

QEEG Patterns, Psychological Status and Pain Reports of Fibromyalgia Sufferers

by

Mary Donaldson M.Ed.¹, Horst Mueller Ph.D.², Stuart Donaldson Ph.D.^{1, 4},
and Gabriel Sella M.D.³

1. Myosymmetries Calgary
2. Myosymmetries Edmonton
3. Martins Ferry, Ohio
4. Please address all correspondence to this author at Suite #300, 290 Midpark Way SE, Calgary, Alberta T2X 1P1 or email myo@cadvision.com

Abstract

Forty carefully screened fibromyalgia sufferers were tested with pain measures (MPQ and VAS), a psychological test (SCL 90-R) and QEEG. Analysis of the SCL 90-R showed the presence of 3 distinct groups reflected in the elevation of the Global Severity Index (GSI) scale. The MPQ pain report varied with the GSI scale groups ($p < .01$). The QEEG data did not differentiate fibromyalgia sufferers from the norm when analyzed as a whole. However, when covaried with the GSI scale, the QEEG data showed different patterns of relative power Z-scores for each group. Theta activity was significantly ($p < .03$) elevated for the most severely distressed group. Pearson correlation between Alpha and Theta was also significant ($p < .01$) in a negative direction for the 3 groups. The discussion reviews the implications of the SCL 90 - R, and QEEG results and concludes with a discussion of treatment with medications.

Key words: QEEG, psychological status, pain, fibromyalgia, SCL 90-R, medications

QEEG Patterns, Psychological Status, and Pain Reports of Fibromyalgia Sufferers

Mary Donaldson M.Ed., Stuart Donaldson Ph.D., Horst Mueller Ph.D.,
and Gabriel Sella M.D.

The primary feature of fibromyalgia (FM) is chronic widespread musculoskeletal pain with the finding of numerous “tender points” throughout the body that are excessively tender to digital palpation. The American College of Rheumatology (ACR) has defined 18 tender point locations and set a criterion of pain occurring with digital palpation of less than 4.0 kg/cm² of pressure at a minimum of 11 of these sites for inclusion into this diagnostic category (93). More recently, the construct of Fibromyalgia Syndrome (FMS) has been proposed to include the core symptoms of chronic widespread pain and allodynia as specified in the ACR 1990 definition but also the presence of disturbed and non-restorative sleep, persistent fatigue, and subtle cognitive dysfunctions (i.e., “fibro fog”); including problems of concentration, decreased immediate recall and difficulty with multi-tasking.

The pathogenesis of fibromyalgia tender points and the associated symptoms of FMS have been subjected to extensive investigation over the last two decades with inconclusive results. Early reports of findings supporting a primary peripheral abnormality as causal in the development of FMS have failed to be replicated (80) and the evidence for postulated psychogenic causes (6, 19, 38, 39, 70, 94, 95) remains inconclusive (2, 7, 90). Other researchers have focused their efforts primarily on a search for systemic factors such as: central neurotransmitter imbalances (71, 73), neuroendocrine-immune dysfunction (9, 13, 62, 63), thyroid hormone resistance (49, 50), stress-related physiological changes (33), and sleep disturbance (Alpha intrusion) (57, 58, 59, 69), and muscle dysfunction (20, 21, 22, 43). Although the presence of fibromyalgia tender points has been found to correlate with depression, fatigue, anxiety, and somatic symptoms as well as the presence of widespread pain itself (15, 92), the etiopathogenesis of FMS tender points remains largely a mystery.

More recently attention has focused upon dysfunctional activity in the central nervous system (CNS) as a possible source or cause of the pain amplification and poor pain localization that is so apparent in FMS (24, 26, 27, 56). Flor and Turk (27) stated that “chronic pain patients display increased perceptual and pain sensitivity that is mirrored in cortical hyperreactivity to both sub- and suprathreshold painful stimulation... It is important, therefore, to recognize that chronic pain may lead to massive plastic changes in spinal and supraspinal mechanisms related to the processing of non-nociceptive and nociceptive information. These changes may induce a type of processing of nociceptive and non-nociceptive information that is quite different from that of a person without chronic pain.” (pp. 74-75).

The background electrical activity of the brain reflects states of attention, arousal, sleep, narcosis, and various mental processes (10, 34) and may be easily studied as in the recorded spontaneous electroencephalograph (EEG). Quantitative EEG (QEEG) utilizes Fast Fourier Transform (FFT) techniques to break down complex brain waves into their constituent components. Examination of how the different frequencies compare in power or amplitude at various sites over the brain cortex is commonly conducted, as well as the measurement of other aspects of the brain wave (e.g., coherence, phase, and asymmetry) (87). The QEEG recorded from an individual may be examined against a QEEG database allowing for conversion into standard Z-scores for comparison purposes (86).

Examinations of the spectral or quantitative electroencephalogram (QEEG) as a marker of functional CNS differences between persons with FMS and others has led to a number of interesting preliminary findings. Flor-Henry (28) has indicated that there is a significant change in brain wave activity that differentiates FMS sufferers from normal controls during mental tasks. Two of the present authors (21, 61) have also reported finding significant increases in slow wave activity frontally in clinical samples of FMS patients, which appeared associated with patients' complaints of increased pain sensitivity and cognitive dysfunction. Mueller (61) also reported that reductions in excessive slow wave EEG power from primarily somatosensory and frontal cortex following neurotherapy was associated with improvement in the neurosomatic symptoms of their FMS patients.

Chronic Fatigue Syndrome (CFS) sufferers (a diagnostic group that overlaps substantially with FMS) (31), when studied with QEEG, appear to show similar patterns of brain wave activity, particularly elevations of slow wave Delta and Theta activity and a reduction of Beta (2, 29, 42, 65, 66, 85).

With few exceptions, (83), the QEEG studies of FMS and CFS samples would appear to be in agreement that these disorders are characterized by significantly abnormal QEEG brain maps that show increased amplitudes of slow Delta and/or Theta activity. There is less agreement with respect to changes in Beta activity.

Corroborating the above studies are findings from the use of such techniques as single photon emission tomography (SPECT) and positron emission tomography (PET), which examine brain metabolism by measuring regional cerebral blood flow (rCBF) or glucose uptake in brain tissue. Compared to healthy individuals, persons with FMS appear to be characterized by differences in metabolism in several brain structures— i.e., thalamus, caudate nucleus, cingulate gyrus, inferior pontine tegmentum, lentiform nucleus— involved in pain processing and modulation both at rest and during experimental pain induction (3, 8, 9, 29, 33, 44, 45, 48, 60). In summary, reduced metabolism in the thalamus appears to be the most consistent finding of SPECT/PET studies of persons with FMS. However, these findings may not be entirely unique to FMS, as various

researchers (36, 41, 44, 45, 77) have demonstrated reduced thalamic metabolism with various medical conditions.

Although recent SPECT/PET studies (45, 60) have failed to observe any significant relationships between brain metabolism and psychological measures of depression and anxiety in their FMS or control samples, a strong association between psychological dysfunction and FMS has commonly been reported (4, 37) and needs further investigation.

The above literature suggests changes in brain electrical activity and metabolism may be associated with FMS. However, these findings are not clear and must be treated as preliminary. It also remains unclear to what extent psychological dysfunction plays a role in FMS. The purpose of this study was to further examine the QEEG brain wave activity of FMS sufferers and investigate the relationship of QEEG activity to psychological status and subjective pain report.

METHOD

Subjects

The subjects were volunteers, who were recruited by advertisements in a local paper and by word of mouth. A total of 316 individuals applied for information packages of which 76 were completed and returned. No attempt was made to inquire as to the reasons for such a high non-response rate. Medical specialists (i.e., rheumatologist, internist) prior to the study had diagnosed all 76 individuals as suffering from fibromyalgia. Of these, a further 29 were rejected because they were taking prescribed medications that were known to affect EEG and which they could not stop taking for reasons of unacceptable medical risk, and 1 dropped out due to distance from the study center. Demographic information was collected on the remaining 46 subjects.

A dolorimeter evaluation as recommended by Fischer (25) was administered to all subjects according to the criteria of the American College of Rheumatology (ACR) (93). Two individuals did not meet the criteria of 11 of 18 tender points positive for pain at pressures of less than 4.0 kg/cm² and were disqualified from the study. A board-certified specialist in hematology and internal medicine screened the remaining 44 subjects for concurrent and secondary diseases (e.g., rheumatoid arthritis, lupus). Thirty blood measures; urine and stool samples were collected. Another 4 individuals were disqualified at this point, 2 because they would not complete the medical examinations and 2 on the basis of having active infections or diseases, which could affect the EEG measurements (89). The demographic data from the 6 subjects who did not pass this phase of the screening were dropped from the data pool.

The remaining 40 subjects were assessed using the following psychometric instruments: *Symptom Checklist-90-Revised* (SCL-90-R) (16), *McGill Pain Questionnaire* (MPQ) (54), *Visual Analogue Scale* (VAS) (40) of subjective pain intensity, and a *Memory Assessment Scale* (MAS) (91). All testing was conducted following standard procedures. The same examiner verbally instructed the subjects in the standard procedures as outlined in the various testing manuals. The subjects were left alone to complete the questionnaires except for the MAS. A psychologist trained in test administration administered the MAS.

Within one week of completing the written tests, all 40 subjects underwent a full-cap QEEG evaluation. The QEEG was administered in a standard fashion to all subjects. All subjects were required to be off their medications for a minimum of 5 elimination half-lives prior to their QEEG assessment. (Note: an elimination half-life is the average time for a single dose of medication to be 50% inactivated or eliminated from the body; with each subsequent half-life another half of the remaining drug is inactivated or eliminated until, by the passing of five half-lives, fully 97% of the drug has been deactivated or eliminated from the body.) Prior to the day of the assessment all subjects were instructed to wash their hair twice with a non-conditioning shampoo, and not to apply any conditioner, mousse, or hair spray products after washing. All QEEG assessments were conducted between the hours of 10:00 AM and 2:00 PM, with all subjects seated and fully supported in a recliner chair. All subjects were fully informed as to the nature of the assessment and signed a consent form. Data were collected from 19 sites using an ECI electro-cap, with electrodes located in the Standard International 10/20 locations. Impedance was checked at each site, with levels of resistance below five K ohms established before data collection. Standardized instructions were given to all subjects for each condition. They were instructed to advise the examiner if they became fatigued and a break was given. Data were collected over 4 conditions: eyes open, 2 eyes closed, and serial 7s with eyes closed. The order of presentation was randomized to control for any order effect.

Data were collected using the Lexicor^a NeuroSearch 24 System, sampling at a rate of 128 samples per second with a gain of 32 K. A montage reference was utilized from both earlobes with 4 SEMG channels monitoring for muscle artifact. These SEMG electrodes were located 1 cm out from the lateral aspect of the cornea, 4 cm down from the zygomatic bone and 2 cm anterior to the ear, 4 cm above T1, and 2 cm to the left and over the strongest felt pulse over the carotid artery.

Data Analysis

QEEG: The EEG frequencies were grouped together following standard nomenclature into bands with Delta from 0.5 to 3.5 Hz, Theta 3.5 – 7.5 Hz, Alpha 7.5 – 13 Hz, and Beta 13 Hz to 22 Hz. The relative power of each band for each site for each subject was

neurometrically normalized using the *Lifespan Reference EEG Database* (86) producing a 4 x 19 matrix of Z-scores for each of the 40 subjects. This procedure, called Significance Probability Mapping (SPM) (23), avoids the problem of having to find, match, and assess a normal control group and demonstrates good temporal stability (53, 64).

As the *Lifespan Reference EEG Database* gives norms for the eyes closed condition only, the eyes open data was not included for analysis. The database also offers no norms for active tasks (i.e., mental arithmetic) so the serial 7s data were examined using the eyes closed norms, as this task was conducted with the eyes closed.

First the two separate sets of resting eyes closed data were examined for consistency of variation. This measure reported in percentage compares the first set of data to the second set examining the frequencies for the coefficient of variation. A high percent score (coefficient of variation) suggests consistency of data or decreased artifact, while a low percent score suggests the presence of artifact. The percent values showed scores above 90% for all frequencies.

Second, given the very high similarity between the two sets of resting eyes closed obtained data, these two recordings for each subject were averaged to produce a single Z-score for eyes closed for each of the 19 recording sites for each of the 4 frequency bands. These derived Z-scores were then averaged across the 40 subjects.

Psychological Tests: All psychological tests were scored following the recommended procedures in the various test administrative manuals.

SCL 90-R: The *Symptom Checklist 90-R* is a 90-item, norm-referenced, self-report symptom inventory designed to reflect the psychological symptom patterns of community, medical, and psychiatric respondents. Each item is rated on a 5-point scale of distress (0-4) ranging from “Not at All” to “Extremely” distressing. The SCL 90-R is scored and interpreted in terms of nine primary symptom dimensions and three global indices of distress. Respondent’s raw scores for each of the primary scales and global indices are converted to *T*-scores with a mean of 50 and standard deviation of 10 based on a set of published norms. For the purposes of this study, only the SCL 90-R *Global Severity Index* (GSI) was selected for examination because this score is considered the best single indicator of the current level or depth of psychological distress (16).

The obtained GSI *T*-scores were grouped by standard deviations above the mean of 50. Those individuals who scored in the 50-59 range (50-83%tile) were assigned to Group #1, those who scored in 60-69 range (84-97%tile) were assigned to Group #2, and those scoring in the 70 - 80 range (> 97%tile) were assigned to Group #3 for data analysis.

Pain Scores: The *McGill Pain Questionnaire* (MPQ) (54) has become one of the most widely used tests for the measurement of pain (55). The MPQ was designed to provide a quantitative profile of three aspects of experienced pain: sensory-discriminative, motivational-affective, and cognitive-evaluative. For the purposes of this study, the MPQ was scored as recommended by Reading (68) with the component scores summated to arrive at a Total Score.

Visual analog scales (VAS) provide simple, efficient, and minimally intrusive measures of pain intensity in clinical and research settings where a quick index of subjective pain is required and to which a numerical value can be assigned (55). The VAS used in this study consists of a 10-centimeter horizontal line with two endpoints labeled “no pain” and “worst pain ever” (40). The subject is required to place a mark on the 10-centimeter line at a point that corresponds to the level of pain intensity he/she presently feels. The distance, in centimeters from the low end of the VAS to the subject’s mark is used as the numerical index of the severity of experienced pain. For this study, the mark on the VAS of subjective pain intensity was measured to the nearest millimeter (1/10th of a centimeter) and recorded as centimeters to one decimal place.

Finally, the scores on the MAS were calculated according to the instruction manual (91) with results reported in percentages.

Statistical Analysis

All statistical analyses were performed using SPSS software and assumed data to be normally distributed. Obtained statistical probability values were not corrected for multiple comparisons and, although a highly conservative alpha-level might have been selected for the study to counter possible increased Type I error due to multiple comparisons, it was decided to set the study alpha-level at 0.05 because of the small sample size and exploratory nature of the study. The small sample size will unfortunately reduce the power of the statistical analyses and increase the probability of Type II error. With a one-sided *t*-test and Type I error set at 5%, a sample size of 40 subjects is sufficient to detect a standardized effect of 0.82 with a power of 80%, which is considered moderate. But in multiple group analyses using *F*-tests in which individual groups consist of 10 or less subjects, the power to detect a standardized effect of 0.90 drops to slightly less than 50%. In this case, a standardized effect of 2.2 can be detected with 80% power.

RESULTS

Demographics

The demographic results indicated that the subjects were predominately female ($n = 37$), average age of 44.3 ($SD = 8.2$) years, right-handed ($n = 38$), and had been in pain for 8.8 ($SD = 7.5$) years.

Psychological Results

The *SCL 90-R Global Severity Index* (GSI) showed that 7 (17.5%) individuals had a *T*-score between 70 and 80 (Group #3), 24 (60%) had a score between 60 and 69 (Group #2), and 9 (22.5%) had a score between 50 and 59 (group #1). A One-Way ANOVA was significant ($F = 111.46, p < .001$) for between group differences. The overall mean GSI *T*-score was 65.9 ($SD = 6.9$) suggesting a moderate level (approximately 86%tile) of psychological distress for the group as a whole. The average scores and number of subjects in each cell are given in Table 1 below.

The relative contribution of the *SCL 90-R*'s nine clinical scales (e.g., Somatization, Obsessive-Compulsive, Depression, Anxiety, etc.) to the GSI score was examined by regressing all 9 clinical scales against the GSI. These results showed that for the FMS sample the Somatization Scale ($t = 3.211, p < .003$), and Depression Scale ($t = 4.321, p < .001$) both significantly contributed to the GSI score. The Hostility Scale ($t = 1.787, p < .09$), Obsessive-Compulsive Scale ($t = 1.94, p < .07$), and Psychoticism Scale ($t = 1.974, p < .06$) all approached significance while the remaining scales showed no significant contributions.

The subjects reported a mean Total Score of 34.1 ($SD = 12.7$) on the *McGill Pain Questionnaire*, an average score of 5.5 ($SD = 2.1$) on the *VAS-Pain*, and an average score of 95.7% ($SD = 13.5$) on the *MAS Global Memory Scale*. Using the *SCL 90-R* GSI-level groups as the independent variable, a One-Way ANOVA was performed on the pain scores (MPQ and VAS) and MAS scores examining for a relationship amongst the variables. Significant results were obtained for the MPQ ($F = 5.173, p < .01$) with the VAS approaching significance ($F = 1.778, p < .183$). The MAS scores showed no significance. Examination of the MPQ subscales using a One-Way ANOVA showed all 3 subscales significantly related to psychological group (Sensory $F = 3.341, p < .05$, Affective $F = 4.313, p < .02$, Evaluative $F = 6.978, p < .01$). This suggests that psychological status has a significant relationship to reported pain for FMS.

These scores are presented for the entire subject group, as well as the three GSI-level groups, in Table 1 below.

<Insert Table 1 about here>

QEEG Results: The average *Z*-scores for each QEEG frequency band and task are reported in Table 2 below. As expected, the scores clustered around zero with Beta showing the greatest deviation in a positive direction.

<Insert Table 2 about here>

Pearson correlational analysis of the above scores showed significant correlation occurring between the eyes closed and serial 7s states with an r -value of 0.956. In view of the fact that the two data sets were over 90% similar, no further analysis was conducted of the serial 7s data.

In view of the results using the SCL 90-R GSI scores as reported above, a similar analysis was conducted on the QEEG data examining each group's Z -scores. The QEEG scores were examined against the GSI (SCL 90-R) group scores using One-Way ANOVA procedures. The results showed a significant relationship between the QEEG scores and the GSI groupings for Theta ($F = 3.840, p < .031$). The results for all groups and all frequencies are reported in Table 3.

<Insert Table 3 about here>

As may be seen, certain patterns emerge from the data. Alpha decreases across groups, decreasing as psychological distress increases. Beta shows increased activity for Group 2 as compared to the other groups. Delta is relatively decreased for all groups. Theta shows an inverse relationship to Alpha, increasing as distress increases. Alpha power in the spectral EEG of these 40 FMS subjects is negatively correlated ($r = -.583$) with Theta power over the cortex.

The individual clinical scales of the SCL 90-R were correlated with the QEEG Z -scores using a Pearson one-tailed procedure. Alpha showed significantly negative correlation with the Interpersonal Sensitivity Scale ($r = -.372, p > .009$) and the Phobic Anxiety Scale ($r = -.198, p < .021$). Beta showed no significant correlations. In view of the increased Beta Z -scores for Group 2, a correlational analysis was conducted for this group only with these results significant ($r = .367, p < .031$) for Somatization. Delta was significantly correlated to only one scale, the Interpersonal Sensitivity Scale ($r = .367, p < .01$). Theta was significantly related to 7 scales including: 1) Obsessive-Compulsive Scale ($r = .266, p < .05$), 2) Depression Scale ($r = .327, p < .02$), 3) Hostility Scale ($r = .394, p < .006$), 4) Psychotism Scale ($r = .353, p < .013$), 5) Interpersonal Sensitivity Scale ($r = .479, p < .001$), 6) Anxiety Scale ($r = .310, p < .026$) and 7) Paranoid Ideation Scale ($r = .285, p < .037$).

Finally a Pearson one-tailed procedure was run comparing the Theta Z -scores to the scores from the MPQ and VAS. Only the MPQ scores showed a significant relationship ($r = .290, p < .035$).

DISCUSSION

The results of this study suggest that a carefully selected group of symptomatic FMS subjects fail to show a characteristic abnormal QEEG pattern either at rest with eyes

closed or during an active cognitive task with eyes closed. Although the QEEG of many of our subjects may have differed from the QEEG norms for their gender and age, when the data were collapsed, these individual differences cancelled each other out and no characteristic pattern for the group was apparent. This lack of a characteristic group pattern was equally apparent for both the eyes open resting condition and the cognitively active serial 7s-task condition

However, when the FMS subjects were divided into three separate groups on the basis of each subject's level of psychological distress as indexed by the SCL 90-R GSI score, significant group differences in both subjective pain experience and the QEEG readily became apparent. The group showing the *least* amount of psychological distress and experienced pain revealed the *greatest* Alpha power in the resting EEG and relatively little Theta. The group showing the *greatest* amount of psychological distress and experienced pain revealed the greatest Theta power and relatively little Alpha. Compared to normative values, Delta was decreased across all groups, while Beta was increased for only the second group (i.e., mild to moderate elevated distress). Theta power was positively correlated with both SCL 90-R GSI scores and the MPQ total scores, the first a measure of broad psychological distress with respect to symptoms and the second a measure of the psychological experience of pain. The positive correlation between Theta power and VAS pain intensity (a subjective sensory experience of pain) only approached significance. This pattern of results suggests there is a greater involvement with the affective or emotional components of pain as opposed to the purely sensory components in the fibromyalgia population— a finding that is consistent with the chronic pain literature generally.

These results also suggest that the Alpha/Theta power balance in the EEG reflects the level of psychological distress due to experienced pain as opposed to the pain experience itself. The GSI score for the FMS subjects in the current study was most strongly influenced by their scores on SCL 90-R clinical scales of Somatization, Depression, Psychoticism, and Obsessive-Compulsive. High scorers on these scales are generally expressing a greater degree of current distress over physical symptoms of the body, depressed mood, feelings of alienation from others, and cognitive inefficiency— all common complaints of fibromyalgia patients. Apparently, the more severely the fibromyalgia symptoms are experienced, the greater the shift from Alpha to Theta in the EEG. The broadly decreased Delta as found across all groups would appear to be associated with the problems of reduced deep restorative sleep as commonly reported by this group.

The negative relationship between Alpha and Theta power was quite robust; suggesting that greater psychological distress is associated with a slowing of the EEG from Alpha to Theta. Increased Theta power is associated with decreased vigilance and reduced

cognitive efficiency. Thus the rise in Theta power associated with pain may be a protective response of the brain. Enhanced slow wave activity in the Theta band may indicate an active reaction to lower the perception of pain through a self-induced hypnosis-like state (46). It is well known that the enhancement of Theta power is strongly predictive of the depth of sedation and duration of unconsciousness whereas changes in the Alpha rhythm parallel changes in cardiovascular variables such as blood pressure, heart rate, and cerebral blood flow (10, 11).

There appears to be differences in the way the brain reacts to acute and chronic pain. It is well known that effects of a painful stimulus on spontaneous EEG are seen in a large increase in Delta-band power with relatively smaller increases in Theta and Beta activity together with some decrease or increase in Alpha activity (10, 11). Tonic muscle pain in particular, has been found to be associated with a significant and long-lasting increase of Delta power and low Alpha power over the contralateral parietal locus (47). Moreover, effective analgesia is associated with a decrease in Delta and Theta EEG power whereas sedation is associated with reductions in Alpha power (10). Chen (12) concludes that EEG spectral power of low frequencies in Delta and Theta bands is most often found to be associated with human pain. While appearing to be contradictory to the present findings these studies were focused on acute pain, suggesting that the acute pain populace may differ from the chronic pain populace as seen in the changes in the EEG activity. The possible differences between acute and chronic pain brain wave activity needs more research.

Chronic pain states have also been associated with decreased thalamic blood flow (rCBF) whereas acute pain states have been associated with increased thalamic rCBF (44, 45). The reason for this difference is postulated to be an increasing disinhibition of the medial thalamus with chronic pain that results in activation of the limbic network and various brain structures involved in cortical pain processing such as the prefrontal cortex, amygdala, and locus ceruleus to which the medial thalamus is linked. Positron emission tomography (PET) has also revealed a similar pattern of decreased thalamic activity with chronic neuropathic pain (36, 41). Moreover, as the intensity of experienced pain increases, there is increasing reduction in caudate rCBF together with an increase in rCBF in the anterior cingulate gyrus (77). A part of the limbic system, the anterior cingulate gyrus is known to be involved in the integration of affect, cognition and motor response aspects of pain (18) and exhibits increased activity in chronic pain patients (36). Certainly there is evidence that the affective response to pain is encoded in the anterior cingulate (67).

The limbic system is a high-order functional regulator in the brain; strongly influencing brain homeostasis. It plays a primary role in human emotional and behavioural responses to pain as well as stability of body temperature, mood, appetite, and sleep; sympathetic

nervous system function; immune system efficiency; control of hormonal balance; and the selection of adaptive responses to stress. Perhaps these alterations in brain metabolism are reflected in the shift in the EEG activity and the increasing negative affect or “suffering” that comes with chronic pain.

Our QEEG findings and the SPECT/PET findings of increased activation of the limbic network with chronic pain are not inconsistent with research findings implicating neurotransmitter and neurohormonal imbalances in the pathogenesis of FMS (62, 71, 73). In this regard, serotonin (5-hydroxytryptamine, 5-HT) metabolism has been implicated in the pathogenesis of FMS and many of the symptoms of the syndrome with findings of lower than normal levels of tryptophan (TRP)— the amino-acid precursor of serotonin— in plasma; 5-HT in serum and plasma, and 5-hydroxyindole acetic acid (5HIAA)— the end product of 5HT metabolism— in cerebral spinal fluid (CSF) of FMS patients (73).

Serotonin has been shown to influence a broad range of physiological systems such as cardiovascular regulation, respiration, and thermoregulation as well as a variety of behavioural functions, including circadian rhythm entrainment, sleep-wake cycle, appetite, aggression, sexual behaviour, sensorimotor reactivity, pain sensitivity, and learning (51). Most 5-HT-containing neurons are localized along the midline of the brain stem and send long axons to innervate a wide distribution of receiving areas throughout the nervous system from the cortex to the spinal cord (51). In the brain and spinal cord, 5HT-containing neurons are known to play an important role in modulating pain sensitivity and response through their effect on inhibiting the release of substance P, a highly potent mediator of pain, that has been found in higher than normal concentrations in both the CSF and the perivascular of skeletal muscle of FMS patients (1, 74, 79, 82, 88). One hypothesized connection between the low serotonin levels and high substance P levels frequently found in FMS is that too much serotonin is bound to 5-hydroxytryptamine-3 (5-HT₃) receptors which, in turn, leads to the release of substance P (84). Research examining the effects of 5-HT₃ receptor antagonists on nociception holds some real promise in the treatment of FMS (32, 84).

Animal research has demonstrated a dose-dependent increase in the sensitivity of dorsal horn neurons, an increase in the size or number of mechanosensitive receptive fields in nociceptive neurons, and a lowering of the threshold for post-synaptic potentials, all consistent with facilitation of nociception by SP (72). Alberts, et al. (1) have shown that higher CSF levels of SP in persons with FMS correlate with a decrease in blood flow within the caudate nucleus and thalamus. As well, acute tryptophan depletion results in a corresponding reduction in caudate blood flow (81).

Differences in the results of the present study versus those of previous studies may be due to several different factors such as differences in EEG sampling techniques and conditions under which the EEG was examined. Those research subjects who were

unwilling or unable to stop their medications prior to their QEEG were not included. In the authors' earlier studies of clinical patients, they were generally encouraged to reduce or go off their medications prior to the QEEG but those who did not were still included. It is likely that those FMS patients who cannot reduce their medication use prior to assessment are different in a number of ways from those who can. In particular, those who cannot manage without their medications for even a few days are likely to be more chronic with more severe symptoms or have problems of drug dependency.

In the current study, the subjects were very carefully selected to ensure that they fully met all ACR 1990 criteria for classification as fibromyalgic as well as not having any co-morbid medical conditions or active infections or viral diseases. Of particular importance is to control for viral or other infections (e.g., CBC white cell counts within normal limits) as such infections are known to elevate Theta-band EEG activity (89). This also points to the need to control for co-morbid conditions (i.e., lupus, clinical depression, etc.) as such conditions may also alter EEG activity. This highlights the problem of comparing fibromyalgia as the only dysfunction versus fibromyalgia as secondary to, or associated with, other pathologies.

Another important aspect of the current study was its implementation of controls for the use of psychotropic medications that alter the EEG. Certainly, the sedative antidepressants (e.g., amitriptyline, imipramine) so commonly prescribed for fibromyalgia are known to significantly increase both Delta (primarily posteriorly) and Theta (primarily anteriorly) EEG power (Theta > Delta) while attenuating Alpha (76). Desipramine-type antidepressants only augment Alpha, whereas the newer selective serotonin reuptake inhibitors appear to primarily enhance fast Beta frequencies without significantly changing slow wave activity (75, 76). Hypnotics increase both Delta and Theta (Delta > Theta) as well as slow and fast Beta activity while greatly decreasing Alpha (75). Anxiolytics such as Valium, attenuate Alpha and increase all Beta activity (35). Analgesic medications, both narcotic and non-narcotic agents, increase Delta and slow and fast Beta activity while strongly attenuating Alpha (35). Even the peripherally acting analgesics such as the commonly prescribed NSAIDs have central effects (78). Specifically, analgesic efficacy is positively related (approximately $r = 0.94$) to reduction of the Delta power of pain-related cerebral potentials; with NSAIDs showing relatively small effects on Delta and opioids showing marked effects on Delta (78).

The results need to be regarded with caution. The number of statistical tests conducted may lead to an increased risk of Type I error. Conversely the limited sample size may lead to an increased risk of missing trends that a larger sample would demonstrate. This study needs to be replicated with a larger sample size.

The present study presents several novel and important results that contribute to the clinical appreciation of fibromyalgia. Other studies utilizing EP, PET, SPECT techniques

supports the theory that fibromyalgia may be a disorder marked by significant changes in brain function. The QEEG data appears to take this a step further suggesting that the psychological status of fibromyalgia sufferers effect brain wave patterns and pain responses. This suggests the presence of subgroups in this populace. Hopefully this study will lead to more research and increased success particularly in using medications to treat this difficult condition.

REFERENCES

1. Alberts, K., Bradley, L., Alarcon, G., Alexander, M., Mountz, J. Weigert, D., Liu, H., Blalock, J., Aaron, L., Alexander, R., San Pedro, E., Martin, Y., Morell, A. (1996). Abnormal brain regional cerebral blood flow (rCBF) and cerebrospinal fluid (CSF) levels of substance P (SP) in patients and non-patients with fibromyalgia (FM). *Arthritis & Rheumatism*, 39(Suppl): S12 [abstract].
2. Aaron, L., Bradley, L., Alarcon, G., Alexander, R., Triana-Alexander, M., Martin, M. (1996). Psychiatric diagnoses in patients with fibromyalgia are related to health care-seeking behavior rather than to illness. *Arthritis & Rheumatism*, 39: 436-445.
3. Apkarian, A., Thomas, P., Krauss, B., Szeverenyi, N. (2001). Prefrontal cortical hyperactivity in patients with sympathetically mediated chronic pain. *Neuroscience Letters*, 311(3), 193-197.
4. Benjamin, S., Morris, S., McBeth, J., MacFarland, G., Silman, A. (2000). The association between chronic widespread pain and mental disorder: A population study. *Arthritis & Rheumatism*, 43: 561-567.
5. Billiot, K., Budzynski, T., Andrasik, F. (1997). EEG patterns and chronic fatigue syndrome. *Journal of Neurotherapy*, 2(2): 20-30.
6. Boissevain, M., McCain, G. (1991). Toward an integrated understanding of fibromyalgia syndrome: II. Psychological and phenomenological aspects. *Pain* 45: 239-248.
7. Bradley, L. (1998). Fibromyalgia: A model for chronic pain. *Journal of Musculoskeletal Pain*, 6(3): 19-27.
8. Bradley, L.A., McKendree-Smith, N.L., Alberts, K.R., Alarcon, G.S., Mountz, J.M., Deutsch, G. (2000). Use of neuroimaging to understand abnormal pain sensitivity in fibromyalgia. *Current Rheumatology Reports*, 2(2): 141-148.
9. Bradley, L., Sotolongo, A., Alberts, K., Alarcon, G., Mountz, J., Liu, H-G., Kersh, B., Domino, M., DeWaal, D., Weigert, D., Blalock, J. (1999). Abnormal regional cerebral blood flow in the caudate nucleus among fibromyalgia patients and non-patients is associated with insidious symptom onset. *Journal of Musculoskeletal Pain* 7(1/2): 285-292.
10. Bromm, B. (1995). Consciousness, pain, and cortical activity. In B. Bromm & J. Desmedt (Eds), *Pain and the Brain: From Nociception to Cognition*, Chapter 3.

- (pp.35-59). *Advances in Pain Research and Therapy, Volume 22*. New York, NY: Raven Press.
11. Bromm, B., Lorenz, J. (1998). Neurophysiological evaluation of pain. *Electroencephalography & Clinical Neurophysiology*, 107: 227-253.
 12. Chen, A.C.N., (1993). Human Brain Measures of Clinical Pain: A Review. *Pain* 54:115-122.
 13. Cianfrini, L., McKendree-Smith, N., Bradley, L., Alarcon, G., Deutsch, G., Sotolongo, A., Kersh, B., Liu, H-G., Mountz, J., Powell, T. (2001). Pain sensitivity and bilateral activation of brain structures during pressure stimulation of patients with fibromyalgia (FM) is not mediated by major depression (DEP). *Arthritis & Rheumatism*, 44(9): S395.
 14. Crofford, L., Demitrack, M. (1996). Evidence that abnormalities of central neurohormonal systems are key to understanding fibromyalgia and chronic fatigue syndrome. *Rheumatic Diseases Clinics of North America* 22: 267-284.
 15. Croft, P., Schollum, J., Silman, A. (1994). Population study of tender point counts and pain as evidence of fibromyalgia. *British Medical Journal*, 309: 696.
 16. Derogatis, L. (1994). *SCL-90-R: Administration, scoring and procedures manual (3rd Ed)*, Clinical Psychometric Research.
 17. Dessein, P., Shipton, E., Joffe, B., Hadebe, D., Stanwix, A., Van der Merwe, B. (1999). Hyposecretion of adrenal androgens and the relation of serum adrenal steroids, serotonin and insulin-like growth factor-1 to clinical features in women with fibromyalgia. *Pain* 83(2): 313-319.
 18. Devinsky, O. (1995). Contributions of the anterior cingulate to behaviour. *Brain*, 118: 279.
 19. Diamond, H. (1984). Psychogenic rheumatism. *Clinics of Rheumatological Practice* 2: 131-133.
 20. Donaldson, S., MacInnis, A., Snelling, L., Sella, G., & Mueller, H. (2002). Characteristics of diffuse muscle coactivation (DMC) in fibromyalgia sufferers- Part 2. *Neurorehabilitation*. Vol 17 (No.1)
 21. Donaldson, S., Sella, G., Mueller, H. (1998). Fibromyalgia: A retrospective study of 252 consecutive referrals. *Canadian Journal of Clinical Medicine* 5(6): 116-127.

22. Donaldson, S., Snelling, L., MacInnis, A., Sella, G., Mueller, H. (2002). Diffuse muscular coactivation (DMC) as a potential source of pain in fibromyalgia - Part 1. *Neurorehabilitation*. Vol. 17 (No.1).
23. Duffy, F., Bartels, P., Burchfiel, J. (1981). Significance probability mapping: An aid in the topographic analysis of brain electrical activity mapping. *Electroencephalography & Clinical Neurophysiology*, 51: 455-462.
24. Elbert, T., Flor, H., Birbaumer, N., Knecht, S., Hampson, S., Larbig, W., Taub, E. (1994). Extensive reorganization of the somatosensory cortex in adult humans after nervous system injury. *NeuroReport*, 5: 2593-2597.
25. Fischer, A. (1998). Algometry in diagnosis of musculoskeletal pain and evaluation of treatment outcome: An update. *Journal of Musculoskeletal Pain* 6(1): 5-32.
26. Flor, H., Braun, C., Elbert, T., Birbaumer, N. (1997). Extensive reorganization of primary somatosensory cortex in chronic back pain patients. *Neuroscience Letters*, 224: 5-8.
27. Flor, H., Turk, D. (1996). Integrating central and peripheral mechanisms in chronic muscular pain. *Pain Forum*, 5(1): 74-76.
28. Flor-Henry, P. (1999). Personal communication with regard to the findings of his recent and as yet unpublished QEEG brain mapping study of fibromyalgia syndrome, chronic fatigue syndrome, and matched normal controls.
29. Garloch, K. (1994). Chronic fatigue syndrome treatment patented. *CFIDS Chronicle*, Spring: 7-8.
30. Gibson, S., Littlejohn, G., Gorman, M., Helme, R., Granges, G. (1994). Altered heat pain thresholds and cerebral event-related potentials following painful CO₂ laser stimulation in subjects with fibromyalgia syndrome. *Pain*, 58: 185.
31. Goldenberg, D., Simms, R., Geiger, A., Komaroff, A. (1990). High frequency of fibromyalgia in patients with chronic fatigue syndrome seen in a primary care practice. *Arthritis & Rheumatism*, 33: 381-387.
32. Greenshaw, A. (1993). Behavioural pharmacology of 5-HT₃ receptor antagonists: A critical update on therapeutic potential. *Trends in Pharmacological Sciences*, 14: 265-269.
33. Griep, E., Boersma, J., de Kloet, R. (1993). Altered reactivity of the hypothalamic-pituitary-adrenal axis of the primary fibromyalgia syndrome. *The Journal of Rheumatology*, 20(3): 469-474.

34. Harmon-Jones, E., Allen, J. (1997). Behavioral activation sensitivity and resting frontal EEG asymmetry: Covariation of putative indicators related to risk for mood disorders. *Journal of Abnormal Psychology*, 106 (1): 159-163.
35. Herrmann, W.M. (1995). The pharmaco-electroencephalogram of analgesic and psychotropic drugs. In B. Bromm & J. E. Desmedt, (Eds.), *Advances in Pain Research and Therapy, Volume 22*. (pp. 459-471). New York, NY: Raven Press.
36. Hsieh, J., Belfrage, M., Stone-Elander, S., Hansson, P., Ingvar, M. (1995). Central representation of chronic ongoing neuropathic pain studied by positron emission tomography. *Pain*, 63: 225-229.
37. Hudson, J., Goldenberg, D., Pope, H., Keck, P., Schlesinger, L. (1992). Comorbidity of fibromyalgia with medical and psychiatric disorders. *American Journal of Medicine*, 92:363-367.
38. Hudson, J., Hudson, M., Pliner, L., et al. (1985). Fibromyalgia and major affective disorder: a controlled phenomenology and family history study. *American Journal of Psychiatry*, 142: 441-446.
39. Hudson, J., Pope, H. (1989). Fibromyalgia and psychopathology: Is fibromyalgia a form of "Affective Spectrum Disorder?" *Journal of Rheumatology*, 16: 15-22.
40. Huskisson, E. (1983). Visual analog scales. In R. Melzack (Ed.), *Pain Measurement and Assessment*. (pp. 33-37). New York, NY: Raven Press.
41. Iadarola, M.J., Max, M.B., Berman, K.F. (1995). Unilateral decrease in thalamic activity observed in positron emission tomography in patients with chronic neuropathic pain. *Pain*, 52: 259.
42. James, L.C., Folen, R.A. (1996). EEG biofeedback as a treatment for chronic fatigue syndrome: A controlled case report. *Behavioral Medicine*, 22: 77-81.
43. Kent-Braun, J., Sharma, K., Weiner, M., Massie, B., Miller, R. (1993). Central basis of muscle fatigue in chronic fatigue syndrome. *Neurology*, 43: 125-131.
44. Kwiatek, R., Barnden, L., Rowe, C., Pile, K. (1997). Pontine tegmental regional cerebral blood flow [rCBF] is reduced in fibromyalgia. *Arthritis & Rheumatism*, 40: S43.
45. Kwiatek, R., Barnden, L., Tedman, R., Jarrett, R., Chew, J., Rowe, C., Pile, K. (2000). Regional cerebral blood flow in fibromyalgia: Single photon emission computed tomography evidence of reduction in the pontine tegmentum and thalami. *Arthritis & Rheumatism*, 43 (12): 2823-2833.

46. Larbig, W., Elbert, T., Lutzenberger, W., Rockstroh, B., Schnerr, G., Birbaumer, N. (1982). EEG and slow brain potentials during anticipation and control of painful stimulation. *Electroencephalography & Clinical Neurophysiology*, 53: 298-309.
47. Le Pera, D., Svensson, P., Valeriani, M., Watanabe, I., Arendt-Nielsen, L., Chen, A. (2000). Long-lasting effect evoked by tonic muscle pain on parietal EEG activity in humans. *Clinical Neurophysiology*, 111(12): 2130-2137.
48. Lorenz, J., Grasedyck, K., Bromm, B. (1996). Middle and long latency somatosensory evoked potentials after painful laser stimulation in patients with fibromyalgia syndrome. *Electroencephalography & Clinical Neurophysiology*, 100: 165.
49. Lowe, J. (1997). Thyroid status of 38 fibromyalgia patients: Implications for the etiology of fibromyalgia. *Clinical Bulletin of Myofascial Therapy*, 2(1): 36-41.
50. Lowe, J., McCullum, M., Graf, L., Yellin, J. (1997). Mutations in the c-erbA₁ gene: Do they underlie euthyroid fibromyalgia? *Medical Hypotheses* 48(2)Feb: 125-135.
51. Lucki, I. (1998). The spectrum of behaviors influenced by serotonin. *Biological Psychiatry*, 44: 151-162.
52. Mannion, R., Woolf, C.J. (2000). Pain mechanisms and management: A central perspective. *The Clinical Journal of Pain*, 16: S144-S156.
53. McEvoy, L.K., Smith, M.E., Gevins, A. (2000). Test-retest reliability of cognitive EEG. *Clinical Neurophysiology*, 111: 457-463.
54. Melzack, R. (1983). The McGill Pain Questionnaire. In R. Melzack (Ed.), *Pain Measurement and Assessment*. (pp. 41-61). New York, NY: Raven Press.
55. Melzack, R., Katz, J. (1992). The McGill pain Questionnaire: Appraisal and current status. In D. Turk & R. Melzack (Eds.), *Handbook of Pain Assessment*. (pp. 152-168). New York, NY: Guilford Press.
56. Mense, S., Simons, D., Russell, J. (2001). *Muscle Pain: Understanding its Nature, Diagnosis, and Treatment*. Baltimore, MD: Lippincott Williams & Wilkins.
57. Moldofsky, H. (1993). Sleep, neuroimmune and neuroendocrine functions in FMS and chronic fatigue syndrome. *Advances in Neuroimmunology*, 5: 39-56.
58. Moldofsky, H., Lue, F. (1992). Sleep and symptoms in 205 fibromyalgia patients versus 876 patients with various sleep disorders. *Sleep Research*, 2: 303-315.

59. Moldofsky, H., Scarisbrick, P., England, R., Smythe, H. (1975). Musculoskeletal symptoms and non-REM sleep disturbance in patients with fibrositis syndrome and healthy subjects. *Psychosomatic Medicine*, 37: 341-351.
60. Mountz, J.M, Bradley, L.A., Modell, J.G., Alexander, R.W., Triana-Alexander, M., Aaron, L.A., Stewart, K.E., Alarcon, G.S., Mountz, J.D. (1995). Fibromyalgia in Women. Abnormalities of regional cerebral blood flow in the thalamus and caudate nucleus are associated with low pain thresholds. *Arthritis & Rheumatism*, 38: 926-938.
61. Mueller, H., Donaldson, S., Nelson, D., Layman, M. (2001). Treatment of fibromyalgia incorporating EEG-driven stimulation: A clinical outcomes study. *Journal of Clinical Psychology* 57(7): 933-952.
62. Neeck, G., Crofford, L. (2000). Neuroendocrine perturbations in fibromyalgia and chronic fatigue syndrome. *Rheumatic Diseases Clinics of North America*, 26(4): 989-1002.
63. Peter, J., Wallace, D. (1988). Abnormal immune regulation in fibromyalgia. *Arthritis & Rheumatism*, 31:S24.
64. Pollock, V.E., Schneider, L.S., Lyness, S.A. (1991). Reliability of topographic quantitative EEG amplitude in healthy middle-aged and elderly subjects. *Electroencephalography & Clinical Neurophysiology*, 79: 20-26.
65. Preston, M. (1996). The abnormal QEEG showing atypical slowing and spikes. *CFIDS Journal*, Winter 1996.
66. Preston, M. (2000). *Cognitive dysfunction in CFMS and FMS: Assessment and treatment*. Seminar presented at the Harris Methodist Hospital, Bedford, Texas, April 29, 2000.
67. Rainville, P., Duncan, G., Price, D., Carrier, B., Bushnell, M. (1997). Pain affect encoded in human anterior cingulate but not somatosensory cortex. *Science*, 277: 968.
68. Reading, A. (1989). Testing pain mechanisms in persons in pain. In: P.D. wall & R. Melzack (Eds.), *Textbook of Pain, 2nd Edition*. (pp. 269-280). Edinburgh: Churchill Livingstone.
69. Roizenblatt, S., Moldofsky, H., Benedito-Silva, A., Tufik, S. (2001). Alpha sleep characteristics in fibromyalgia. *Arthritis & Rheumatism*, 44(1): 222-230.
70. Russell, A.S. (1995). Fibromyalgia— A historical perspective. *Journal of Musculoskeletal Pain*, 3(2): 43-48.

71. Russell, I. (1995). Neurohormonal: Abnormal laboratory findings related to pain and fatigue in fibromyalgia. *Journal of Musculoskeletal Pain*, 3(2): 59-65.
72. Russell, I. (1998). Substance P and fibromyalgia. *Journal of Musculoskeletal Pain*, 6(3): 29-35.
73. Russell, I. (1999). Neurochemical pathogenesis of fibromyalgia syndrome. *Journal of Musculoskeletal Pain*, 7(1/2): 183-191.
74. Russell, I., Orr, M., Littman, B., Vipraio, G., Alboukrek, D., Michaloeck, J., Lopez, Y., MacKillip, F. (1994). Elevated cerebrospinal fluid levels of substance P in patients with fibromyalgia syndrome. *Arthritis & Rheumatism*, 37(11): 1593-1601.
75. Saletu, B. (1987). The use of pharmaco-EEG in drug profiles. In: I. Hindmarch, P. Stonier (Eds.), *Human psychopharmacology: Measures and methods, Volume 1*. (pp. 173-200) Lichester, MD: Wiley & Sons.
76. Saletu, B., Grunberger, J. (1988). Drug profiling by computed electroencephalography and brain maps, with special consideration of sertraline and its psychometric effects. *Journal of Clinical status in primary fibromyalgia. Arthritis & Rheumatism*, 35: 15-21. *Psychiatry*, 49 (8): 59-71.
77. San Pedro, E.C., Mountz, J.M., Mountz, J.D., Liu, H.G., Katholi, C.R., Deutsch, G. (1998). Familial painful restless legs syndrome correlates with pain dependent variation of blood to the caudate, thalamus, and anterior cingulate. *Journal of Rheumatology*, 25 (11): 2270-2275.
78. Scharein, E., Bromm, B. (1995). Comparative evaluation of analgesic efficacy of drugs. In B. Bromm & J. E. Desmedt, (Eds.), *Advances in Pain Research and Therapy, Volume 22*. (pp. 473-500). New York, NY: Raven Press.
79. Schwarz, M., Spath, M., Muller-Bardorff, H., Pongratz, D., Bondy, B., Ackenheil, M. (1999). relationship of substance P, 5-hydroxyindole acetic acid (5HIAA) and tryptophan in serum of fibromyalgia patients. *Neuroscience Letters*, 259: 196-198.
80. Simms, R. (1998). Fibromyalgia is not a muscle disorder. *American Journal of Medical Sciences*, 315: 346-350.
81. Smith, K., Morris, J., Friston, K., Cowen, P., Dolen, R. (1999). Brain mechanisms associated with depressive relapse and associated cognitive impairment following tryptophan depletion. *British Journal of Psychiatry*, 174: 525-529.

82. Spath, M., Stratz, Th., Schmalisch, P., Fischer, P., Haslinger, A., Schwarz, M., Muller, W., Pongratz, D. (1998). Substance P in skeletal muscle of patients with fibromyalgia syndrome. *Journal of Musculoskeletal Pain*, 6 (Suppl. 2): 78.
83. Stevens, A., Batra, A., Kotter, I., Bartels, M., Schwarz, J. (2000). Both pain and EEG response to cold pressor stimulation occurs faster in fibromyalgia patients than in control subjects. *Psychiatry Research*, 97: 237-240.
84. Stratz, T., Farber, L., Varga, B., Haus, U., Baumgartner, C., Muller, W. (2000). Treatment of fibromyalgia with intravenous application of Tropicisetron. *Journal of Musculoskeletal Pain*, 8(4): 31-41.
85. Tansey, M.A. (1993). EEG neurofeedback and chronic fatigue syndrome: new findings with respect to diagnosis and treatment. *CFIDS Chronicle*, Fall: 30-32.
86. Thatcher, R. (1997). Federal registered copyright (TXU 347-139) of the Lifespan EEG Normative Database and all its derivatives.
87. Thatcher, R. (1998). Normative EEG databases and EEG biofeedback. *Journal of Neurotherapy*, 2(4): 8-39.
88. Vaeroy, H., Helle, R., Forre, O., Kass, E., Terenius, L. (1988). Elevated CFS levels of substance P and high incidence of Raynaud phenomenon in patients with fibromyalgia: New features for diagnosis. *Pain*, 32: 21.
89. Westmoreland, B. (1993). The EEG in cerebral inflammatory processes (291-304). In: Niedermeyer, E., Da Silva, F. (Eds), *Electromyography Basic Principles, Clinical Applications, and Related Fields*. New York, NY: Williams & Wilkins.
90. White, K., Nielson, W., Harth, M. (1999). Psychological distress and healthcare seeking behaviour: caution warranted in interpreting data. *Journal of Rheumatology*, 26: 244-246.
91. Williams, M. (1991). *Mental Assessment Scale*. Psychological Assessment Resources Inc., Lutz, Fl.
92. Wolfe, F., Ross, K., Anderson, J., Russell, I., Herbert, L. (1995). The prevalence and characteristics of fibromyalgia in the general population. *Arthritis & Rheumatism*, 38: 19.
93. Wolfe, F., Smythe, H., Yunus, M., Bennett, R., Bombardier, C., et al. (1990). The American College of Rheumatology 1990 criteria for the classification of fibromyalgia: report of the multicenter criteria committee. *Arthritis & Rheumatism*, 33: 160-172.

94. Yunus, M. (1994). Psychological factors in fibromyalgia syndrome: An overview. *Journal of Musculoskeletal Pain*, 2(1): 87-91.
95. Yunus, M., Ahles, T., Aldag, J. et al. (1991). Relationship of clinical features with psychological status in primary fibromyalgia. *Arthritis & Rheumatism*, 35: 15-21.

Table 1

Average Psychological Test Results by Groups

Psychological Test	Average Scores Total Group (N=40)	Group 1 (n = 9)	Group 2 (n = 24)	Group 3 (n = 7)
SCL 90-R (GSI)	65.9 (<i>SD</i> =6.9)	57.9 (2.6)	65.8 (3.1)	77.1 (2.8)
MPQ	34.1 (<i>SD</i> = 12.7)	23.4 (11.1)	34.1 (12.4)	41.3 (10.0)
VAS	5.5 (<i>SD</i> = 2.1)	4.4 (2.9)	5.4 (1.6)	7.1 (1.7)
MAS	95.7% (<i>SD</i> = 13.5)	95.6 (12.3)	95.8 (12.8)	92.0 (18.4)

Table 2

Average Z-scores of EEG Power by Frequency Band and Task
Fibromyalgia Patients Compared to Normative Data

Frequency	Task	
	Eyes Closed	Serial 7s
Alpha (7.5 – 13 Hz)	0.01	-0.10
Beta (13.0-22.0 Hz)	0.34	0.47
Delta (0.5-3.5 Hz)	-0.03	-0.22
Theta (3.5-7.5 Hz)	0.12	0.05

Note: Z-scores are standard scores ranging from -3.00 to +3.00; with a mean of 0 and standard deviation of 1.0. Z-scores of -1.00 and +1.00 lie at approximately the 34%tile and 68%tile of the distribution respectively. Z-scores of less than -1.93 or greater than +1.93 are significant at approximately the 0.05 level of probability or greater.

Table 3.

Average QEEG Z Scores by Frequency and Group

	Group 1 (n = 9)	Group 2 (N = 24)	Group 3 (N = 7)
Alpha (7.5 – 13 Hz)	0.20 (0.75)	0.05 (0.95)	-0.37 (1.24)
Beta (13.0-22.0 Hz)	0.17 (1.52)	0.49 (1.21)	0.06 (1.23)
Delta (0.5-3.5 Hz)	-0.29 (0.64)	-0.47 (0.57)	-0.18 (0.57)
Theta (3.5-7.5 Hz)	-0.15 (0.64)	0.03 (0.68)	0.81 (1.00)