Fibromyalgia and Chronic Myofascial Pain: A Patient Review
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Scope and Purpose: Between 1992 through 1999, over 1,000 patients with diagnosed or suspected fibromyalgia syndrome (FMS) and/or chronic myofascial pain (CMP) were interviewed. A chart was prepared for each patient, combining information from these interviews with information gleaned from medical records they had provided. While further insights were gained over the course of gathering these data, the information presented here is summarized only from those questions which did not change and which were posed to each of these greater than 1,000 patients. In 2003 and 2004, two hundred of these patient charts were randomly selected and reviewed to identify and assess possible symptom clusters and patterns.

Definitions:

Myofascial Trigger Points (TrPs) are areas of hyperirritability caused by excessive release of acetylcholine at dysfunctional motor end plates. [1, 2] Referred pain from myofascial TrPs characteristically produces recognizable regional pain patterns. These primary TrPs can develop satellite and secondary TrPs, and may, in some patients, develop composite pain patterns covering much of the body. There is painfully restricted stretch range of motion, often with apparent muscle weakness due to inhibition, and/or autonomic symptoms. When the term TrP is used in this patient review, the essential criteria used to define it are: palpable taut band; exquisite spot tenderness of a nodule in a taut band; patient pain recognition of current pain complaint/referred pain pattern by pressure on the tender nodule; and painful limit to full stretch range of motion. [1] Visual or tactile identification of twitch response and/or pain or altered sensation in the expected pain pattern distribution and/or associated proprioceptor and autonomic dysfunction on compression of tender nodule were at times confirmed. A myofascial TrP creates a localized region of great oxygen and energy demand; because of limited supply of both, a TrP contains multiple foci that are in constant energy crisis.

Central TrPs are usually in the belly of muscle, where the motor endplates lie. They cause local tenderness, referred pain, altered sensation, referred motor dysfunction, and referred autonomic changes due to sensitization of local nerves and induced central nervous system changes.

Attachment TrPs occur in areas of tenderness where the muscle attaches to other structures, resulting from the inability of the muscle attachment to withstand the sustained tension produced by the taut band. In response, these tissues develop

changes that are likely to produce irritants which could sensitize local nociceptors. [1, p 76] Attachment TrPs are caused by the sustained tension of central TrP-involved muscle fibers.

Chronic Myofascial Pain (CMP) is used in this review to describe those conditions when a primary TrP has developed secondary and/or satellite TrPs, whether these be active or latent, in more than one quadrant; or if there are tissue changes such as fibrosis or multiple attachment TrPs associated with one or more primary TrPs. CMP is not to be confused with the generalized term, "chronic pain syndrome", which is often dismissed as psychological, nor used as synonymous with temporomandibular dysfunction. In CMP, the layers of fascia tend to stick to other microscopic fascial layers and to other tissues. The fascia loses elasticity, and this process compromises function, causes muscle weakness and may cause pain. Autonomic function and proprioception may be disturbed. Chronic myofascial pain is a primary cause of disability, and may develop secondary to trauma such as low back surgery, cervical whiplash, overuse, or repetitive strain. CMP often complicates other medical illnesses and injuries.

Fibromyalgia Syndrome (FMS) as used in this review refers to the American College of Rheumatology research definition of widespread pain and mild or greater tenderness in greater than or equal to 11 of 18 tender point sites. [3] FMS is essentially a centrally generated hypersensitivity to painful stimuli. [4] There is also body-wide allodynia, the sensation of pain from normally non-painful stimuli. Lack of restorative sleep is a common symptom. [5] It is vitally important to understand that FMS is not primarily a musculoskeletal condition, but is a dysfunction of the central nervous system function.

Observations:

Table 1. Symptoms in Numerical Order

Post-nasal drip	189
Fatigue	160
Sleep unrestorative	149
Short term memory impairment	144
Trouble concentrating	144
Sensitivity to cold	143
Morning stiffness	142
Numbness/tingling	131
Difficulty getting out known words	127
Muscle twitching	127
Nail ridges	125
Handwriting difficulties/pain	124
Headaches	124
Irritable bowel	121
Mood swings	118
Weak ankles	118
Carbohydrate cravings	117
Unaccountable irritability	114

Table 2. Symptoms in Alphabetical Order

Adrenalin surges	21
Allergies	73
Appendicitis-like pains	49
Balance problems/staggering gait	88
Belly fat pad	15
Bloating	102
Bothered by pressure of glasses/ headbands/coats	57
Breast pain	70
Bruising	45
Bruxism	54
Buckling knee	102
Burping	14
Carbohydrate cravings	117
Carpal-tunnel-like pain	64
Chest pain	24
Chest tightness	29
Chocolate cravings	109

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Chest tightness	29
Motor coordination problems	29
Chronic cough	28
Sensitivity to environmental	28
Night sweats	25
Sensitivity to blackfly/mosquito bites	25
Chest pain	24
Dry nares with bleeding	22
Adrenalin surges	21
Nails that curve under	21
Weight loss	21
Vision "Floaters"	20
Deep hip pain	19
Immune dysfunction	19
Raynaud's	18
Hair loss	16
Sleep Apnea	16
Belly fat pad	15
Burping	14
Eye dysfunction	11
Ingrown hairs	11
Tennis elbow	11
Hears florescent lights	9
Dysnomia	8
Choking on saliva	7
Ear Dysfunction	6
Impotence	6
Eye pain	5
Sicca	5
Ear Stuffy	4
Ear Pain	3

Table 2. Symptoms in Alphabetical Order

Sicca	5
Sleep:	
Apnea	16
Unrestorative	149
Sore spot on top of head	61
Sore throat	84
Stair climbing problems	113
Stiff neck	40
Stripe/check patterns cause dizziness	55
Sweats	47
Swollen glands	102
Tennis elbow	11
Thick secretions	48
Thumb pain/tingling numbness	51
Tight Achilles tendons	60
Tight hamstrings	63
Tilted feeling when cornering in car	39
Tinnitus	111
TMJD	106
Trouble concentrating	144
Unable to recognize familiar surroundings	42
Unaccountable irritability	114
Unexplained toothaches	80
Vision:	
Blurry	67
Changing	64
Double	31
"Floaters"	20
Perception problems	101
Weak ankles	118
Weak/painful grip	75
Weakness	41
Weight gain	101
Weight loss	21

Comments:

Of the 200 patients, 189 had either FMS, CMP or both. The remaining 11 patients were those who had only been suspected to have these conditions and their data are not included here. Symptoms from these 189 patients are presented in Tables 1 and 2. To diagnose these conditions, the diagnostician must be familiar with single muscle TrPs, combined and overlapping complex TrP pain patterns, and CMP occurring in the context of other conditions such as FMS. In many cases, the TrP component had been undiagnosed or misdiagnosed. Some of these patients had CMP only on one side, or only on the upper or lower half of the body. Many had TrPs in at least three quadrants. Some had TrPs in all quadrants, with multiple TrPs occurring in many layers of many muscles. Some had lack of muscle definition due to severe fibrosis. That is long-standing, full-blown bodywide CMP. By the time a patient reaches that state of CMP, there are usually multiple perpetuating factors. Many of these patients had both FMS and CMP, as well as

other conditions, and some patients had been exposed to work hardening, weight training and other inappropriate physical therapy.

For many years, the emphasis has been on *pain* caused by myofascial TrPs, because the pain can be so prominent. *But TrPs can also cause muscle weakness and restricted range of motion***.** Physical and occupational therapists, as well as doctors, often recommend strengthening exercises with myofascial TrPs without understanding that it is the TrP that is inhibiting the muscle and making it weak. ***You cannot strengthen a muscle with a trigger point***. The muscle is already contractured and tight. Strengthening exercises and repetition exercises will worsen the TrP, and may cause the development of satellite and secondary TrPs.

Some of the latter patients did not have full-blown CMP when they started the inappropriate therapies, but were disabled by the time they finished or dropped out of the training. "A considerable portion of the chronic pain due to myofascial TrPs could have been prevented by prompt diagnosis with appropriate treatment...When the myofascial nature of pain is unrecognized...the symptoms are likely to be diagnosed as neurotic, psychogenic, or behavioral. This adds frustration and self-doubt to the patient's misery and blocks appropriate diagnosis and treatment.... The total cost is incalculable, but enormous, and most of it is unnecessary." [(1)]

There is widespread lack of training in diagnosis and treatment of myofascial TrPs, and widespread misunderstanding in diagnosing and treating FMS. Many of these patients described worsening conditions with time, yet research indicates that FMS is not progressive. [(6)] If it is getting significantly worse with time, there is at least one perpetuating factor that is not being addressed. Doctors and other care providers often noted specific indicators of developing FMS and/or the development of secondary and satellite TrPs without knowing their significance. In many cases, new symptoms were dismissed as part of FMS or diagnosed as carpal tunnel or other conditions without checking for possible myofascial TrPs. Patients were at times given diagnoses such as "atypical lupus," "atypical rheumatoid arthritis" or "atypical multiple sclerosis" only to have these diagnoses refuted by other doctors. One patient reported that her blood work was ANA positive sometimes, the lupus results positive only once, yet she developed the butterfly mask every year. A subset of the FMS patients had positive ANA results. One patient did have co-existing drug-induced lupus. Two patients had been diagnosed with systemic lupus erythematosus, and one diagnosis was later refuted. One previously undiagnosed patient was diagnosed with systemic lupus. Four patients had Sjogren's syndrome diagnosed previously, and one had scleroderma. One was diagnosed with rheumatoid arthritis (RA) but neither FMS nor CMP (she had all three). Two others had been diagnosed with RA but the diagnoses were later refuted. Three were diagnosed with dystonia but only one diagnosis was confirmed. One had been diagnosed with Reiter's Syndrome but had no sign of any inflammatory processes. She did have multiple TrPs, including lower abdominal TrPs causing bladder urgency, and sternocleidomastoid (SCM) TrPs

causing eye dysfunction. One patient was diagnosed with ankylosing spondylitis and this was confirmed. Two were diagnosed with psoriatic arthritis, and one was confirmed and one did not have any form of arthritis. Three may have had post-polio syndrome as well as FMS and TrPs. There was one case of benign monoclonal gammopathy. One patient had been diagnosed with FMS, but instead had facial TrPs perpetuated by Ehlers-Danlos Syndrome. One patient without either FMS or CMP had rhabdomyolosis, possibly drug induced. One patient had depression and a few TrPs.

Many patients came with the diagnosis of FMS but did not have any signs of central sensitization. They did have multiple TrPs, and some had CMP with complex overlapping pain patterns. The areas outside the pain patterns were not painful and had no symptoms. Some patients had bilateral carpal tunnel surgery and some had repeat surgery on the same side, without pain reduction. One patient had 4 back surgeries; including the removed of the tail bone and repair of 3 ruptured discs, a left knee replacement and was waiting for the right to be replaced. There had been no check for TrPs that could be involved in nerve entrapment. When I touched the TrPs involved, the patient recognized the pain pattern. Many patients had had multiple surgeries, and these patients often developed adhesions and scar TrPs.

The central sensitization of FMS can be both *initiated* and *perpetuated* by peripheral pain sensations [7], such as those caused by myofascial TrPs. The symptoms of myofascial TrPs can be perpetuated and affected, and sometimes amplified, by the central sensitization and imbalances of FMS. The histories of many of these patients indicated that the presence of myofascial TrPs preceded central sensitization.

Many physicians had not even checked the patients for tender points, but simply ascribed widespread pain automatically to FMS. We are now aware of the central sensitization and abnormal temporal summation pain (wind-up) associated with FMS, as well as widespread pain, fatigue, sleep abnormalities and distress. [7] Research has found that spinal glial cell activation is part of central sensitization, and interstitial swelling can be an integral part of this process. [8] Central sensitization may be a central nervous system (CNS) response to an attack, such as that from nerve injury, inflammation, infection or other source. Also interesting is the observation of cellular adhesion molecules in the lumbar spinal cord following peripheral inflammatory stimuli. [8] This may indicate a similar process occurring in the central nervous system similar to the myofascial cellular adhesion in response to mechanical or biochemical trauma. The start of each case of FMS probably has multiple causes. “A combination of multiple, mild impaired responses may lead to more profound physiologic and clinical consequences as compared with a defect in only one system, and could contribute to the symptoms of fibromyalgia.” [9] Initiating factors for FMS might even begin before birth. Some patients were illegitimate, or born of otherwise stressed mothers. Research indicates that exposing a developing fetus to stress biochemicals can impair coping

and hypothalamus-pituitary-adrenal (HPA) axis regulation after birth. [10] These patients often had histories indicating that they were born with sleep dysfunctions, sensitivities, and other problems. One patient was told she was "born with arthritis," although she did not have rheumatoid arthritis. Some patients started with TrPs, some with central sensitization. Sometimes trauma of acute or repetitive nature caused both simultaneously, although in acute cases it often took the TrPs a while to surface as the patient started to move.

No two FMS patients are alike. They don't even all share the same pain processing dysfunctions. [11] Each patient is a unique individual, with unique needs, and must be so treated. Yet many patients had been placed in arthritis classes and rehabilitation programs that showed no understanding of the basic neurohormonal imbalances of FMS. Co-existing conditions were not sought nor identified in many cases. "Most of the six million Americans with fibromyalgia have at least one associated syndrome which mandates specialized attention in addition to traditional therapeutic approaches. The successful treatment of fibromyalgia-associated syndromes improves the symptoms, quality of life, and prognosis of fibromyalgia." [12] For example, although many patients were diagnosed with FMS and FMS is associated with HPA-axis dysfunction and HPA-axis dysfunction is associated with insulin resistance [13], these patients were not checked for insulin resistance, in spite of abdominal obesity, high cholesterol, craving for carbohydrates, and hypoglycemic symptoms. [13] What I had considered the FMS belly fat pad at the start of the patient interview process seems to be, on retrospect, linked to insulin resistance.

Some patients with multiple myofascial TrPs were denied any treatment or adequate treatment for pain because they did not have 11 of 18 tender points indicative of FMS. Their doctors did not recognize myofascial TrPs. They thought erroneously that trigger points were part of fibromyalgia, and much of the literature reflects that lack of knowledge. Research indicates that patients who do not yet have the 11 of 18 tender points may benefit from *aggressive* pain control to *prevent* further central sensitization. [14] Some of these patients developed FMS that might have been prevented had they only received adequate care. Some doctors tested for pain on areas that were not part of the accepted FMS tender point diagram and pressed myofascial TrPs instead. When the patients reacted, they were accused of malingering.

CMP in this review is not defined by specific time limits, due to the nature of the formation of myofascial TrPs. I do not believe that chronicity can be defined as simply a function of time. For example, one patient had multiple TrPs, occurring in three quadrants, and had no perpetuating factors. She had had TrP pattern pain for over two years. She came with a diagnosis of FMS. The TrPs were not associated, each being the result of individual falls or other physical trauma. There were no signs of tissue change. I did not consider her to have chronic myofascial pain, nor even chronic myofascial TrPs. She certainly didn't have FMS. What she had was chronic misdiagnosed and untreated TrPs. With a diagnosis and

proper treatment, she was fine within weeks. Multiple TrPs can arise bodywide in a trauma case, but if they are promptly treated and the patient has no perpetuating factors and the TrPs are diagnosed and treated properly, there is no development of CMP.

The most common symptom reported was post-nasal drip. Of the 189 patients with FMS, CMP or both, 189 reported this symptom. This result was unexpected. In 1992, an article linked chronic rhinitis to FMS. [15] This team studied 47 consecutive patients with allergic rhinitis in a general allergy clinic, and found congestion in 91%, rhinitis in 87% and postnasal drip in 83%. Forty-nine percent met the ACR criteria for FMS, and the team concluded: "Rhinitis...is associated with fibromyalgia and may be an underdiagnosed but important causative factor." Yeast or mold sensitivity was reported by 82 patients. One review noted how neurogenic mechanisms can complicate sinusitis. [16] Stimulation of nasal sensory nerves leads to pain and congestion. Pain receptors cause release of substance P, stimulating mucosal defense mechanisms. Sympathetic dysfunction then can cause sinuses to fill and the mucosal lining to thicken. Fibromyalgia is associated with sympathetic hypersensitivity.

On March 23, 2004, at the annual meeting of the American Academy of Allergy, Asthma and Immunology in San Francisco, a paper was presented. A Mayo Clinic team of physicians led by David A. Sherris found that airborne fungi commonly found in the mucus linings of the sinuses can adversely affect individuals prone to chronic sinusitis. These fungi provoke an immune response, which in turn attacks the fungi, resulting in symptoms of chronic sinusitis. Could this immune response provoke central sensitization? The team ran a placebo-controlled, double blind pilot study using Amphotericin-B intranasally. Seventy percent of the linings of the sinus membranes of those patients on the drug decreased in thickness, and the symptoms abated. Approaching chronic sinusitis as an immune disorder creates a different perspective, especially if FMS is part of the picture. And myofascial TrPs can complicate it as well.

Specific head and neck TrPs can cause drippy nose and congestion. Trigger points in the sternocleidomastoid muscles (SCM) alone can cause, among other things, coordination problems, proprioceptor dysfunction, dizziness, imbalance, neck soreness, a swollen glands feeling, runny nose, maxillary sinus congestion, tension headaches, eye problems (tearing, blurred or double vision, inability to raise the upper eyelid, dimming of perceived light intensity), spatial disorientation, postural dizziness, Many patients reported what I call the FMS/MPS sinus syndrome (at the time the questionnaire was developed the term used was MPS rather than the current CMP). At night, as a patient slept on his/her side, the sinus congestion would develop on the side close to the pillow. If they rolled over to the other side, the congestion would move as well. This often resulted in frequent bed position shifts, and the development of worsening SCM TrPs as the head was raised during roll-over. One patient reported face surgery to open sinuses, but associated TrPs

were untreated and the patient remained symptomatic until they were treated.

In myofascial pain, local tissue changes are very similar to mechanically induced muscle damage. In acute stages, they are accompanied by edema, and in chronic forms by local fibrosis. [17] Fibrosis or tissue swelling may make it impossible to palpate for TrPs, but if you take an adequate history, check range of motion and look for the patterns of pain and associated symptoms, you will know where the TrPs probably are located. Attachment TrPs often respond well to ice, whereas central TrPs, unless there is nerve entrapment, often respond better to moist heat. [1] The patient may often recognize the TrP pain patterns. As the patient gets better and perpetuating factors are brought under control, the individual TrPs will appear. This takes time, patience, and endurance, but return of function is the key.

Some TrPs are small, and some are large. It depends on how many contraction knots are in place, as well as how much infiltration of biochemicals and excess fluid are in the area. EMG studies indicate "...in muscles with active TrPs, the muscle starts out fatigued, it fatigues more rapidly, and it becomes exhausted sooner than normal muscles." [1 p, 22] Exercise programs must reflect this if TrPs are part of the picture. "Myofascial TrPs are aggravated by high histamine levels and active allergies." [1 p105] This can be difficult for people who have allergies and have TrPs. Red welts may appear from even gentle bodywork. Antihistamines may help, but patients with FMS and allergies may already be on maximum dosage of antihistamines. The biochemicals entrapped in myofascia and released by successful treatment may temporarily worsen FMS or even cause flare. Bodywork for TrPs may be painful due to hyperalgesia and allodynia due to co-existing FMS, and the effects may be delayed.

One doctor had noted that the patient required a physical therapy stretching and strengthening exercise program. A later comment indicated that the patient reported that the patient had given the physical therapy program her best effort but it did not give her the anticipated help. Some patients dropped out when work hardening and strength training programs caused extreme worsening of undiagnosed TrPs. They were then called noncompliant and in some cases their insurance companies refused to pay for the program, so they went back. They became disabled. Delayed reactions to overworking stressed muscles were common. One patient stated "If I overdo it I pay for it for four days, and the second is the worst."

Therapies such as bodywork and stretching may activate latent TrP. This can be discouraging and frustrating. It's also not uncommon to experience nausea, headaches, and exhaustion after bodywork or TrP injections have moved toxins and wastes from constricted muscles. Patients may need to sleep after a session. The therapy isn't what causes the symptoms, yet this effect had caused many patients to decide that specific therapies or treatments worsened their illness. Any trauma sustained throughout life causes cells and tissues to tend to accumulate

substances in abnormal quantities, a phenomenon referred to as “infiltration” in the older literature. Most commonly, such accumulations consist of molecules that are normally present such as triglycerides, glycogen, calcium, uric acid, melanin, and bilirubin. Successful therapies release these substances. The liver and kidneys can handle only so much of the biochemical by-products released during a good bodywork session at one time, so treatments must be paced. Strenuous activity should be restricted in the 2-3 day post-treatment period. [1, p 186] When TrPs are extremely active, they cause pain even at rest. Trying to force them into action might cause them to contract even more tightly in response, called a rebound contraction.

On review, symptoms seemed to come in clusters in patients, and it was often possible to see the probable pattern of TrP cascades. Patients with allergies causing constant respiratory infections often had nasal congestion, itchy skin, tinnitus, dizziness when turning the head fast, swollen-glands feeling, unexplained toothaches, headache, itchy ears, drooling during sleep, problem with pressure of hats and headbands or glasses, eye and ear dysfunctions, and chronic cough. At times these started with a long or painful session of dental work, or an upper respiratory infection. These patients usually had neck and facial TrPs. TrPs often developed with childhood allergies or chronic runny nose. Some reported massive amounts of dental work with insufficient anesthesia or lack of time during work to stretch. Bite was often corrected without recognizing and correcting TrPs first, so that when TrPs changed, the bite was not corrected.

One patient told me his problems began after dental equilibration about 10 years ago. The stubs of his front teeth had been capped and uncapped several times with no relief of pain. He had multiple facial TrPs. Pain spread to his tongue and hard palate problems developed. Surgery was considered but not done. Tightness spread to his shoulders, trunk and finally encompassed his whole body. Many patients reported similar symptom progression, with frequent tonsil and ear infections in childhood resulting in multiple antibiotic use. These patients often developed frequent candida infections. Irritable bowel often developed after antibiotic use, infection or food sensitivity, and then was perpetuated by abdominal and pelvic floor TrPs. Frequently, another antibiotic was given to stop the diarrhea that may have resulted from abdominal TrPs. Bloating and nausea were often tied to upper abdominal TrPs as well as abdominal TrPs. Irritable bladder was often part of the picture, as was vaginismus, and some patients developed stress incontinence. This incontinence was sometimes reversible with myotherapy, even when the patient had had incontinence for over 10 years.

In one case, scar TrP injection relieved both incontinence and urinary urgency. The patient had been diagnosed with interstitial cystitis (IC). After proper treatment, she still had TrPs and FMS, but her urinary symptoms were gone. “Referred pain and motor activity to the pelvic floor muscles (sphincters), as well as to the pelvic organs, can be the sole cause of IC, IPP, and irritative voiding dysfunction…” [18] Many patients (male and female) had developed sexual

dysfunction, and this was often relieved by appropriate TrP therapy.

Many women started TrP pain with their first menses. The pain eventually spread down the legs and up the back. Satellite and secondary TrPs were present in these areas. Some woman started TrP pain with a pregnancy. One woman and two men developed chronic pain after massive doses of steroids for other conditions. Several men had back injuries from lifting and developed spreading pain that eventually included prostadynia. This pain was reversed by TrP therapy. In one case, the pain had been disabling. In another, it had been severe for over five years.

Patients reported fibroadenoma, lipomas, fibroids and cysts (dermoid and ovarian), overgrown hair and raised scars. They reported scarring from minor injuries, and fragile skin along the cuticle. The cuticle itself is often overgrown and thick. One had bleeding fingers from "easy work" filing a card catalogue. Many of these patients also reported thick secretions including saliva, mucus, eyelashes stuck together when they woke up, difficulty cleansing themselves after bowel movements, difficulty getting mucus stuck on eyeglasses, etc.

Cognitive deficits reported included short-term and long-term memory impairment, difficulty getting out known words especially nouns and pronouns or nouns and names, difficulty multitasking, difficulty doing tasks in sequence, hearing sounds coming from a different direction than which they issued, inability to tolerate stimuli such as a crowd or mall or movie, directional disorientation, spatial disorientation, spelling difficulty, inability to recognize familiar surroundings, typing or writing words with letters in improper order, trouble concentrating, confusional states, and a fugue-like state. The latter could occur during a conversation, or during a simple task such as putting on socks in the morning. One sock would be on, and then the person would sit and stare into space until something would jog them back into motion, or their brain caught up processing. Often, as patients improved, they became aware of shifting into the fugue state and could make a successful deliberate shift from it. Of all the symptoms, including pain, the cognitive deficits were most often considered as the most life-disruptive. Ten patients referred to brain "storms." This symptom was not part of the questions. These referred to states of hypersensitivity and were described as "brain frenzy" or the sense of a hyperactive and out-of-control brain that could not slow down to normal. One called it "warp speed." This often occurred at night when they were trying to get to sleep. Some patients called these "adrenalin surges." Some patients reported syncope, and some had documented seizure states or seizure-like states. These occurred more frequently (or only) when the pain was more intense.

Many patients reported amplification of pain and other sensations, hyperalgesia, allodynia, amplifications of dysfunctions including those of the autonomic nervous system, proprioception and mechanoreception. Many indicated what now would be called interoception dysfunctions. [19] Myofascial TrPs can cause peripheral

nociceptive perpetuation of central sensitization, proprioceptive and autonomic concomitants, and I believe that either or both FMS and CMP may be involved in interoceptive dysfunctions. Although the SCM TrPs are often associated with proprioceptive dysfunction, I have observed that in many other muscles TrPs may also be involved. For example, medial pterygoid and other TRPs may be associated with frequent biting of the inner cheek. This problem was often mentioned. It often seemed as if the patient did not know where the teeth were in relation to the cheek. Others described frequent inadvertent biting of the tongue. Several TrPs were active in the area, and they were all treated successfully, and the problem disappeared. Patients with multiple pelvic floor TrPs reported problems cleansing themselves after bowel movement and urination. Others described itches that they could not quite locate, and they often spent much time trying to figure out the origin of the itch. If the itch source could be found, it often responded to an ice compress. Several patients had what they called permanent goose bumps on their upper arms, and a few had them on their outer thighs as well. This pilomotor response can be secondary to myofascial TrPs. [(1)] Several reported unexplained episodes of syncope. Some of these patients had Neurally Mediated Hypotension, and some had Metabolic Syndrome, but some did not. Fugue states similar to petit mal seizures were not uncommon. It was as if the brain had to catch up processing and took a time out. This could occur mid-conversation. Several patients reported sensory distortions and spatial disorientations. These included difficulty hearing over the phone, altered taste of specific foods, unusual response to specific tactile textures and vibrations, inability to differentiate right from left, or mistaking the directional origin of a sound.

There seemed to be a subset of patients with FMS who were tall and thin, but I could not find a meaningful specific common pattern among their symptoms. There was also a subset of FMS patients who were paradoxically stimulated by diphenhydramine, nortriptyline hydrochloride, paroxetine hydrochloride and venlaxifine hydrochloride. If one of these medications stimulated them, they were likely to be stimulated by the others. Family members who also had FMS, when information was available, usually had the same pattern.

Patients often recognized the term environmental sensitivity. Both electromagnetic hypersensitivity (developing health symptoms due to exposure of environmental electromagnetic fields) and electromagnetic sensibility (the ability to perceive electric and electromagnetic exposure) have been scientifically documented. [(20)] People with electromagnetic sensibility do not necessarily have electromagnetic hypersensitivity. Reports of symptoms varied, including affecting watches (stopping them or affecting the timing), ability to hear dog whistles, ability to hear aurora or infrasound, stopping clocks in cars, draining phone batteries, hearing florescent lights, hearing electricity, feeling electricity. Some reported that their brains seemed to be “wound up” by electrical storms, the full moon, aurora, wind (especially Mistral, Coen, Santa Anna, etc.), solar flares and coronal mass ejections. Some reported empathic or psychic sense, or sixth sense. Others reported affecting street lights, VCRs, computers, or other electronic

devices. This symptom was one of the least reported to their doctors. I have no proof that these are related to CNS hyperalgesia and allodynia of FMS and altered sympathetic activity. One review indicated that electrical fields can have greater effect on neural tissue "...in conditions where field sensitivity is enhanced." [21] Biofields, including electromagnetic fields, affect the structural extracellular matrix (ECM) proteins. [22] Many patients described these symptoms as worsening in times of heightened pain or anger. With the rapid weight fluctuations and interstitial swelling reported by many of these patients, there are many potential variables. There is much we do not yet know about the interactions between biofields at the cellular membrane level. We do know that exposing the human head to a specific electromagnetic field can alter pain sensitivity and other sensory parameters. [23] It is clear that we need more research in this field, but for this to happen patients must be able to trust their care providers. Right now they are not being believed about even the more common and well-known FMS and CMP symptoms.

Many patients reported thermoregulatory difficulties and symptoms of interrupted peripheral circulation and/or microcirculation dysfunction. Many developed typical cold spots, especially on the buttocks or thighs. Some patients were afraid to yawn because their necks went into spasm or their jaws locked.

Subsets of FMS and CMP patients may be like Venn diagrams, overlapping. It is still important to define them, or attempt to do so, because the subsets may offer clues as to possible effective treatment regimens. This starts with listening to the patient, knowing what to ask, and knowing what to look for. After one interview of a patient and her spouse, one woman said, "My husband wants to know how you already know so much about me!" After I described a fugue state, one man asked, "How do you know so much about what goes on in my bedroom?" I am not psychic. I knew what to look for and what to ask, because I listened and I believed and tried to find out why.

A child with chronic lack of restorative sleep needs attention. A child with growing pains needs attention. Trauma patients need adequate treatment of pain, and adequate follow up on soft tissue injuries including range of motion studies. One woman with menstrual cramps so severe she was losing consciousness was told by her doctor there was nothing he could do. She needed to find a way to keep busy and get her mind of fit. One woman with incapacitating headaches was told by her nurse practitioner, "Go home, honey. Keep busy, be happy. Find yourself a good man. Have a baby." One construction worker was called a wimp by his boss, and then told by his doctor to work more hours so that he would get tired enough to sleep.

Fibromyalgia may be a major dysfunction in the CNS, and a myofascial trigger point may be a major dysfunction at the motor end plate, but my observation is that the main problem is a major dysfunction in the medical care system. Inattention to these "invisible" conditions often results in depression, disability and

even suicide. The costs are high. It is essential that doctors and other care providers, including insurance providers, are adequately trained concerning myofascial TrPs and FMS. Chronic myofascial pain, like central sensitization, is often iatrogenic. Many cases of chronic pain may be preventable.

Developing central sensitization can often be halted by recognizing signs and symptoms, identifying initiating and perpetuating factors and bringing them under control. Patients often develop CMP because their doctors don't recognize single TrPs and treat them promptly, and they don't identify and control perpetuating factors.

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