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Diffuse Muscular Coactivation (DMC) as a potential source of pain in fibromyalgia – Part 1

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Abstract: Fibromyalgia is characterized by diffuse pain, the origin of which remains obscure. This study explored a phenomenon labeled Diffuse Muscle Coactivation (DMC) as a possible source of pain in fibromyalgia. DMC is defined as an increase from resting levels (tonus) in the electrical activity of any muscle during a movement which does not involve that muscle and is not part of the agonist-antagonist unit. When compared to controls this activity in persons with fibromyalgia was 1.75 times more prevalent and demonstrated significantly higher peak amplitudes. Possible neurological mechanisms are discussed.

Keywords: Fibromyalgia, muscle activity, coactivation, electromyography

1. Introduction

The primary feature of fibromyalgia is that of pain occurring at 18 tender points throughout the body. The American College of Rheumatology (ACR) has adopted the criteria of pain occurring at 11 of these sites for inclusion into this diagnostic category. Wolfe [1] reported this criterion discriminated between fibromyalgia sufferers and controls reliably and accurately.

The cause of the development of the tender points has been subject to much investigation, primarily examining for systemic factors. Numerous systemic theories as to the etiology and pathophysiology of fibromyalgia abound, including: central neurotransmitter imbalances [2,3], neuroendocrine-immune dysfunction [4–6], thyroid hormone resistance [7,8], stress-related physiological changes [9], psychopathology [10–14], psychosocial factors [15], and sleep disturbance (alpha intrusion) [16–18].

Numerous investigators [1,19] have also examined muscle activity as a source of the pain. A main focus has been on the relationship of trigger points to tender points without any conclusive results noted. Trigger points and tender points present in fundamentally different ways. Trigger points are hyperirritable spots located within a taut band of skeletal muscle that, upon compression, produce local pain, and may produce referred pain, a twitch response, jump sign and an autonomic response. Trigger points are thought to be a result of some damage to localized muscle cells [20] and may be alleviated though therapeutic techniques (i.e. ischemic compression, acupuncture, injection, stretch and spray, moist heat, ultrasound, etc.). In contrast, tender points are spots throughout the body, which are painful to applied pressure below 4 kg. They are not located in tissue which is noticeably hard or nodular in texture, do not refer pain and do not produce a twitch response. Tender points are thought to arise from a systemic dysfunction and local treatment of these sites produces little relief. Trigger points may be found in fibromyalgia patients but tender points may not be present in a myofascial pain syndrome [21].

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Surface electromyography (sEMG) offers the scientist a chance to study muscle activity in a functional manner. Examination of several characteristics of muscle activity including amplitude, frequency, interactions with other muscles, and recovery of pre-movement baseline are all possible with sEMG. Of particular interest to this study is the ability to measure muscle interactions during movements. Studies in the physical rehabilitation literature examining muscle interactions clearly demonstrate the existence of cocontractions (coactivations) in what are otherwise healthy muscles [22]. (Authors note: the terms cocontraction and coactivation are used interchangeably in this paper.) Coactivation may be readily seen in agonist-antagonist interactions. (Note: the terms agonist and antagonist are used in the traditional sense with agonist referring to the muscle which is the prime mover and initiates a movement while antagonist refers to a muscle which provides a negative contribution). Coactivation of agonists and antagonists produce high stiffness of the joint and little torque [23]. It is the antagonist that shows the presence of increased electrical activity, when it should be electrically quiet.

Janda and Stara (Janda and Stara, 1965 as cited in [23]) demonstrated the presence of coactivation in children, showing a high incidence of grouped responses of a predictable nature, even in muscles far removed from those which produced a required movement. This disappeared with maturity. Several other authors (Gatev, 1967; Okhnyanskaya et al., 1974 as cited in [23]) have also shown this phenomen suggesting they are of a suprasegmental origin which disappears with maturation.

Two of the authors [24,25] have reported results in which the muscle activity of persons with fibromyalgia differs from that of patients with myofascial pain. The authors reported what they termed muscle cocontractions (coactivation), in which movement of the head produced increased muscle activity in muscles other than in the agonist – antagonist unit. In order to avoid further confusion the authors have introduced a new term called Diffuse Muscular Coactivation (DMC). DMC is defined as an increase from resting levels (tonus) in the electrical activity of any muscle during a movement which does not involve that muscle and is not part of the agonist – antagonist unit.

Given that fibromyalgia sufferers report fatigue and morning stiffness, DMC, as a possible cause of these phenomena, needs to be investigated. The purpose of this study was to explore the relationship of DMC to the pain of fibromyalgia. Skubick [26] and Hellebrandt [27] reported an increase in the electrical activity of the arms during movement of the head. The purpose of this study was to examine if DMC occurred during movement of the head and was the DMC related to the pain of fibromyalgia.

Due to the complex nature of the study the results are divided into 2 parts. This part (Part 1) explores the interactions of muscles for DMC, comparing individuals suffering from fibromyalgia to controls. (Part 2 examines the nature of the DMC in detail.) Due to the lack of literature and prior research of this phenomenon in relationship to fibromyalgia, this study should be considered exploratory in nature.

2. Method

2.1. 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, 29 were rejected for issues concerning medications (i.e., taking medications which would alter some of the study's measures, but to stop taking them would put their life at risk), and one 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 [28] was administered according to the criteria of the ACR [1]. Two individuals did not meet the criteria of 11 of 18 tender points and were disqualified from the study. Thirty blood measures, urine and stool samples were collected and a board-certified specialist in hematology and internal medicine screened for concurrent and secondary diseases (i.e., rheumatoid arthritis, and lupus). Four individuals were disqualified, 2 because they would not complete the medical examinations and 2 on the basis of having active infections or diseases, which could affect the measurements [29]. The demographic data from the 6 who did not pass this phase of the screening were dropped from the data pool.

2.2. Measures

The remaining 40 subjects all completed psychological tests including the Symptom Checklist-90-Revised (SCL-90-R) [30]. McGill Pain Questionnaire (MPQ) [31], Visual Analogue Scale (VAS) measuring pain [32], Fibromyalgia Impact Questionnaire (FIQ) [33] and a Memory Assessment Scale (MAS) [34].

An sEMG evaluation designed specifically by the senior author for this study was administered to each of the 40 subjects. Data was collected on a Physiotech 4000^{TM} system (Myotronics-Noromed, Inc. 15425-53 Ave. S., Tukwila, WA 98188, USA), with a sampling rate of 240 Hz per channel, with a bandpass filter of 40–450 HZ. Eight channels of processed Root Mean Square (RMS) data were collected simultaneously. Electrodes were placed bilaterally over the cervical paraspinals at C4-6 for the purpose of monitoring the movements of the head, which included flexion from the sitting position and rotations to the left and right from the supine position. Electrodes were also placed bilaterally over the forearm extensor bundles (2 cms distal to the epicondyles), bilaterally over the gluteus medius – parallel to the belly of the muscle, and over the vastus medialis – near the fat pads proximal to the belly of the muscle. Bipolar electrode placements were 2 cms center to center. Subjects were asked to flex their heads three times, rotate their head to the left three times, and to the right three times. Degree of rotation was controlled with the use of a goniometer and held constant for each movement. Readings were taken before any of the movements occurred (labeled baseline) and during each movement with the third repetition (labeled peak) analyzed. The third repetition was selected in order to avoid any warm-up effects and on the basis of research by various authors [22,35] that shows consistency of amplitude over time. A total of 18 readings were taken for both baseline and peak. All data was stored to disk for future data analysis.

Six individuals who reported no significant health problems or history of a whiplash were recruited as controls. These individuals completed the same evaluation as the subjects, except did not receive the medical evaluations.

3. Results

3.1. Demographics

The demographic results indicated that the subjects were predominately female ($N = 37$), average age of 44.3 ($SD = 8.2$) years, right-handed ($N = 38$), had been in pain for 8.8 ($SD = 7.5$) years. The average age for the controls was 33.3 ($SD = 6.6$) which while lower, did not significantly differ from the subjects. All controls were female, right handed with no time in pain.

3.2. Psychological results

The subjects reported an average score of 34.1 ($SD = 12.7$) on the McGill Pain Questionnaire. This score is elevated from the norm established for individuals suffering from low back pain. An average score of 5.5 ($SD = 2.1$) was demonstrated on the VAS. An average score of 95.7% ($SD = 13.5$) on the MAS Global Memory Scale, indicating no significant impairments in short term memory. The controls showed scores of 3.2 ($SD = 3.9$), 0.22 ($SD = 0.4$), and 95% respectively. The SCL 90-R Global Severity Index (GSI) was used as a general indicator of psychological dysfunction [30]. The GSI showed 10 individuals with a T score greater than 70, 25 with a score between 60 and 69, and 5 with a score between 50 and 59, with an overall average score of 65.9 ($SD = 6.9$) suggesting a moderate level of dysfunction. All control subjects showed a score of 50 or less with an average of 42.3 ($SD = 7.3$) indicating little psychological disturbance.

3.3. sEMG results

Utilizing a criteria level of 20% [23] all 40 subjects and all 6 controls showed the presence of DMC. The subjects averaged 5.35 DMC ($SD = 2.89$) with a range of 1 to 12, while the controls averaged 3.3 DMC ($SD = 1.22$) with a range of 1 to 5. Figure 1 illustrates increased muscle activity as seen in fibromyalgia, while Fig. 2 illustrates a set of tracings from a control subject with no increase in activity. (Note the activity from lead 1 – the left CPS is not evident in this particular tracing due to a broken lead).

The subjects showed a total number of 214 DMC (out of a possible 720 – 29.7%), consisting of 110 on the left and 104 on the right side. The left-sided activity showed increased activity in 56 wrist extensors, 35 gluteus medius and 19 vastus medialis. The right side total score was made up of 60, 25 and 19 respectively. This compared to a total of 20 (out of a possible 108 – 18.5%) in total for the control group, consisting of 11 on the left side and 9 on the right side. This consisted of increased wrist extensor activity of 7 on the left and 4 on the right, for the gluteus medius 3 and 2 respectively, and for the vastus medialis 1 and 3 respectively. When

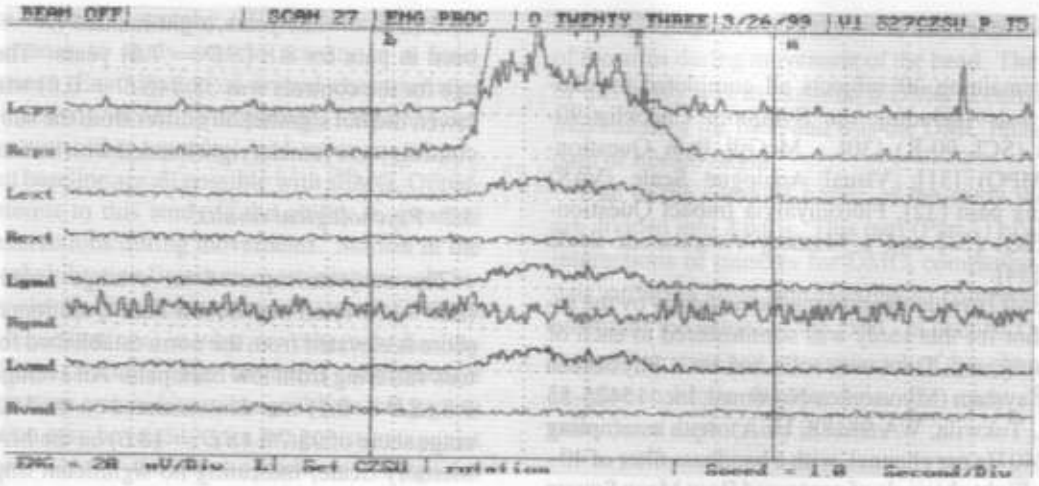

Fig. 1. Fibromyalgia sufferer – DMC.

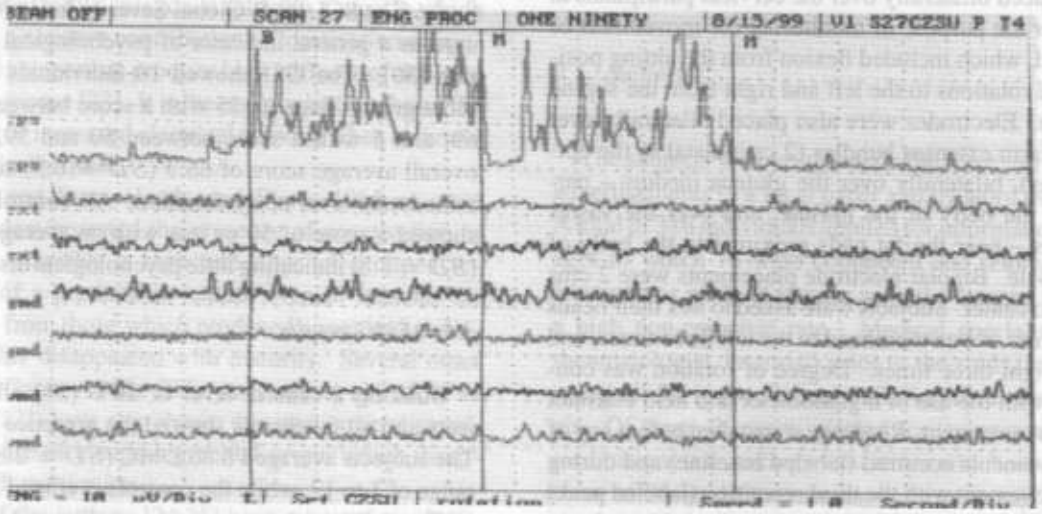

Fig. 2. Control subject – No DMC.

adjusted for group size, the ratio of increased activity in the fibromyalgia group to controls is approximately 1.75:1.

The baseline level of muscle activity was measured in microvolts for all muscles and showed an average of 3.4 microvolts for all the fibromyalgia subjects and 2.99 microvolts for controls. A one-way ANOVA for between group differences was not significant.

The peak level of activity measured in microvolts was 3.1 microvolts for the control group and 5.0 microvolts for the fibromyalgia group. One-way ANOVA for group by site showed differences ($p > 0.05$) for a) flexion – left wrist extensors, left gluteus medius, right vastus medialis, b) rotation to the left – right wrist extensors and c) rotation to the right – left wrist extensors, right wrist extensors, right vastus medialis.

Finally, the peak activity was correlated with the dolorimeter, McGill Pain Questionnaire and the VAS scores. No significant findings were noted.

The increased activity was then manually compared to the dolorimeter scores for agreement/disagreement. The two readings agreed (both were positive or both were negative) 57.5% of the time for the fibromyalgia group and 58.3% for the control group. Examination of the disagree patterns was then conducted. When the dolorimeter was positive (+) and the increased activity negative (−), this occurred 88.4% of the time for the fibromyalgia group and 11.5% for the control group. When the dolorimeter score was negative (−) and the increased activity was positive (+), this occurred 26.6%

of the time for the fibromyalgia group and 73.3% for the control group.

4. Discussion

The purpose of this part of the study was to investigate the relationship of muscle activity to the pain of fibromyalgia. The results are consistent with Zidar et al.'s [36] work showing that the baseline level of activity is similar to that of controls, discounting resting levels as a source of the development of the tender points or fatigue or morning stiffness. However the appearance of DMC throughout the body has not previously been reported and an argument may be made as to the relationship of the DMC to the appearance of the tender points.

Cocontractions that occur in the agonist-antagonist unit may be seen as a localized expression of the DMC phenomenon. Cocontractions appear to be under peripheral motor control mechanisms [23,24] and appear to be related to the presence of pain. Skubick [26] demonstrated that a reduction in cocontractions between the sternomastoid muscle and the flexor bundles in the forearm were strongly associated with a decrease in pain, and an improvement in nerve conduction measures. One control subject in particular clearly matched the DMC to the site in her hip in which she experienced pain.

In the literature numerous authors have reported cocontractions as localized phenomena and part of various processes including, and beyond, the agonist – antagonist unit. These include: a developmental process involving reflexes and the central nervous system [37] (Janda and Stara, 1965 as cited in [23]), as part of a reflex pattern involving a myotatic unit [27], as part of a disregulation in the Tonic Neck Reflex [26], or as part of the aging process, fatigue, and stress [23]. However, these papers (except for Janda and Stara) did not study muscles in other parts of the body as part of the investigation so it is not known if the appearance of the generalized phenomena would have been noted.

Neurologically these processes can be linked. As previously stated, cocontractions appear to involve a peripheral control mechanism [23,24] while the DMC appears to involve a central control mechanism. Basmajian [23] lists four possible motor control mechanisms: a) centrally mediated reciprocal inhibition, b) centrally mediated coactivation, c) peripherally mediated reciprocal inhibition and d) peripherally mediated coactivation. It is possible to speculate on a progressive disregulation that starts peripherally and moves centrally, involving these mechanisms, causing neural plasticity changes in the motor control systems and leading to the appearance of diffuse pain.

Of interest also, is the presence of the DMC in healthy controls. It is not known if this represents an early breakdown in the central motor control processes or some other phenomenon leading to the development of pain. If this is true the early appearance of the DMC phenomenon may serve as a marker for the detection of this dysfunction.

The data also indicate that when the muscles increase in activity there is no delay in onset between the proximal and distal muscles. Visual examination of Fig. 1 shows all the muscles on the left side increasing in activity simultaneously. This suggests one drive mechanism as opposed to the spreading out of a signal from multiple sites as in the sensory motor cortex. There may be thalamic involvement interacting with the cerebellum to produce this process. It is also possible that this phenomenon may involve segmental and/or suprasegmental activity of the spine. Further research is needed.

The data shows that the increased activity does not occur in all muscles, appearing more frequently in proximity to the neck and decreasing distally from the neck. Flexion and rotation of the head to the right produced a greater increase in the activity than rotation to the left. Whether this is related to the subjects' handedness needs further investigation. It is also not known if a different movement would produce different results.

The increase of 20% in electrical activity from resting levels was chosen on the basis of Basmajian's work in Muscles Alive [23]. Retrospectively, increases of 30% and 50% were investigated as criterion to see if this change would show differences between the groups. Very little change occurred in the differences between groups with the 1.75:1 ratio of fibromyalgia to controls holding consistent.

The lack of significant findings between the sEMG activity and the dolorimeter findings was not surprising. The dolorimeter findings reflect a perceptual process including allodyna, while the sEMG activity reflects a physiological process. This is particularly evident in the data, showing a disagreement with the dolorimeter positive and the increased muscle activity negative. There may also be a measurement error operating here. The DMC sEMG results are based upon three repetitions of a movement. It is not known what an increase in the number of repetitions would do. Daily activity would produce 1000s of movements of the head,

changing the sEMG results accordingly. It is assumed that the sEMG activity levels would increase with repetition, perhaps matching the dolorimeter findings more exactly. This remains to be investigated.

While not a primary focus of the study, the results of the SCL 90 R are intriguing as these results show a wide variation in the scores. The wide variation in scores and lack of correlation to the activity suggest that psychological factors are not directly involved in the causation of the DMC. While needing further investigation, these results suggest that screening for psychological issues in the treatment of fibromyalgia is essential, as some individuals will need psychological treatment while others have a limited emotional component to this dysfunction.

Finally, it is not known if the diffuse increase in muscle activity reflects epiphenomenona or is characteristic of a specific dysfunction as seen in fibromyalgia. The literature (i.e. Basmajian [23]) suggests the emergence of the coactivation may be due to a breaking-down of inhibitory control mechanisms. More study is needed before any conclusive statements may be made.

This study should be considered exploratory in nature, as it was included initially as part of another study searching for diagnostic markers for fibromyalgia. It is reported in this manner because the DMC was not specifically looked for and appeared as a serendipitous finding. The lack of a significant number of controls further limits the use of appropriate statistics as examination of this data shows a significant problem with homogeneity of variance. Regardless of these limitations, further investigation of this phenomena is recommended. The presence of DMC throughout the body has not been reported in the literature and may represent a breakdown in the inhibitory control mechanisms in the body. Part 2 of this study represents further examination of this phenomenon.

References

[1] F. Wolfe, H. Smythe and M. Yunus et al., The American College of Rheumatology 1990 criteria for the classification of fibromyalgia: report of the multicenter criteria committee, *Arthritis & Rheumatism* **33** (1990), 160–172.

[2] I. Russell, Neurohormonal: Abnormal laboratory findings related to pain and fatigue in fibromyalgia, *Journal of Musculoskeletal Pain* **3**(2) (1995), 59–65.

[3] I. Russell, Neurochemical pathogenesis of fibromyalgia syndrome, *Journal of Musculoskeletal Pain* **7**(1/2) (1999), 183–191.

[4] L. Crofford and M. Demitrack, Evidence that abnormalities of central neurohormonal systems are key to understanding fibromyalgia and chronic fatigue syndrome, *Rheumatic Diseases Clinics of North America* **22** (1996), 267–284.

[5] P. Dessein, E. Shipton, B. Joffe, D. Hadebe, A. Stanwix and B. Van der Merwe, 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) (1999), 313–319.

[6] J. Peter and D. Wallace, Abnormal immune regulation in fibromyalgia, *Arthritis & Rheumatology* **31** (1988), S24.

[7] J. Lowe, Thyroid status of 38 fibromyalgia patients: Implications for the etiology of fibromyalgia, *Clinical Bulletin of Myofascial Therapy* **2**(1) (1997), 36–41.

[8] J. Lowe, M. McCullum, L. Graf and J. Yellin, Mutations in the c-erbAb1 gene: Do they underlie euthyroid fibromyalgia? *Medical Hypotheses* **48**(2) (1997), 125–135.

[9] E. Griep, J. Boersma and R. de Kloet, Altered reactivity of the hypothalamic-pituitary-adrenal axis of the primary fibromyalgia syndrome, *The Journal of Rheumatology* **20**(3) (1993), 469–474.

[10] M. Boissevain and G. McCain, Toward an integrated understanding of fibromyalgia syndrome: II. Psychological and phenomenological aspects, *Pain* **45** (1991), 239–248.

[11] H. Diamond, Psychogenic rheumatism, *Clinics of Rheumatological Practice* **2** (1984), 131–133.

[12] J. Hudson, M. Hudson and L. Pliner et al., Fibromyalgia and major affective disorder: a controlled phenomenology and family history study, *American Journal of Psychiatry* **142** (1985), 441–446.

[13] J. Hudson and H. Pope, Fibromyalgia and psychopathology: Is fibromyalgia a form of "Affective Spectrum Disorder?", *Journal of Rheumatology* **16** (1989), 15–22.

[14] M. Yunus, T. Ahles and J. Aldag et al., Relationship of clinical features with psychological status in primary fibromyalgia, *Arthritis & Rheumatism* **35** (1991), 15–21.

[15] A. Russell, Fibromyalgia – A historical perspective, *Journal of Musculoskeletal Pain* **3**(2) (1995), 43–48.

[16] H. Moldofsky, Fibromyalgia, sleep disorder and chronic fatigue syndrome, in: *Chronic Fatigue Syndrome*, G. Block and J. Whelan, eds, J. Wiley Chicester, 1993.

[17] H. Moldofsky and F. Lue, Sleep and symptoms in 205 fibromyalgia patients versus 876 patients with various sleep disorders, *Sleep Research* **2** (1992), 303–315.

[18] H. Moldofsky, P. Scarisbrick, R. England and H. Smythe, Musculoskeletal symptoms and non-REM sleep disturbance in patients with fibrositis syndrome and healthy subjects, *Psychosomatic Medicine* **37** (1975), 341–351.

[19] R. Bennett, The contribution of muscle to the generation of fibromyalgia symptomatology, *Journal of Musculoskeletal Pain* **4**(1/2) (1996), 35–59.

[20] M. Schneider, Tender points/fibromyalgia vs. trigger points/myofascial pain syndrome: a need for clarity in terminology and differential diagnosis, *Journal of Manipulative and Physiological Therapeutics* **18**(6) (1995), 398–406.

[21] D. Pongratz and M. Sievers, Fibromyalgia – symptom or diagnosis: a definition of the position, *Scandinavian Journal of Rheumatology* **113** (2000), 3–7.

[22] G. Sella, *Muscular Dynamics: Electromyographic Assessment of Energy and Motion*, GENMED Publishing: Martins Ferry, OH, 2000.

[23] J. Basmajian and C. De Luca, *Muscles Alive*, (5th ed), Their Functions Revealed by Electromyography, Williams & Wikins, New York, NY, Williams & Wilkins, 1985.

[24] S. Donaldson, G. Sella and H. Mueller, Fibromyalgia: A retrospective study of 252 consecutive referrals, *Canadian Journal of Clinical Medicine* **5**(6) (1998), 116–127.

[25] H. Mueller, S. Donaldson, D. Nelson and M. Layman, Treatment of fibromyalgia incorporating EEG-driven stimulation: a clinical outcomes study, *Journal of Clinical Psychology* **57**(7) (2001), 933–954.

[26] D. Skubick, R. Clasby, S. Donaldson and W. Marshall, Carpal tunnel syndromes as an expression of muscular dysfunction in the neck, *Journal of Occupational Rehabilitation* **3**(1) (1993), 31–44.

[27] F. Hellebrandt, S. Houtz, M. Partridge and C. Waltos, Tonic neck reflexes in exercises of stress in man, *American Journal of Physical Medicine* **35** (1956), 144–159.

[28] A. Fischer, Algometry in diagnosis of musculoskeletal pain and the evaluation of treatment outcome: an update, *Journal of Musculoskeletal Pain* **6**(1) (1998), 5–32.

[29] B. Westmorland, The EEG in cerebral inflammatory processes, in: *Electromyography Basic Principles, Clinical Applications, and Related Field*, E. Niedermeyer and F. Da Silva, eds, Williams & Wilkins, New York, NY, 1993.

[30] L. Derogatis, *SCL-90-R: Administration, scoring and procedure manual*. (3rd ed.), Clinical Psychometric Research, 1994.

[31] R. Melzack, The McGill Pain Questionnaire, in: *Pain Measurement and Assessment*, R. Melzack, ed., Raven Press, New York, NY, 1983.

[32] E. Huskisson, Visual Analogue Scales, in: *Pain Measurement and Assessment*, R. Melzack, ed., Raven Press, New York, NY, 1983.

[33] C. Burckhardt, R. Clark and R. Bennett, The fibromyalgia impact questionnaire: Development and validation, *The Journal of Rheumatology* **18** (1991), 728–733.

[34] M. Williams, *Mental Assessment Scale*, Psychological Assessment Resources Inc., Lutz, FL, 1991.

[35] T. Sihnovan, J. Partanen and O. Hanninen, Averaged (rms) surface EMG in testing back function, *Electromyography and Clinical Neurophysiology* **28** (1988), 335–339.

[36] J. Zidar, E. Bachman, A. Bengtsson and K. Henriksson, Quantitative EMG and muscle tension in painful muscles in fibromyalgia, *Pain* **40**(3) (1990), 249–254.

[37] C. Leonard, T. Moritani, H. Hirsehfeld and H. Forssberg, Deficits in reciprocal inhibition of children with cerebral palsy as revealed by H reflex testing, *Developmental Medicine and Child Neurology* **32** (1990), 974–984.