

# **Primary Fibromyalgia and Chronic Fatigue Syndrome: Upper Cervical Management of 23 Successive Cases.**

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## **ABSTRACT**

The current scientific literature offers little with regards to published studies on chiropractic management of primary fibromyalgia and chronic fatigue syndrome (1). The clinical observations of 23 successive cases, along with a case presentation, are reported. 92-100% improvement in symptoms was achieved in both these syndromes subsequent to corrections of aberrant arthrokinematic function of the occipito-atlanto-axial complex. A causal relationship between biomechanical faults in the upper cervical spine, abnormal central neurophysiologic processing, and subsequent peripheral neuropathophysiology, is suggested as the possible genesis of these two syndromes.

**Key words:** Fibromyalgia, Chronic Fatigue Syndrome, Infrared Imaging, Upper Cervical Spine

## **INTRODUCTION**

The initial patient profile of fibromyalgia syndrome (FMS) includes a history of widespread pain of at least three months duration, both above and below the waist and including both sides of the body. The pain is described as deep aching, radiating, gnawing, shooting, or burning. The overwhelming characteristic of fibromyalgia is long-standing pain at defined tender points. This disorder can appear in patients of all ages with an overall incidence of 9:1,000. FMS, however, has an increased frequency in women with the greatest incidence in the ages of 20 to 50 years (2-3).

In 1990, the American College of Rheumatology released their criteria for the clinical classification of FMS. The criteria was drawn from the results of a prospective blinded investigation involving more than 550 fibrositis patients at multiple clinic and hospital out-patient settings across the U.S. (4). A condition in which tender points are confined to a specific region, associated with specific muscles, and exhibiting a characteristic pain referral pattern was referred to as myofascial pain syndrome (MPS). Patients with generalized tender points in the presence of widespread pain for at least 3 months, along with disturbed sleep, were classified as having FMS. Patients with FMS may have concomitant MPS, but not the reverse.

The study committee adopted specific definitions for use in making the diagnosis of FMS. Along with the previously mentioned widespread pain, tender points must be

found in at least 11 of 18 sites which include the: occiput, lower cervical, trapezius, supraspinatus, anterior 2<sup>nd</sup> rib, lateral epicondyle, gluteal, greater trochanter, and knees. Palpatory pain elicited by 4 kg of pressure (measured by algometry) is considered positive at these tender points. Those with FMS tend to waken with body aches and stiffness. Pain generally improves during the day and often increases again during the evening. Symptom flare-ups can occur with activity; cold, damp weather; anxiety, hormonal fluctuations (premenstrual and menopausal states), and stress. The majority of patients with FMS also report that they awaken unrested and complain of daily fatigue.

FMS can present as a constellation of symptoms. Therefore, other disease states must be ruled out before a diagnosis of FMS can be made. Conditions that can mimic the symptoms of FMS include: RA, hypothyroidism, SLE, Lyme disease, primary sleep disorders, depression, and HIV infection. The primary clinical features of FMS, rated in percent of patients experience, are as follows: 90-100% -- generalized pain, fatigue, stiffness, morning fatigue; 70-90% -- sleep disturbances, post-exertional malaise, headaches, swollen feeling in tissues, numbness and tingling, cognitive impairment, dizziness, sensitivity to noise and stress, dysmenorrhea, dry mouth; 50-70% -- irritable bowel syndrome, blurred vision, affective lability, heart palpitations, cold extremities, feverous feeling, allergies; 15-50% -- restless legs, muscle twitches, itchy skin, hearing disturbances, night sweats, migraines, breathing problems, infection proneness, skin manifestations, interstitial cystitis, TMD; Below 15% -- major depression (concurrent).

Current medical sources state that there is no known specific cause or prevention for FMS (2-3). However, it is suggested that there may be many different "triggering events" that precipitate the condition's onset. Possible events include physical trauma such as sports injuries, automobile accidents, falls, etc. or post-illness onset. Interestingly, it is thought that these triggering events do not cause FMS, but rather, they may awaken an underlying physiologic abnormality that is already present.

One hypothesis suggests that the disorder may be associated with changes in skeletal muscle metabolism, such as decreased blood flow, which could cause overall chronic fatigue and weakness. Another premise is that an infectious microbe, such as a virus, triggers the illness. At this point, no virus or microbe has been identified. The latest research is being directed toward investigating alterations in neurotransmitter regulation (particularly serotonin, norepinephrine, and substance-P), immune function, sleep physiology, and hormonal control as a possible etiology of both FMS and chronic fatigue syndrome. Repeated studies have found substance-P to be elevated threefold in the CSF of patients with FMS. A defective neural feedback loop is thought to be responsible for this finding. Abnormal variations in two hormones, cortisol and GH, have also been found in some patients. A great deal of research is being spent on investigating alterations in normal sympathetic nervous system function. Patients with FMS and chronic fatigue syndrome have been found to have lower levels of sympathetically released neuropeptide-Y. As it can be seen, the majority of current research is being directed toward investigating abnormal neurophysiology as the possible underlying cause of FMS and chronic fatigue syndrome.

Chronic fatigue syndrome (CFS), as previously mentioned, appears in 90-100% of patients with FMS. CFS usually surfaces sometime after the initial symptoms of FMS. However, CFS can appear in the early stages of FMS in many patients. Under current guidelines it would be very difficult to find a FMS patient who did not meet the new CDC criteria for CFS. Research also suggests that CFS may actually be present in some patients before the symptoms of FMS. From this it is proposed that patients with only chronic fatigue may have a dormant form of FMS, which may appear much later.

CFS can range from mild to incapacitating. The fatigue may be described as tiredness, a total drain of energy, heavy limbs, and/or poor concentration. The etiology of CFS encompasses some of the same factors as FMS, but also includes objective signs of EEG abnormalities. Most FMS patients exhibit an associated alpha-EEG anomaly sleep disorder. Studies show that most FMS patients can fall asleep without much trouble, but that their deep level (stage 4) sleep was constantly interrupted by bursts of alpha wave activity. Patients report that they awake feeling as if they have had little to no sleep. Two common sleep disorders that may also be present in FMS/CFS patients include restless leg syndrome and periodic limb movement during sleep (2-3).

The most common medical treatments for FMS and CFS can include one or more of the following: tricyclic antidepressants, nonsteroidal anti-inflammatories, physical therapy, gentle stretching, low impact exercises, stress reduction, counseling, and lidocaine injections with or without hydrocortisone. Currently, the medical prognosis for FMS and CFS is not favorable. It is reported that as many as 40% of patients may significantly improve over time, but few are thought to completely recover (2-3).

It is interesting to note that Masi and Yunus (5) speculate that FMS and CFS are part of a larger spectrum of conditions, which they term Dysregulation Spectrum Syndrome. Yunus uses the term dysregulation to denote biophysiological abnormalities, most likely in the neurohormonal system. This follows the latest research into the nervous system as the possible primary etiology of both FMS and CFS.

## **STUDY REPORT**

Over a 5-year period, 23 cases of fibromyalgia syndrome with chronic fatigue syndrome were treated in an outpatient setting. All cases were followed for a minimum of 1½ years to observe treatment effectiveness. The study group was comprised of 5 males and 18 females ranging from 11 to 76 years of age. Statistically, the subject group, consisting of 78% females with a mean age of 35, fits the published data on the most common patients with FMS and CFS (2-3).

Of the 23 patients, 96% presented with an incoming medical diagnosis of both FMS and CFS. A thorough initial history and physical examination confirmed this diagnosis. The chronicity of this condition ranged from 2 to 35 years. The intensity of the presenting symptomatology varied from moderate (ADL interrupted) to severe (inability to work). Each patient's progress was assessed on every office visit by rating the

intensity of his or her symptoms on a 1-10 VAS. All of the 23 subject's presenting symptoms fit the profile previously mentioned.

Upon stabilizing the upper cervical spine (determined by consistently presenting normal paraspinal infrared images – TyTron C-3000 Paraspinal IR System – Fig. 1 and 7), improvement in the symptomatic profile of both FMS and CFS was 92-100% (VAS) for all 23 patients. Every patient was able to resume his or her normal activities including full time work. The total time of treatment to reach this point ranged from 3 to 7 months with a mean treatment time of 15 weeks. The most common initial treatment frequency used was 3 times per week with tapering frequency after 4-8 weeks. Total treatment visits ranged from 20 to 48 with a mean of 31 office visits to stabilization. All 23 patients reported maintaining their improvements at 1½ years or more of follow up. Treatment consisted solely of corrections to aberrant arthrokinematic function of the occipito-atlanto-axial complex. The method of adjusting used was Applied Upper Cervical Biomechanics (International Upper Cervical Chiropractic Association).

## CASE REPORT

From the 23 subjects in this study, an average case has been selected for this report. The patient's presenting symptomatology, treatment profile, and case outcome is representative of most of the patients in the group with the exception of chronicity.

A 55-year-old female was referred to our clinic with the chief complaints of constant bilateral neck, trapezius, mid-back, elbow, hip, and foot pain, along with constant fatigue. She also experienced frequent bilateral upper extremity and right trapezius paresthesias. The patient advised that upon arising she felt sore, stiff, and as if she never really slept. Cold temperatures and physical or emotional stress caused an increase in all her symptoms. She described her life as constant pain and perpetual fatigue.

The patient recalled that her symptoms began after a fall 35 years previously when she was employed as a gymnastic circus performer. Her symptoms gradually increased to a point where over the past 10 years she had been able to maintain only light part-time work (12-20 hours per week deskwork) when her condition permitted – she was currently on sick leave due to the severity of her FMS/CFS. Approximately 15 years previously, a thorough medical workup yielded a diagnosis of primary FMS with accompanying CFS. Over the past 25 years the patient had tried many forms of treatment including physical therapy, massage, acupuncture, chiropractic, and exercise with little to no improvement. At the initial consultation, the patient had been using a combination of light exercise when tolerated, muscle relaxants, and a tricyclic anti-depressant at bedtime for 10 years with limited success. She rated her FMS/CFS as being constant and at an intensity level between 7-9 (1-10 VAS). The patient advised that her life used to be filled with activity, exercise, and happiness; and now it had been reduced to pain and very little else.

Upon examination, the patient presented with overall motions that were careful and deliberate. The patient was cooperative, but expressed that she was doubtful that treatment would be of benefit. Vital signs, ear, nose, and throat examinations were unremarkable.

Orthopedic examination revealed significant palpatory hypertonicity and tenderness of the paraspinal musculature from C0-T9 and including the trapezius, levator scapulae, and anterior cervical musculature bilaterally. A marked increase in myohypertonicity and tenderness was noted in the right occipital region. Upon algometric examination, the patient tested positive for 14 of the 18 designated FMS tender points with 1-3 kg of pressure. Myofascial trigger points were elicited in the trapezius, levator, and infraspinatus musculature. A 40% overall decrease in cervical active and passive ROMs was noted along with paraspinal and trapezius pain in 4 of 6 ranges. Cervical orthopedic tests were found positive for facet joint irritation. Multi-axis articular end-range examination revealed biomechanical abnormalities in the cervical, costovertebral, and thoracic spine. The remainder of the patient's lumbosacral, lower extremity, and upper extremity orthopedic evaluation was unremarkable.

Gross neurologic examination was also found to be unremarkable. A high-resolution paraspinal digital infrared imaging analysis was performed in accordance with thermographic protocol (6-8) (A TyTron C-3000 Paraspinal Imaging System was utilized, as it is the only paraspinal system accepted by the international thermographic community). A continuous paraspinal scan consisting of approximately 423 infrared samples was taken from the level of S1 to the occiput (Fig. 1). The data was analyzed against established normal values and found to contain wide thermal asymmetries indicating abnormal autonomic regulation or neuropathophysiology (9-12) (Fig. 2 and 3). Since the cervical spine displayed highly abnormal thermal asymmetries, a focused scan was performed with approximately 81 infrared samples taken from T1 to the occiput (Fig. 4). For the purpose of ruling out other pathologies, and to further characterize this condition, a computerized high-resolution infrared camera study (Inframetrics Forensic System 535) was also performed in accordance with accepted protocols (6-8). The scans included all surface aspects of the face, neck, upper extremities, and posterior thorax. The posterior neck and thorax image showed disruption of the normal thermal gradient, significant thermal asymmetries, large areas of hyperthermia, and focal zones of high infrared emissions (Fig. 5). These images were indicative of abnormal sympathetic regulation and broad-based myohypertonicity interspersed with myofascial trigger points. As such, these findings are consistent with the clinical presentation of FMS/CFS.

A high suspicion of abnormal upper cervical arthrokinematics was also gained from this examination. Consequently, a precision upper cervical radiographic series was performed for an accurate analysis of specific segmental biomechanics (13). Neutral lateral, AP, APOM, and BP films were taken using an on-patient laser-optic alignment system to precisely align the patient to the central ray. With this system, maintenance of precision patient alignment can be facilitated from the source of the X-ray beam rather than the bucky (Fig. 6).

An analytical radiographic method of combined mensuration and arthrokinematics was performed (13). Biomechanical abnormalities were noted at the atlanto-occipital and atlanto-axial articulations.

## CHIROPRACTIC MANAGEMENT

From the accumulated degree of aberrant biomechanics found at the atlanto-occipital articulations, correction of the C1 subluxation was chosen as the first to be adjusted. Before treatment was rendered, the patient was advised that exacerbations in symptomatology might occur as part of the normal response to care due to the global impact of neural reintegration.

In order to insure proper segmental contact and LOD control, the patient was placed on a specially designed knee-chest table with the posterior arch of atlas as the contact point. An adjusting force was introduced using a specialized upper cervical adjusting procedure (14). The patient was then placed in a post-adjustment recuperation suite for 15 minutes as per thermographic protocol (6-8). Correction of the subluxation was determined by resolution of the patient's presenting neuropathophysiology on the post-adjustment paraspinal infrared scans (Fig. 4). All subsequent office visits included an initial cervical paraspinal scan, and if care was rendered another scan was performed to determine if normal neurophysiology was restored (Fig. 4). Since the patient's care was focused in the upper cervical spine, only cervical paraspinal infrared scans were taken during normal treatment visits with full spine paraspinal scans performed at 30-day re-evaluation intervals.

During the first week of care no change in pain was noticed. A definite decrease in fatigue, however, was reported by the end of the week. The patient began to notice a reduction in neck and trapezius pain along with decreased paresthesias during the second week of care.

Significant reductions in the patient's symptoms began during the third week of treatment. She noted that, for the first time in over 10 years, she could lift her head without assistance after having her hair shampooed in a salon. The intensity and frequency of her neck and trapezius pain, along with the paresthesias, continued to decrease. Her mid-back and elbow pain was also beginning to improve. She noticed that her level of sleep seemed to be improving. The patient reported that her stress levels had increased over this week and was very surprised that there were little to no effects on her symptoms.

By the end of the fourth week of care, the patient was noting that there were times when she was experiencing no neck or trapezius pain. Pain in the elbows and mid-back region were also continuing to decrease. Her paresthesias were becoming mild and infrequent. She was also beginning to report improvements in her hips and feet. The patient noticed that she was now waking feeling more refreshed.

A re-evaluation was also performed at this time. The patient noted a 65% overall improvement in her condition. At this time she was reporting a pain intensity level of 4 (1-10 VAS). The examination revealed significantly decreased overall palpable myohypertonicity and tenderness. The patient tested positive for only 8 of the 18 FMS tender points with 2-4 kg of pressure on algometry. All of the previously found myofascial trigger points were markedly reduced. Cervical active and passive ROMs were found to be normal with mild paraspinal and trapezius stiffness in 2 of 6 ranges. Mild cervical facet joint irritation remained positive upon testing. Six-axis palpable spinal joint examination noted residual biomechanical abnormalities in the cervical and thoracic spine. A full spine paraspinal infrared scan was performed at this time noting total resolution of the patient's presenting neuropathophysiology (Fig. 7 and 8).

Weeks five and six showed a consistent, but slower improvement. The patient noted a mild continued decrease in pain including her hips and feet. Her paresthesias remained mild and infrequent. Upon waking she continued to feel more rested and started to notice that her daily fatigue levels were reducing. With this in mind, she began to slowly decrease her sleep medication under the guidance of her medical physician. Her progress, however, reached a plateau at week six. The normalization usually seen on her post-adjustment paraspinal scans was waning. LOD changes were made on C1 with little to no improvement in her scans. By week seven it was decided to change her listing to C2. The post-adjustment infrared scans showed an immediate return to normal with a corresponding improvement in the patient's symptomatic profile.

By the end of week eight, a significant improvement was noted upon re-evaluation. The patient rated her current pain levels at a 2 (1-10 VAS) with an intermittent frequency. She reported an overall improvement of 80% in her condition. All her examination findings continued to improve. The majority of the myofascial trigger points were resolving. Algometric testing revealed only 4 of the 18 FMS tender points with 4-6 kg of pressure. The frequency in which the patient was presenting with normal paraspinal infrared scans indicated that stabilization of the upper cervical joint complex was occurring. Consequently, a decrease in treatment frequency to 2 office visits per week was made at this time. Working with her medical physician, she continued to decrease her sleep medication and began reducing her muscle relaxants. Her daily level of fatigue had significantly reduced to the point where she began to increase her activities along with gardening for the first time in 10 years. Based upon her current symptom level, she decided to return to work part-time.

Over the next four weeks the patient continued to improve. Her pain and paresthesia levels continued to decrease in intensity and frequency. Work duties and stresses had not caused any exacerbation in her condition. The patient continued to wean off her medications with termination by week 11. Physical examination noted complete resolution of her myofascial trigger points and FMS tender points. She noted that her sleep had normalized and upon awakening she felt refreshed and pain free. Her daily energy continued to improve with complete resolution of her chronic fatigue. By the end of this time the patient had taken up bike riding and a gym based exercise program. A high-resolution computerized infrared camera re-evaluation was performed with the

images indicating a return of the thoracic thermal gradient and normalization of autonomic neurophysiology (Fig. 9).

The patient continued to improve over the next few weeks with complete resolution of her FMS and CFS. The patient returned to full time work during this time and experienced only two minor temporary exacerbations in her condition. Over the past two years the patient has been seen on a check-up basis of four times per year. She remains FMS and CFS free with only an occasional sore neck due to long deskwork hours.

## NEUROBIOLOGICAL MECHANISMS

Some form of initiating trauma followed by an aberrant neural cascade conforms with current research that points to a “triggering event” as the genesis of FMS/CFS. As previously mentioned, experts in this field feel that these “triggering events” probably do not cause FMS, but rather, they may awaken an underlying physiologic abnormality that is already present. What is commonly seen in our center, regardless of the patient’s symptomatic profile, is long standing neurophysiologic dysfunction followed by symptoms arising from months to decades later after some form of “triggering event”. The possibility of CFS alone preceding the symptoms of FMS, may also fall under this awakening of a long-term underlying pathophysiology.

There are two extensively studied neurophysiologic mechanisms that may explain the profound changes seen in the patients in this study. The first is CNS facilitation (15-19). This condition arises from an initiating trauma (birth, falling, etc.) which causes entrapment of intra-articular meniscoids resulting in segmental hypomobility and ultimately compensatory hypermobility. Consequently, hyperexcitation of intra and periarticular mechanoreceptors and nociceptors occurs. Over time, this bombardment of the central nervous system can cause facilitation. Facilitation results in an exponential rise in afferent signals to the cord and/or brain. This may cause a loss of central neural integration due to direct excitation, or a lack of normal inhibition, of pathways or nuclei at the level of the cord, brainstem, and/or higher brain centers. The upper cervical spine is uniquely suited to this condition, as it possesses inherently poor biomechanical stability along with the greatest concentration of spinal mechanoreceptors.

The second mechanism has been termed cerebral penumbra or brain cell hibernation (20-26). Previous research held that the neuron had two basic physiologic states: function and dysfunction. A third state, however, was uncovered which may explain the rapid and profound changes seen in some patients. When a certain threshold of ischemia is reached, a neuronal state of hibernation occurs; the cell remains alive, but ceases to perform its designated purpose. Entire functional areas of the cerebral cortex or cerebellum may be affected. The mechanism of hyperafferency, as mentioned above, plays the initiating role. Hyperafferent activation of the central regulating center for sympathetic function in the brain may cause differing levels of cerebral ischemia. A second route via the superior cervical sympathetic ganglia, may

also cause higher center ischemia. CNS facilitation and cerebral penumbra, or both acting at once, can result in: motor and sensory function abnormalities, hormonal dysregulation, immune system dysfunction, aberrant organ system regulation, autonomic/sympathetic dysregulation, and a loss in overall systemic neural integration.

These advances in neurophysiologic research correlate well with the pathophysiology currently proposed in the etiology of FMS and CFS (2-3, 5). Research into the role that the autonomic nervous system plays may lead to understanding the true pathophysiology of both FMS and CFS. The discovery of sympathetic innervation of intrafusal muscle fibers has added a significant contribution to the mechanism of sustained muscle contraction and chronic pain in FMS (27). This mechanism also explains why patients who are under emotional stress exhibit exacerbations in their symptoms (28). Thus, sympathetic hyperactivation can cause direct muscle contraction, changes in skeletal muscle metabolism via ischemia, and altered pain mediation resulting in overall chronic fatigue and weakness. The mechanisms previously covered could also explain the finding of altered neurotransmitter regulation, immune dysfunction, sleep pathophysiology, disrupted hormonal control, and lower levels of neuropeptide-Y.

Many experts in the field of FMS/CFS point to broad-based systemic dysfunction as the genesis of these two conditions (5). Terms such as Dysfunction Spectrum Syndrome have been used to describe this global systemic problem. Our findings agree with this premise. Patients exhibiting the classic profile of either FMS or CFS fit the constellation of symptoms seen in broad systemic pathophysiology. The sympathetic division of the autonomic nervous system has the single ability to affect the entire systemic function of the human body. Hence, the reason for devoting a great deal of research into this area. Dysfunction of the sympathetic nervous system may be the underlying pathology which is “triggered” to precipitate FMS or CFS. All 23 subjects in this study presented with abnormal infrared imaging markers; thus, uncovering significant sympathetic nervous system abnormalities. Consequently, the answer to these conditions may lie in the restoration of normal sympathetic function. Our specialized upper cervical spine approach supports this theory by addressing abnormalities in an area of the body that has the ability to significantly affect global autonomic physiology.

## **CONCLUSION**

The single most important factor in the management of these cases was our ability to objectively monitor the adjustment's affects on the patient's neurophysiology. Many different examinations for “subluxation abnormalities” are used in our profession such as leg length, cervical challenge, motion and static palpation, and others. However, these tests lack objectivity, posses inherent errors, and have no confirmation of their ability to monitor neurophysiology (29-32). Infrared imaging, however, has been researched for over 30 years compiling almost 9,000 peer-reviewed and indexed studies confirming its use as an objective measure of neurophysiology. By using this

technology, our center has been able to consistently determine the correct adjustive procedures that produce reproducible and dramatic positive neurophysiologic improvements in our patients.

Considering that our profession at large maintains, and our educational institutions have resolved (33), that homeostasis is dependent upon coordinated neurophysiology, then we must directly and objectively monitor this system as an outcome measure to our care. But not just any method will suffice. We need to directly examine the autonomic nervous system if we are to monitor the global systemic aspect of the nervous system's control and the affect we are having with adjustive procedures. Digital infrared imaging completely fulfills this need by directly observing the function of the sympathetic nervous system. Paraspinal scanning objectively measures the autonomic changes of all 32 spinal nerves as they exit to effect visceral and systemic regulatory functions. Since testing does not involve patient compliance, such as movement or a verbal response, digital infrared imaging becomes as objective a test of neurophysiology as possible.

To what magnitude the upper cervical spine is involved in the genesis of FMS and CFS remains to be seen. A cohort of 23 subjects represents only a pilot study at best. However, in an atmosphere where much of the public see our profession as useful for neck and back pain treatment at most, patients with complex disorders are left unaware of the possible benefits of care. The body of literature detailing the upper cervical spine's role in affecting global physiology is substantial. Further research into this area of the spine, combined with objective monitoring of neurophysiology, may reveal that chiropractic does indeed offer a consistent conservative solution for patients with fibromyalgia and chronic fatigue syndrome.

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