due to the study's single-subject status and the limitations previously discussed. More research is needed and recommended. It is interesting that many clinicians have reported significant progress with EBF in a variety of conditions associated with cognitive decline (Ayers, 1991, 1995; Budzynski, personal communication; Hoffman et al., 1995). Due to the staggering cost of cognitive dysfunction to the individual and society at large, and the lack of other viable treatment alternatives, the potential for EBF to revolutionize the field is profound. We recommend, however, that a great deal more research be conducted, documented, and reported before EBF is accepted as a "cure all." This will give EBF more credence with patients, skeptical clinicians, and insurance companies.

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