Treatment of Fibromyalgia Incorporating EEG-Driven Stimulation: A Clinical Outcomes Study

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Thirty patients from a private clinical practice who met the 1990 American College of Rheumatology criteria for fibromyalgia syndrome (FS) were followed prospectively through a brainwave-based intervention known as electroencephalograph (EEG)-driven stimulation or EDS. Patients were initially treated with EDS until they reported noticeable improvements in mental clarity, mood, and sleep. Self-reported pain, then, having changed from vaguely diffuse to more specifically localized, was treated with very modest amounts of physically oriented therapies. Pre- to posttreatment and extended follow-up comparisons of psychological and physical functioning indices, specific FS symptom ratings, and EEG activity revealed statistically significant improvements. EDS appeared to be the prime initiator of therapeutic efficacy. Future research is justified for controlled clinical trials and to better understand disease mechanisms. © 2001 John Wiley & Sons, Inc. J Clin Psychol 57: 933–952, 2001.

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Fibromyalgia syndrome (FS) is a common rheumatologic disorder seen disproportionately in females that is characterized by diffuse musculoskeletal aching, the presence of multiple tender points at specific soft tissue sites, and often by morning stiffness, persistent fatigue, and nonrestorative sleep (McCain, 1993; Moldofsky, 1989; Moldofsky, Scarisbrick, England, & Smythe, 1975; Wallace, 1997; Wolfe et al., 1990). It also is frequently seen in association with various other physical complaints such as auditory and photo sensitivity, dysmenorrhea, headaches, irritable bowel syndrome, and neurovascular disturbances as well as psychological distress and cognitive complaints (Aaron et al., 1996; Baumstark & Buckelew, 1992; Hudson, Goldenberg, Pope, Keck, & Schlesinger, 1992; Wolfe, Ross, Anderson, Russell, & Hebert, 1995). In addition, FS is frequently associated with chronic fatigue syndrome (CFS) in that over 60% of persons diagnosed with primary CFS also will meet the classification criteria for FS (Goldenberg, 1989).

The cause of FS is unknown, and the onset may or may not be preceded by an identifiable illness or other physical or emotional trauma (Aaron et al., 1997; Goldenberg, 1993; Greenfield, Fitzcharles, & Esdaile, 1992; Turk, Okifuji, Starz, & Sinclair, 1996; Waylonis & Heck, 1992; Waylonis & Perkins, 1994). Explanations that focus on hypothesized central nervous system (CNS) mechanisms (e.g., electroencephalograph [EEG] abnormalities detected during sleep studies associated with nonrestorative sleep, generalized hypervigilance and perceptual amplification of pain, neuroendocrine deficiencies, neurosensitization) have been increasingly prominent (Dessein, Shipton, & Cloete, 1997; Johansson et al., 1995; McDermid, Rollman, & McCain, 1996; Mountz et al., 1995; Russell, 1993; Wall, 1993). The efficacy of available treatments (e.g., pharmacologic, aerobic exercise, biofeedback, hypnosis, physical therapies, multimodal/multidisciplinary, and so on) have typically demonstrated only moderate success, and large numbers of patients remain very impaired and even disabled (Bennett et al., 1996; Burckhardt, Mannerkorpi, Hedenberg, & Bjelle, 1994; Goldenberg, 1994; Haanen et al., 1991; McCain, 1994; McCain, Bell, Mai, & Halliday, 1988; Schwartz, 1995; Wallace, 1997; White & Harth, 1996).

Evaluations of biofeedback treatments involving surface electromyography (sEMG) procedures have suggested potential benefits for some patients (Schwartz, 1995). However, few studies actually exist. Ferraccioli and colleagues (Ferraccioli et al., 1987; Ferraccioli et al., 1990) demonstrated the efficacy of sEMG biofeedback combined with relaxation training and also identified a subset of nonresponders who were characterized by a specific set of neuroendocrine abnormalities. More recently, Minhoto, Roizenblatt, and Tufik (1997) documented decreases in the number of tender points reported and pain thresholds of patients treated with biofeedback. Our own attempts at neuromuscular retraining using sEMG that we have developed to treat myofascial pain (Donaldson & Donaldson, 1990; Donaldson, Romney, Donaldson, & Skubick, 1994; Donaldson, Skubick, Clasby, & Cram, 1994; Skubick, Clasby, Donaldson, & Marshall, 1993; cf. Cram & Kasman, 1998) appeared to be less successful clinically with patients diagnosed with FS and/or those with related symptoms including persistent fatigue, poor localization of pain, cognitive complaints (e.g., reduced ability to focus attention and maintain concentration, "foggy" thinking, forgetfulness), and nonrestorative sleep (Donaldson, Nelson, & Schulz, 1998; Donaldson, Sella, & Mueller, 1998).

In addition, our clinical experience with generalized chronic pain and FS specifically convinced us that the pain experienced by these patients is largely maintained by changes in the CNS along the lines suggested by Coderre and colleagues (Coderre, Katz, Vaccarino, & Melzack, 1993) in their central plasticity model of chronic pain. This model of chronic pain holds that prolonged sensory disturbances associated with tissue injury at the periphery can result from either a reduction in the threshold of pain receptors or an increase in

the excitability of CNS neurons involved in pain transmission and perception. Increased pain perception may disturb sleep and mood and, in turn, negatively affect neurotransmitter and hormonal balances. Moreover, such a neurosensitization process may become self-perpetuating through CNS-activated autonomic and musculoskeletal reactions such as sympathetic nervous system activation and increased muscle tension. These may, in turn, result in muscle ischemia and hypoxia and the release of pain-producing substances in the periphery which will feedback to the CNS (cf. Donaldson, Nelson, & Schulz, 1998; Flor, Birbaumer, & Turk, 1990). The overall result may be a chronic generalized pain syndrome that has prominent associated neurosomatic symptoms (i.e., cognitive, mood, sleep, and related disturbances).

In line with this view of FS and CFS as forms of neurosensitization syndromes as well as the findings of specific EEG abnormalities during sleep studies for a substantial subset of patients with FS (Moldofsky et al., 1975), recent reports of EEG abnormalities in the waking EEG of FS and CFS patients (Billiot, Budzynski, & Andrasik, 1997; Garloch, 1994; James & Folen, 1996; Tansey, 1993), and the recent surge of interest in brainwave-based biofeedback (also known as EEG biofeedback or neurofeedback; Budzynski, 1996; Byers, 1996; Evans & Abarbanel, 1999; Lubar, 1997; Russell, 1997), we were led to explore the application of brainwave-based neurotherapy techniques oriented toward the remediation of the neurosomatic symptoms associated with FS. Since such symptoms appeared to be associated with poorer response to typical physically based treatments directed toward peripheral pain sites (e.g., massage and/or physical therapy), we selected these symptoms for the initial primary focus of intervention in a multimodal treatment strategy. We utilized a specific form of neurotherapy known as EEG-driven stimulation (EDS).

This form of neurotherapy is related to findings on EEG entrainment along these lines. It is well established that a person's EEG will respond to frequency-modulated stimulation in the range of approximately 1 to 25 Hz by increasing power at approximately the same frequency as the stimulation frequency. This phenomenon is called EEG entrainment, which actually falls within a classical conditioning learning paradigm (Cantor, 1999). The EDS neurotherapy system used in the present study may be understood, in large part, as an interactive EEG entrainment device. That is, the system monitors the patient's EEG in the 0 to 30 Hz frequency band from a single monopolar electrode and uses the Fast Fourier Transformation-calculated dominant frequency (i.e., frequency with the greatest momentary power) to set the rate of the frequency-modulated light stimulation that is fed back to the patient to entrain the EEG. By having the person's own dominant frequency set the stimulation frequency, the entrainment effect is enhanced because the stimulation frequency follows the dominant and maintains a constant difference from the dominant. The difference in frequency between the dominant and the stimulation frequencies can be increased or decreased as necessary to enhance entrainment, and by leading or lagging the dominant, the person's dominant EEG can be entrained upwards or downwards. This article is a report on a preliminary series of 30 patients who have demonstrated substantial improvements on a variety of FS symptoms when treated primarily or exclusively with EDS.

Methods

Participants

Participants were 30 consecutive outpatients diagnosed with FS referred to Myosymmetries International Inc., a multidisciplinary clinic specializing in the treatment of persistent musculoskeletal pain in Edmonton, Alberta, Canada. The sample reflected the

disproportionate gender mix typical of FS (27 women, 3 men). The mean age was 50.7 years (SD = 12.0, range = 27–69), and the mean education level was 13 years (range = 8–16).

All patients had been previously diagnosed with FS by a treating physician. Physical examination by one of the clinic's chartered physical therapists confirmed that all patients met the 1990 American College of Rheumatology (Wolfe et al., 1990) criteria for FS, including tender point assessment by pressure algometer using 4.0 kg/cm^2 as the standard for determining a point as positive for FS pain. Five patients also met current U.S. Centers for Disease Control (CDC) criteria for CFS (Fukuda et al., 1994). Four of these five patients initially were diagnosed with CFS before fully meeting all criteria for FS; one was diagnosed with FS but also met CDC criteria for CFS. Four reported a gradual onset of their CFS symptoms without any identifiable precipitating factors whereas one developed CFS quite rapidly following a severe parasitic gastrointestinal infection. Two of the CFS patients eventually also developed FS subsequent to sustaining traumatic muscle injuries to the neck and shoulder girdle.

Other medical problems (with >1 of the following sometimes occurring in the same individual) included angina (n = 1), a history of anorexia (n = 1), arteriosclerosis (n = 2), asthma (n = 4), elevated cholesterol (n = 4), diabetes (n = 1), epilepsy (n = 1), gastrointestinal disturbances (irritable bowel syndrome n = 9, two of whom also had diverticulitis), hypertension (n = 6), hypoglycemia (n = 2), hypotension (n = 2), hypothyroidism (n = 5), migraine (n = 2) and other headache (n = 8), mitral valve prolapse (n = 2), osteoarthritis (n = 6), polycystic ovary disease (n = 1), polymyalgia rheumatica (n = 1), premenstrual syndrome (n = 4), tinnitus (n = 1), and significant past surgeries of cholecystectomy (n = 1), hysterectomy (n = 10), and lobectomy (n = 1). Further, based on an intake interview by a chartered psychologist (H.H.M.) and symptom questionnaire responses, clinically significant (major) depression was noted in six patients, one of whom also had a concurrent independent psychiatric diagnosis of anxiety disorder with panic attacks, and another had an independent psychiatric diagnosis of obsessive-compulsive disorder (OCD). This co-occurrence of other multiple medical problems and psychological syndromes is not unusual for samples of patients with FS.

The onset of FS had been gradual (i.e., full-blown FS developed over the course of 3 months or longer) in 24 cases, following such diverse events as viral or flu-like illnesses (n = 6, one of whom had later sustained traumatic injuries in boating and motor vehicle accidents), traumatic soft tissue injuries involving the neck or back (n = 9), infection of the ovaries after birth of last child and subsequent hysterectomy (n = 1), severe psychological trauma (n = 4), and for no apparent reason (n = 4). Sudden onset (i.e., symptoms developed within less than 3 months) was reported by six participants, including following severe gastrointestinal infection, possibly giardiasis (n = 1), rubella immunization (n = 1), surgery (n = 2), and viral or flu-like illness (n = 2).

Prescription and nonprescription medications for analgesia (e.g., acetaminophen with and without opioids) and inflammation (various nonsteroidal antiinflammatories [NSAIDs] such as diclofenac), migraine (e.g., sumatriptan), sleep (e.g., amitriptyline, cyclobenzaprine, zopiclone), psychological disorders (e.g., buspirone, fluvoxamine), and various other concurrent medical conditions (e.g., antiseizure drugs, diuretics, estrogen/progesterone, insulin, thyroxin, and so on) were supplemented with a variety of complementary or alternative over-the-counter (OTC) preparations such as borage oil, coenzyme Q10, DHEA, flaxseed oil, garlic powder, gingko biloba, ginseng, L-tryptophan, melatonin, NADH, peppermint oil, and high potency multiple vitamin and mineral supplements. This is consistent with the reported high use of alternative medicine therapies by patients with FS in Canada (Pioro-Boisset, Esdaile, & Fitzcharles, 1996).

The mean duration since formal medical diagnosis with FS was 5.7 years ($SD = 4.9$, range = 1–24), although the first appearance of symptoms often extended further back in time. Regardless of previous treatments, all patients were still quite symptomatic with FS on a daily basis at intake. Patient age and duration of FS symptoms were not significantly correlated ($r = .28$, $p = .13$). Furthermore, at the time of intake, 16 patients were off work on short-term medical or long-term disability benefits, five were homemakers not in the workforce, six were fully retired, one was retired but also working on a part-time basis, and two were working part-time only.

Measures

Modified Fibromyalgia Impact Questionnaire. A modified version of the Fibromyalgia Impact Questionnaire (mFIQ; Burckhardt, Clark, & Bennett, 1991) was administered at the initial intake evaluation and again at extended follow-up. Questions were added to reflect the history and the impact of illness in terms of both FS and CFS. Three items from the original FIQ were deleted. The retained items queried regarding interference in a variety of activities of daily living, quality of sleep, time missed from work and other interference at work, severity of pain, degree of fatigue, feelings of rest and stiffness on awakening, anxiety/nervousness/tension, and depression. The time frame on which patients were to base their ratings was specified as the previous 7 days. Those items that had originally been rated on 10-cm visual analog scales were changed to 0 to 9 Likert-type rating scales with similar anchors specified.

Symptom Checklist 90-Revised. The Symptom Checklist 90-Revised (SCL-90-R; Derogatis, 1994) is a list of 90 different somatic and/or psychological symptoms which patients were asked to rate individually with respect to the level of distress each symptom caused them over the previous week on a scale from 1 (*not at all*) to 5 (*extremely*). In addition to a Global Severity Index (GSI), which is simply the mean sum of all item ratings and reflects overall distress level, nine separate subscale scores reflecting specific areas of distress may be computed.

It is important to note that some of the SCL-90-R subscales are not entirely as their names may suggest. For example, it is possible for a respondent to obtain a moderately elevated score on the obsessive-compulsive subscale without endorsing any items strongly associated with clinical OCD, but rather by endorsing only items that may reflect cognitive dysfunction such as "Trouble remembering things," "Trouble concentrating," and "Difficulty making decisions," which are commonly endorsed by persons with FS and CFS. Similarly, SCL-90-R respondents can obtain mildly elevated scores on the psychoticism subscale simply by responding in the affirmative to two items commonly endorsed by persons with FS and CFS, namely, "The idea that something is wrong with your body" and "The idea that something is wrong with your mind." Hence, mild to moderate elevations on these two SCL-90-R subscales may have little relation to actual OCD or psychotic thought disorder.

Visual Analog Scales. A separate 10-cm visual analog scale (VAS; Jensen & Karoly, 1992) was used by patients to rate their symptoms (anchors in parentheses) at intake and at the beginning of each treatment session in terms of Current Pain Intensity (no pain, worst pain ever), Quality of Previous Night's Sleep (no sleep, best sleep ever), Current Level of Fatigue (no fatigue, worst fatigue ever), Current Level of Cognitive Clouding (no cognitive clouding, worst cognitive clouding ever), Current Level of Depression (no depression, worst depression ever), and Current Level of Anxiety (no anxiety, worst

anxiety ever). Higher scale scores reflect greater dysfunction, except for Quality of Previous Night's Sleep, for which the direction of the scale is reversed (i.e., higher scores reflect better sleep).

Procedures

Initial Physical Examination and Questionnaire Assessment. The diagnosis of FS was first confirmed by an interview between each patient and a chartered psychologist (H.H.M.) which included a detailed history with regard to the nature of the patient's symptoms as well as their onset and duration, medical examinations and diagnostic tests done, previous therapies and outcomes, previous and current medications, and other standard matters. Patients also completed the mFIQ and SCL-90-R. In addition, one of the clinic's two chartered physical therapists interviewed each patient with respect to their pain complaints, had them draw their pain distribution on a human figure outline, and verified the presence of the requisite number of positive tender points according to ACR classification criteria for FS that included testing pain threshold at each of 18 designated tender point locations (ACR 1990 criteria; see Wolfe et al., 1990) and four control sites (Fischer, 1987, 1998) using a pressure algometer (Pain Diagnostics & Thermography Inc., Great Neck, NY). Tender points were designated as positive if a pain response was elicited at a pressure less than 4.0 kg/cm^2. The percent of the body involved in pain was calculated from each patient's pain distribution drawing using a 5×5 mm grid printed on a clear plastic overlay.

EDS Assessment and Treatment System. The Flexyx EDS photic stimulation system (Ochs, 1993, 1997) uses proprietary DOS 6.0 software (Flexyx LLC, Walnut Creek, CA) to link a digital brainwave recording device (I-410 physiological data module, J & J Engineering Inc., Poulsbo, WA) through a 486DX66 IBM-compatible computer with an installed Synetics Synergizer Light/Sound Control Board to a set of green light-emitting diodes (LEDs) built into a set of eyeglasses with darkened lenses worn by the patient. The Flexyx EDS software permits calculation of the patient's dominant EEG frequency (frequency of momentary peak energy) within the 1- to 30-Hz range from moment to moment and the application of this dominant frequency to control the frequency of the flashing LEDs in the eyeglasses worn by the patient. The software allows the therapist to view the patient's raw evoked EEG and a Fast Fourier Transformation (FFT) processed display of evoked spectral EEG in relative real time and adjust various aspects of the light stimulation (e.g., setting the amount by which the flash rate leads or lags the patient's dominant frequency, a fixed flash rate from 1 to 30 Hz, the light intensity, the flash duration, and/or the degree of flash offset between the right and left lenses of the eyeglasses). Session data can be saved for later review, and analysis and reports with topographic brain maps can be generated. Monopolar referential photo-evoked EEG is recorded with a single active 9-mm gold-plated, Grass-type cup electrode placed using the international 10–20 system with reference to linked ears and with the patient appropriately electrically grounded. Raw evoked EEG is sampled 1,200 times/sec and processed through a series of digital bandpass and FFT filters that average the EEG voltage at each single Hz frequency between 1 and 30 Hz inclusive over 9 samples (128 times/sec). The single frequency showing the greatest EEG root mean square voltage at each averaging is designated as the dominant frequency. A second level of bandpass filters averages the data for seven different wave bands: delta (0–4 Hz), theta (4–8 Hz), alpha (8–12 Hz), beta1 (12–16 Hz), beta2 (16–20 Hz), beta3 (20–24 Hz), and beta4 (24–30 Hz).

The assessment phase calls for a brief 24-sec sample of evoked EEG data from each of 21 individual scalp sites in a standard predetermined sequence: FP, FP1, F7, T3, T5, O1, O2, T6, T4, F8, FP2, F3, C3, P3, P4, C4, F4, Fz, Cz, Pz, and Oz. At each site, four 6-sec samples of evoked EEG are recorded with the flashing LEDs set to their lowest intensity (1 of 49) and shortest flash duration (1 of 50, light on 1% of each on/off cycle) with 0 degrees of right/left offset, and leading frequencies set at +5, +10, +15, and +20 Hz, respectively. Electrode impedances are maintained at less than 5 K ohms. During recording, the patient is sitting comfortably upright and still, fully awake with eyes closed, and wearing the EDS eyesets. Mapping takes approximately 30 to 45 min. Evoked EEG amplitude and *SD* data are averaged across the four different leading conditions for each of the 21 scalp locations by each of the seven wave bands and plotted as a topograph as well as column graphs showing sites arranged by increasing microvolt amplitude and *SD*s.

EEG active treatment sites vary from patient to patient as do the number and length of sessions at each site. Generally, treatment sites are selected on the basis of where each patient demonstrates the highest amplitudes and *SD*s of evoked EEG in the low frequency bands (i.e., delta, theta, and alpha) on the initial mapping. The goals of therapy are to reduce the overall amplitude of evoked EEG in the delta, theta, and alpha wave bands to approximately the same level as the beta activity and to increase the variability of the dominant frequency from all sites where initial mapping shows excess slow wave activity (e.g., slow:fast ratios $\geq$3:1). Treatment sessions are generally one hour in length and scheduled at least twice weekly, although more frequent sessions (4–5 per week) are encouraged early in treatment.

Actual Intervention Protocols. Following the initial EDS assessment and mapping of relative brain wave activity, patients were started on a course of EDS treatment sessions involving photic stimulation at various scalp sites according to the rationale provided earlier. All were initially treated with once-daily one-hour EDS sessions, 3 to 5 sessions per week. EDS treatments focused primarily on reducing the amplitude of slow delta and theta wave activity from those sites that revealed excessive slow wave amplitudes on the initial assessment mapping (i.e., mean amplitudes >3.0 mV). EDS treatments continued until the patient's EEG began to show stability in a more normal pattern of reduced delta and theta amplitudes with frequent and free ranging of the dominant EEG frequency above 12 Hz (as observed during treatment sessions) and patient self-reports of increased cognitive clarity, improved sleep, increased mental and physical energy, reduced affective disturbances, and increased awareness of localized as opposed to generalized pain. Once these criteria were met, patients were generally reduced to 1 or 2 EDS sessions per week and started on approximately once weekly massage and physical therapy treatments focused on obtaining myofascial release and reducing their now more localized pain, on improving muscle stretch and functional balance, and on helping patients better manage any behavioral or postural perpetuating factors. Most of the patients who had massage and physical therapy also had some amount of neuromuscular retraining using sEMG biofeedback to correct muscle imbalance patterns associated with myofascial pain syndromes, as described in previous reports (Donaldson & Donaldson, 1990; Donaldson, Nelson, & Schulz, 1998). Since five patients had been referred and/or desired only to undergo EDS as treatment of their FS, only 25 subjects were involved in these other therapies (A sixth patient had only massage therapy as additional treatment.) Patients discontinued therapy when they experienced sufficient alleviation of their FS symptomatology or ran out of time or money for further therapy. At the start of each EDS session, patients completed the six VAS rating scales pertaining to levels of experienced pain, fatigue, cognitive clouding, quality of sleep, depression, and anxiety.

Posttreatment Assessment. At the conclusion of active treatment, a brain map of quantitative photo-evoked EEG activity via the EDS system was again recorded using procedures identical to those used for the initial map recorded at intake. Patients also completed the SCL-90-R a second time and again drew their pain distribution on a human figure outline. Seventeen patients were reevaluated by a physical therapist for tender points at the end of treatment. This reevaluation was done following the same procedures as outlined earlier using a pressure algometer and a cut-off standard of less than 4 kg/cm^2. Only 17 of 30 patients were reevaluated for tender points because the physical therapy reassessment component was added to the treatment protocol after the first few patients in this series had completed their treatment, and six patients did not elect to have any physical therapy following EDS.

Follow-Up Assessment. A number of patients lived long distances from the clinic (e.g., in another city or province) and it was not feasible, especially within the constraints of a standard clinical practice setting without research funding, to have them return for an actual physical examination follow-up. However, a number of months after completion of treatment sessions, all patients were mailed the mFIQ and asked to complete and return it via the mail.

Data Analyses

Primary data analyses involved pre- to posttreatment and follow-up comparisons on mean levels of the variables of interest using two-tailed, paired *t*-tests. Additional *t*-test comparisons examined changes from the beginning of treatment to the time additional therapies were introduced and to the end of treatment in order to better assess the effects of EDS prior to the initiation of these other therapies. Since this was an exploratory study involving the application of a large number of independent analyses with concomitant risk of Type I error, a conservative significance level of .01 was adopted. No other adjustment for multiple comparisons was applied.

Results

Over the course of their treatment at Myosymmetries, these 30 patients averaged 51.9 ± 23.6 hr (range = 20–135) of treatment, including an average of 37.3 ± 15.6 hr of EDS, 7.1 ± 6.9 hr of massage therapy, 5.6 ± 5.3 hr of physical therapy, and 2.0 ± 2.4 hr of sEMG neuromuscular retraining over the course of approximately 15 ± 7 weeks (Table 1). Only four patients had EDS treatment only; two others had only EDS and massage therapy but no physical therapy or sEMG. Total cost of assessment and treatment averaged approximately c. $3,500 to $4,500.

SCL-90-R Findings

Table 2 shows pre- and posttreatment mean scores on the SCL-90-R. Patients' raw scores for each subscale and the GSI were converted to *T* scores ($M = 50$, $SD = 10$) based on the general population nonpatient norms for females and males given in the SCL-90-R manual (Derogatis, 1994). *T*-score values equal to or greater than 64 (i.e., approximately 1.5 *SD*s above the *M*, 84th percentile) are considered clinically significant in terms of distressed functioning. The GSI as well as all individual subscale scores were statistically significantly different from pre- to posttreatment except for the phobic anxiety and paranoid

Table 1
Hours of Each Type of Therapy and Number of Weeks in Treatment for Each Individual Patient

	Type of Treatment					
Participant No.	EDS	sEMG	Physical Therapy	Massage Therapy	Total Hours of Treatment	Number of Weeks
1	80	3	23	29	135	12
2	37	0	1	9	47	7
3	39	1	4	0	44	16
4	31	6	12	0	49	30
5	33	0	6	6	45	15
6	41	0	0	0	41	16
7	45	1	1	8	55	19
8	50	11	12	13	86	22
9	18	0	0	2	20	12
10	72	0	7	6	85	12
11	34	4	11	11	60	9
12	20	0	0	0	20	6
13	23	0	8	10	41	10
14	29	4	5	3	41	36
15	16	3	7	4	30	5
16	58	0	0	0	58	26
17	70	0	4	10	84	19
18	22	2	5	2	31	5
19	29	2	5	10	46	6
20	43	4	3	7	57	18
21	31	2	7	12	52	12
22	32	1	3	0	36	10
23	30	2	7	15	54	19
24	28	0	0	3	31	13
25	32	3	3	2	40	20
26	29	0	0	0	29	10
27	31	4	16	18	69	18
28	39	2	3	5	49	12
29	30	2	4	10	46	11
30	46	2	10	18	76	15
M ± *SD*	37.3 ± 15.6	2.0 ± 2.4	5.6 ± 5.3	7.1 ± 6.9	51.9 ± 23.6	14.7 ± 7.3

Note. Participant numbers were randomly assigned to patients for this report and do not reflect the order in which patients began or ended their treatment. EDS = EEG-Driven Stimulation; sEMG = surface electromyography.

ideation subscales. Overall, there was evidence of a clear shift of indicators of psychological symptomatology from clinically and, generally, mildly distressed to normal levels. All 30 patients showed a reduction in their GSI over the course of treatment (range = 2–19 *T*-score points). The largest pre- versus posttreatment differences were apparent for the somatization, obsessive-compulsive, and depression subscales and the GSI.

Treatment Session Self-Report Symptom Ratings

The mean of the first 10 VAS ratings for each specific symptom was calculated and compared to the mean of the last 10 VAS ratings for each specific symptom. In all instances, the ratings from the beginning to the conclusion of treatment were in a favorable direc-

Table 2
Pre- and Posttreatment SCL-90-R Comparisons

SCL-90-R Subscale	Pretreatment *M*	Pretreatment *SD*	Posttreatment *M*	Posttreatment *SD*	*t* (*df* = 29)	Statistical Significance (two-tailed)
SOM	67.9	6.8	59.2	7.6	10.14	$p < .0001$
O-C	66.3	7.7	55.0	8.8	9.53	$p < .0001$
I-S	57.7	10.1	51.6	8.7	4.55	$p < .0001$
DEP	63.2	5.9	53.9	6.8	12.61	$p < .0001$
ANX	56.6	9.7	49.1	9.5	5.36	$p < .0001$
HOS	53.5	10.0	46.4	6.9	4.77	$p < .0001$
PHOB	50.1	11.2	45.6	8.3	2.06	$p = .05$, n.s.
PAR	49.9	8.8	46.9	7.6	2.25	$p = .03$, n.s.
PSY	56.1	9.6	49.8	9.0	3.71	$p < .001$
GSI	63.9	5.9	54.2	7.7	10.82	$p < .0001$

Note. SCL-90-R = Symptom Checklist 90-Revised; SOM = Somatization; O-C = Obsessive-Compulsive; I-S = Interpersonal Sensitivity; DEP = Depression; ANX = Anxiety; HOS = Hostility; PHOB = Phobic Anxiety; PAR = Paranoid Ideation; PSY = Psychoticism; GSI = Global Severity Index; n.s. = not statistically significantly different. SCL-90-R GSI and subscale scores are presented as *T* scores ($M = 50$, $SD = 10$) based on general population nonpatient norms published in the SCL-90-R manual (Derogatis, 1994). Higher scores indicate greater dysfunction.

tion, and differences were statistically significant. As a general matter, the various subjective ratings of symptom intensity/severity were reduced to quite low levels by the end of treatment (Table 3). The largest differences with treatment were apparent for ratings of sleep quality, cognitive clouding, and pain intensity. Sense of fatigue or tiredness, while decreased, appeared somewhat more persistently present, albeit at significantly lower levels.

Table 3
Beginning of Treatment Versus Point of Starting Other Physically Oriented Therapies Versus Posttreatment Comparisons of Self-Report VAS Symptom Ratings

	A		B		A minus B	C		B minus C	A minus C
	Beginning of Treatment		Start of Other Physically Oriented Treatment			End of Treatment			
VAS ratings	*M*	*SD*	*M*	*SD*	*t* (*df* = 29)	*M*	*SD*	*t* (*df* = 29)	*t* (*df* = 29)
Pain intensity	5.4	1.6	3.9	1.6	6.79**	2.5	1.7	6.30*	9.91**
Sleep quality[a]	2.9	1.8	6.6	1.4	−11.99**	6.8	1.5	−1.20	−15.29**
Fatigue	5.8	1.5	4.2	1.6	5.02**	3.5	2.9	1.89	4.42*
Cognitive clouding	5.6	1.8	2.4	1.5	10.03**	1.9	1.4	1.85	14.64**
Depression	2.6	2.4	1.2	1.4	5.12**	0.9	1.3	2.01	5.32**
Anxiety	3.4	2.5	1.3	1.4	6.64**	1.1	1.5	1.01	6.39**

Note. VAS = 10-cm visual analog scale; Pain intensity = Current Pain Intensity; Sleep quality = Quality of Previous Night's Sleep; Fatigue = Current Level of Fatigue; Cognitive clouding = Current Level of Cognitive Clouding; Depression = Current Level of Depression; Anxiety = Current Level of Anxiety.

[a]Higher scores on all VAS ratings reflect greater dysfunction, except for Sleep quality, for which the direction is reversed.

*$p < .001$; **$p < .0001$. All other *t* values not statistically significantly different. All significance levels are 2-tailed.

Table 4
Pre- Versus Posttreatment Comparisons of Mean Amplitudes in Root Mean Squared Microvolts of EEG Activity Recorded and Averaged across 21 Scalp Sites

	Pretreatment		Posttreatment			
EEG band	*M*	*SD*	*M*	*SD*	*t* (*df* = 29)	Statistical Significance (two-tailed)
Delta (1–4 Hz)	4.18	1.37	2.87	0.85	6.65	$p < .0001$
Theta (4–8 Hz)	3.81	1.36	2.71	1.15	6.38	$p < .0001$
Alpha (8–12 Hz)	3.75	1.86	3.05	1.47	3.26	$p < .01$
Beta1 (12–16 Hz)	2.33	0.99	2.21	0.68	0.81	$p = .42$, n.s.
Beta2 (16–20 Hz)	2.34	1.07	2.22	0.52	0.63	$p = .54$, n.s.

Note. n.s. = not statistically significantly different.

Pre- versus Posttreatment EEG Findings

Table 4 presents a comparison of pre- to posttreatment mean amplitudes in microvolts of delta, theta, alpha, beta1, and beta2 activity as recorded from 21 scalp sites by taking 24-sec samples with eyes closed from each location. Excessively high amplitude delta and theta and low alpha (8–10 Hz) were most commonly found in the following 11 (generally more anterior and central) locations: FP, FP1, FP2, Fz, F3, F4, F7, F8, Cz, C3, and C4, and these were also the areas most frequently treated. Despite the fact that calculating pre- versus posttreatment mean amplitudes based on all 21 sites, as opposed to only those treated, will tend to minimize the size of changes, statistically significant reductions in delta, theta, and alpha were obtained ($t = 6.63$, $p < .0001$; $t = 6.38$, $p < .0001$; $t = 3.26$, $p < .005$, respectively; *df*s = 29). The greatest pre- versus posttreatment difference was apparent for the delta band. No significant changes were noted for the low (beta1 = 12–16 Hz) or middle beta (beta2 = 16–20 Hz) wavebands.

Pre- versus Posttreatment Tender Points and Pain Distribution

Table 5 presents the pre-treatment algometer data for all 30 patients and a comparison of pre- to posttreatment algometer data for those 17 (57%) patients who were examined twice. Table 5 also presents the mean number of positive tender points as well as the mean algometer pressure (in kg/cm^2) to elicit pain (i.e., pain threshold) for the 18 ACR tender point sites. In addition, Table 5 presents a comparison of pre- to posttreatment percent of the body covered by pain for each of the 30 patients as derived from their pain distribution drawings completed on intake and repeated on discharge.

Before treatment, the 30 patients averaged 15.0 (*SD* = 2.0, range 11–18) positive tender points (i.e., pain elicited at a pressure of <4.0 kg/cm^2) of the 18 ACR tender point sites, with a mean pressure to elicit a pain response of only 2.6 (*SD* = 0.5, range = 1.9–3.6) kg/cm^2. Following treatment, the 17 patients who were reassessed averaged only 8.0 (*SD* = 3.0, range 2–14) positive tender points, with 13 (76%) having fewer than the 11 positive tender points required by the ACR classification criteria for FS. The mean pressure required to elicit pain over the 18 ACR tender point sites had risen to 3.8 (*SD* = 0.9, range = 2.6–6.4) kg/cm^2 with treatment. These differences in number of positive tender points and pain threshold from pre- to posttreatment were statistically significant ($t = 9.29$, $p < .0001$, and $t = -7.39$, $p < .0001$, respectively, *df*s = 16). All 17 patients

Table 5
Percent of Body Involved in Pain, Mean Pressure Readings for Pain Threshold from 18 ACR Tender Point (TP) Locations, and Number of Positive TPs at Pre- and Posttreatment Physical Examinations

	Pretreatment Pain Map and TP Palpation			Posttreatment Pain Map and TP Palpation		
Participant No.	Percent of Body Involved in Pain	Mean Algometer Pressure for Pain for 18 TPs (kg/cm^2)	Number of Positive TPs at <4 kg/cm^2	Percent of Body Involved in Pain	Mean Algometer Pressure for Pain for 18 TPs (kg/cm^2)	Number of Positive TPs at <4 kg/cm^2
1	17.7	3.03	11	1.3	–	–
2	21.6	3.59	14	2.4	6.43	2
3	38.6	2.03	17	16.6	2.96	8
4	36.1	2.19	13	11.9	–	–
5	46.3	2.13	17	7.7	–	–
6	37.8	2.93	15	24.3	–	–
7	55.5	1.92	16	56.8	3.31	14
8	43.1	2.03	18	14.8	2.64	12
9	53.9	2.20	17	8.2	3.73	4
10	26.3	3.09	15	19.1	–	–
11	19.7	2.76	14	4.6	3.88	9
12	21.9	2.49	14	0.8	3.47	11
13	34.0	2.07	18	2.2	–	–
14	58.7	2.29	16	17.3	2.88	10
15	28.8	2.82	16	0.7	–	–
16	19.0	2.23	11	1.6	–	–
17	48.2	2.03	16	12.9	4.67	6
18	31.2	2.60	14	11.6	4.40	5
19	38.3	2.43	15	6.8	3.63	7
20	28.4	3.26	12	5.2	–	–
21	32.7	2.63	17	0.9	–	–
22	41.5	3.23	14	2.7	–	–
23	20.8	2.79	15	3.7	–	–
24	11.7	2.15	16	4.1	3.36	6
25	27.1	2.86	11	7.3	4.15	6
26	23.8	3.06	14	4.8	4.72	5
27	44.9	2.90	16	14.3	3.10	14
28	27.6	3.17	15	14.2	–	–
29	12.6	2.71	18	1.9	3.08	10
30	40.6	2.66	17	22.1	3.80	10
$M \pm SD$	33.0± 12.4	2.6 ± 0.5	15.0 ± 2.0	10.1 ± 11.2	3.8 ± 0.9	8.0 ± 3.0

Note. Participants 5, 10, 13, 21, 23, and 28 did not have any physical therapy treatment following their initial intake assessment. ACR = American College of Rheumatology.

who were assessed for number of positive FS tender points and pain sensitivity at both intake and discharge showed a decrease in number of positive tender points and mean pain sensitivity over the course of therapy.

Similarly, based on pain distribution drawings completed at both intake and discharge, the average patient was experiencing pain covering 33.0% (SD = 12.4%, range = 11.7–58.7%) of the body before treatment and covering only 10.1% (SD = 11.2%, range = 0.7–56.8%) of the body at discharge. Again, these pre- to posttreatment reductions in pain distribution were statistically significant (t = 11.49, $p < .0001$, df = 29). All patients

except one demonstrated a reduction in the distribution of their pain from intake to discharge. The only patient not showing a decrease in pain distribution with treatment showed a very small increase of 1.3% in pain distribution. These results suggest that treatment was effective in reducing the percent of body involved in pain as well as measured pain sensitivity over designated tender point locations and, moreover, that patients generally went from meeting the ACR criteria for FS of $\geq$11 positive tender points to no longer fully meeting this classification criteria by the time of their discharge.

Pre- versus Posttreatment Employment and Disability Benefits Status

While no change in employment/benefits status was reported by 19 of the 30 patients (63%) at the time of their follow-up of an average of nearly 8 months posttreatment, (4 remained homemakers without outside employment, 7 remained retired, 1 remained working only part-time in a family business, 2 remained unemployed without disability benefits, and 5 remained unemployed with long-term disability benefits), the other 11 (37%) reported distinct improvements in their status. By the end of treatment, or shortly thereafter, 7 had returned to full-time employment, 3 had returned to part-time employment, and 1 had accepted a retirement package. Moreover, 8 patients had discontinued receiving all disability benefits. Of the 7 subjects who returned to full-time employment, 4 returned directly to full-time employment and 3 initially returned to part-time work and, over the course of a few months, expanded their work participation to full-time. Of the 3 who returned to part-time employment, 1 had previously been employed full-time and hoped to eventually regain this status, another had been employed full-time before taking long-term disability leave, but then took early retirement from her job and later accepted a part-time job to supplement retirement, and the third was a long-time homemaker who added a part-time home-based business to her activities.

Although less than half of the treated patients changed their employment/benefits status following treatment, it is important to note that 15 (50%) of the patients in this study were 55 years of age or older with 7 officially retired at the time of their treatment. Of the group that did not change their status, a disproportionate 12 (63%) were 55 years or older. By contrast, 10 (91%) of the 11 participants who did change their employment/benefits status were younger than 55 years.

Pre- versus Posttreatment Medication Usage

Generally, there was a decrease in the consumption of both prescription and nonprescription drugs for FS-related symptoms over the course of treatment, although there was some increased use of OTC vitamin and mineral supplements (e.g., multivitamins, vitamins B, C, and E, calcium, magnesium, potassium, and so on) as well as various OTC herbal and nutritional preparations (e.g., gingko biloba, ginseng, primrose oil, valerian, and so on). In nearly all cases, patients substantially reduced their consumption of analgesic, NSAID, and muscle-relaxant medications by the end of their treatment. With respect to analgesic medications, all patients who were taking opioid analgesics at the start of treatment had stopped these and substituted nonopioid analgesics or had stopped taking analgesics all together. Similarly, nearly all patients who were taking prescription antidepressant, muscle-relaxant, sedative, or hypnotic medications substantially reduced or stopped these medications by the end of their treatment. No patients increased their use of analgesic, antidepressant, muscle-relaxant, sedative, or hypnotic drugs over the course of treatment. A few patients who stopped using prescription antidepressant, muscle-

relaxant, sedative, or hypnotic drugs over the course of treatment substituted OTC preparations such as St. John's wort, valerian, Kava Kava, or 5HTP. The two patients who were being treated concurrently by psychiatrists for anxiety disorder with panic attacks and for OCD remained on a stable dose of psychotropic medications throughout their treatment at Myosymmetries, but both reduced their use of analgesics. Generally, there was no change in dosage of medications taken for other current health problems such as high cholesterol, diabetes, epilepsy, heart disease, hypertension, thyroid disease, and so on.

Follow-Up Questionnaire Assessment Findings

Although a number of patients had repeated questionnaires sent to them following their discharge, the follow-up mFIQ was eventually completed and returned by all 30 participants. The range of time from completion of treatment to completion of the follow-up mFIQ was 3 to 18 months (M = 8.2, SD = 4.3 months). Responses to the individual sections of the mFIQ are summarized in Table 6. Information on work outside the home was not relevant (i.e., patient was not employed in the week prior to completing the questionnaire) for 20 of the 30 (67%) participants at intake. Information on work outside the home was still not relevant for 18 (60%) patients at follow-up. Inspection of the data revealed that there was generally no worsening of symptoms, and in most instances, marked improvements were reported.

Of particular interest is that the 30 patients reported "feeling good" for an average of only 1.1 days in the seven days prior to completing the mFIQ at intake, versus an average

Table 6
Pretreatment and Follow-Up Modified Fibromyalgia Impact Questionnaire Comparisons

Questionnaire Item/Subscale	Pretreatment		Follow-Up		t (df = 29)	Statistical Significance (two-tailed)
	M	SD	M	SD		
Activities of daily living in previous week[a]	15.7	4.6	7.8	5.3	11.03	$p < .0001$
Days felt good in previous week	1.1	1.5	4.4	2.1	−7.83	$p < .0001$
Nights slept well in previous week	1.8	1.9	5.2	1.5	−8.46	$p < .0001$
Days missed work in previous week	3.8	3.4	0.0	0.0	3.04	$p = .016$, n.s.
On days worked in previous week, rate amount FS/CFS interfered with ability to do job[b]	7.9	1.1	2.2	1.4	14.42	$p < .0001$
Average level of pain over previous week[c]	6.6	1.7	2.7	1.6	13.05	$p < .0001$
Average level of fatigue over previous week[c]	6.8	1.5	3.7	1.8	8.09	$p < .0001$
Average tiredness on waking over previous week[c]	7.1	1.6	3.3	1.9	8.82	$p < .0001$
Average body stiffness on waking over previous week[c]	6.4	1.7	3.4	1.6	7.40	$p < .0001$
Average anxiety over previous week[c]	4.9	2.5	1.5	1.5	7.22	$p < .0001$
Average depression over previous week[c]	3.5	2.6	0.8	1.3	5.67	$p < .0001$
(Follow-up only) Percent of overall improvement since beginning treatment at Myosymmetries:[d] $M \pm SD$ = 62.2% ± 21.6%, range = 20 to 90%						

Note. n.s. = not statistically significantly different.
[a]Scaled from 0–30, with lower scores indicating a higher level of activities in previous week.
[b]Rated using a 10-point scale from 0 (*no problem*) to 9 (*great difficulty*).
[c]Rated using a 10-point scale from 0 (*no problem*) to 9 (*worst ever*).
[d]Percent improvement was indicated on a 0–100% scale marked off in increments of 10%.

of 4.4 days in the seven days prior to again completing the mFIQ on follow-up an average of 8.2 months after treatment ($t = -9.03$, $p < .0001$, $df = 29$). Similarly, their nights of "good sleep" increased from an average of only 1.8 in the week prior to intake to 5.2 in the week prior to follow-up ($t = -9.69$, $p < .0001$, $df = 29$). Ability to carry out activities of daily living, as measured by the mFIQ Activities of Daily Living subscale, improved significantly from intake to follow-up ($t = 11.82$, $p < .0001$, $df = 29$). As well, those patients who were working prior to intake ($n = 10$) averaged 3.8 days of missed work due to FS in the week preceding intake whereas at follow-up those patients who were working in the week prior ($n = 12$) did not miss any days of work due to FS symptoms.

Finally, as an added question to the follow-up mFIQ, patients were asked to indicate the amount of overall improvement in their condition at follow-up as compared to when they first came into treatment on a scale marked off from 0 to 100% in increments of 10%. At the point of their follow-up, patients were on average 62.2% improved (SD = 21.6%, range = 20–90%) in their overall condition as compared to when they first entered treatment. There was no significant correlation between the length of time to follow-up and the amount of overall improvement reported by patients ($r = -0.16$, $p = 0.40$).

Effects of EDS versus Other Therapies

Although the primary treatment modality applied was EDS, patients also were generally treated with varying (modest) amounts of massage therapy, physical therapy, and sEMG-assisted neuromuscular retraining. To elucidate the extent to which EDS appeared to be a prime initiator of therapeutic efficacy, comparisons were made for each of the treatment session VAS (pain intensity, sleep quality, fatigue, cognitive clouding, depression, anxiety) ratings along these lines. The mean ratings for the first 10 sessions of the beginning of treatment with EDS were compared to the VAS rating just before the start of any other therapy; and the beginning of treatment mean ratings also were compared with the end of treatment ratings computed in terms of the mean for the last 10 sessions of treatment. For those patients who received EDS only, VAS ratings obtained at the half-way point of treatment were used instead of the rating just prior to the start of any other therapy. In all instances, the differences from the beginning of treatment to just before the start of any other therapy were statistically significant in the expected direction. Similarly, the beginning of treatment to end of treatment differences also were all statistically significant. However, only the rating for pain intensity showed any further significantly different change from the start of other therapies to the end of treatment. Visual inspection of the data (Table 3) shows very consistent positive effects from the beginning of treatment to each subsequent time point, but suggest that, in general, only modest additional, incremental improvement was obtained after starting the other therapies except for pain intensity, which continued to improve notably with the other therapies.

Discussion

These 30 patients treated primarily with EDS experienced significant reductions in a broad array of symptomatology associated with FS that also corresponded to changes in EEG patterns from the beginning to the completion of treatment. Notable improvements were seen not only in pain intensity but also in cognitive processing difficulties, mood, sleep, and (to a distinct though less marked extent) tiredness and fatigue. Physical examination procedures including pressure algometry further verified physical improvements. Correspondingly, positive changes were highlighted by general reduction in usage of a

variety of prescription and nonprescription medications for pain control, although in some instances there was a self-reported increase in the use of alternative or complementary (e.g., herbal, vitamin, and so on) preparations. Employment and/or disability status remained unchanged in slightly more than half of the subjects, but in the remaining portion major reengagement in employment and relinquishing of disability status were in evidence. Evidence from follow-up questionnaires pointed to benefits being maintained an average of approximately 8 months after treatment termination.

These findings raise both clinical and basic science implications. Practically speaking, the clinical improvements were accomplished with a multimodal treatment package but one that emphasized a specific brainwave-based treatment. EDS was typically the sole focus of treatment until those symptoms most distinctively reflective of CNS dysfunction improved sufficiently for other therapies to then be implemented more effectively. Most frequently, patients first reported significant improvement in mental clarity, such as ability to focus and maintain concentration, as well as mood and restorative sleep. Self-reported pain then appeared to change in nature and quality to the extent that patients began to experience a reduction in their "all over" body pain and an increase in specific localized aches and pains. In tandem with this change from a diffuse to more localized or regional type of pain experience, patients also seemed better able to describe their pain sensation in specific as opposed to vague terms.

This change in pain experience served as an indicator to begin physical therapies focused on reduction and management of the now more localized pains. In our clinical experience, this involved a combination of massage and myofascial release therapies, biomechanical and postural reeducation, muscle stretching and strengthening exercises, and sEMG-guided neuromuscular retraining of identified myofascial pain syndromes and muscle imbalance patterns. Prior to EDS, attempts by means of the more standard physical therapies to treat the diffusely represented pain and associated symptoms of FS had failed. Following initial treatment with EDS, only a very modest amount of these other therapies was required to lead to successful outcomes. The overall cost-effectiveness of this approach with the patients reported in this series was apparent as well.

Given that a multimodal treatment was employed, it might be argued that the role of EDS remains uncertain. Indeed, within the context of this preliminary investigation and due to the limitations of the design of a study conducted in a clinical practice setting without research funding, it is not possible to conclude with certainty that EDS is a beneficial therapy for FS. However, examination of the treatment session VAS ratings for pain intensity, sleep quality, fatigue, cognitive clouding, depression, and anxiety provided some preliminary support for the central role of EDS in instigating the change process in this group of patients. Indeed, comparisons of these ratings from the beginning of treatment to just before any other therapies were instituted and to those obtained at the end of treatment suggested that the major significant differences in symptom ratings were largely accounted for in this first phase of treatment with EDS. Although the trend to continue improvements in all areas was manifested in further incremental positive changes on all symptom ratings, only the pain intensity rating showed further statistically significant change from this intermediate point in the treatment to the end of treatment. Further, it should be noted that the incorporation of other therapies was typically at a low level of intensity, much lower than might typically be the case, and for which there has previously been reported modest (or hardly any) success under most circumstances. Hence, it would appear that EDS may have contributed a specific and necessary ingredient that was the prime initiator of therapeutic efficacy. Still, this is only a tentative conclusion.

Moreover, these findings do not suggest that EDS would necessarily be successful in isolation for the majority of cases, and a multimodal perspective is likely still to be

indicated. There were, though, four participants in this study who received solely EDS as an intervention and benefited substantially from this treatment alone. In the reality of the clinical practice setting in which all of the participants were treated, it is not possible to fully isolate all therapeutic components and their corresponding efficacy. Moreover, it is the clinically apparent success in alleviating symptoms that drives the treatment approach at any given stage.

Further research is justified to verify these suggestive findings in larger samples and in direct comparison with more standard therapies, including single and multimodal interventions in randomized controlled designs. In future research, it will be important to examine the efficacy of EDS in patients randomly assigned to treatment under double-blinded conditions. It also will be important to assess the effects of EDS alone versus any other therapies. For example, EDS alone could be compared with EDS plus other specific therapies in various combinations and in varying sequences of timing to yield component analyses of differential effects. If there is an optimal timing for the institution of other therapies besides EDS, it will be important to identify reliable and quantifiable indicators of this shift, whether in terms of specific levels of symptom ratings, the change from diffuse to more localized pain, patterns of EEG activity, or other parameters. Moreover, given that there was considerable variability in the number of EDS sessions provided, it will be important to better identify the range of optimal number of treatment sessions. Further, given the central role posited for change in the EEG as a function of EDS and presumably corresponding improvements in symptom reports, it will be necessary to more clearly demonstrate the link between changes in the EEG and various outcome measures. This may require more frequent monitoring and mapping of the EEG coinciding with measurements of various symptoms and functional outcomes.

The findings reported here also are provocative in contributing further to the basic scientific understanding of FS. The improvements in symptoms potentially reflective of CNS dysfunction heralding subsequent amenability to other physically oriented therapies were matched by a corresponding change in brainwave patterns from pre- to posttreatment. The relative preponderance of low frequency (delta, theta, and low alpha) activity detected primarily from relatively anterior and central cortical recording sites that was in evidence at the outset of treatment normalized by the conclusion of treatment. This further underscores the potentially important role of CNS dysfunction in ongoing manifestations of FS. The extent to which this may lead to further developments in the understanding of the etiologic or maintaining factors in FS remains for future research to determine. Investigations that utilize more quantitative EEG assessments, instead of the sequential single site recording of the present EDS procedures, as well as correlation with other measures of CNS functioning would contribute to a broadened understanding of CNS processes and FS. In general, then, this approach holds some promise for reducing the suffering of the many individuals debilitated with FS by immediate clinical applications and future refinements in these techniques, and by contributing to a better understanding of the basic disease mechanisms of FS. Future research under more highly controlled conditions will help to evaluate the fruits of this promise.

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