

What Your Neurologist Should Know About FMS and CMP by Devin J. Starlanyl

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Please read "What Everyone on Your Health Care Team Should Know About FMS and CMP" and "Headaches Due to TrPs."

Many fibromyalgia (FMS) symptoms can lead to a call for a neuro consult (Simms and Goldenberg 1988). So can myofascial trigger point (TrP) symptoms (Simons, Travell, Simons 1999). FMS itself is basically a neurological condition (Bradley, McKendree-Smith, Alarcon et al. 2002). There is, among other things, a state of central sensitization leading to allodynia and/or hyperesthesia. Many patients will come to you undiagnosed or misdiagnosed (Buskila, Neumann, Sibirski, et al. 1997). They may also be under-medicated due to unwarranted fear of addiction on the part of the primary care physician. Pain further stresses the body, leading to further hypersensitizing of the CNS (Lebovits, Florence, Bathina et al. 1997). Research shows that the recent increasing medical use of opioids to treat pain hasn't cause an increase "...in the health consequences of opioid analgesic abuse" (Jornason, Ryan, Gilson et al. 2000). Pain is a perpetuating factor for both FMS and CMP, and as such, must be brought under control with available medicinal and nonmedicinal options.

Functional MRI studies indicate that FMS is associated with augmented pain processing (Gracely, Petzke, Wolf et al. 2002). There may be multiple hormonal and autonomic imbalances in FMS, leading to profound physiological and clinical consequences (Adler, Manfredsdottir, Creskoff 2002). Neuroplasticity plays a significant role in chronic pain states (Coderre, Katz, Vaccarino, et al. 1993). The development of central sensitization from repeat or chronic trauma is well documented (Yaksh, Hua, Kalcheva et al. 1999). In FMS, this trauma may be physical or biochemical.

Regional cerebral blood flow abnormalities have been documented in FMS (Mountz, Bradley, Modell 1995; Johansson, Risberg, Rosenhall, et al. 1995). Central sensitization of FMS may be maintained by peripheral stimulation, such as myofascial TrPs (Staud, Smitherman 2002; Borg-Stein 2002). It is vital that the myofascial TrPs be identified and treated promptly, because many of the "Fibromyalgia" symptoms are not part of fibromyalgia at all, but are part of chronic myofascial pain, and must be treated differently. Following the last stimulus in a series, the after sensations were greater, lasted longer and were more frequently painful in FMS (Staud, Vierck, Cannon et al 2001). Your FMS patient will hurt more and hurt longer from stimuli that others may not find painful at all.

FMS is associated with dysautonomia (Raj, Bruillard, Simpson. 2000). There may be many dysfunctional hormonal axes, and these may be difficult to pinpoint. End-hormone HPA axis glucocorticoids have multiple roles. They inhibit CRH, LC/NE and beta-endorphin systems, stimulate the mesocorticolimbic dopaminergic system and CRH peptidergic central nucleus of the amygdala, and inhibit the pituitary gonadotropin, GH and TSH secretion. They promote tissue resistance to a variety of hormones. They suppress 5' deiodinase, which converts T4 to T3, and promote Metabolic Syndrome (Tsigos, Chrousos G. 2002). Metabolic Syndrome itself is a common perpetuating factor for both FMS and CMP. "Chronic imbalance of the autonomic nervous system is a prevalent and potent risk factor for adverse cardiovascular events, including mortality"(Curtis, O'Keefe 2002). Please don't take these patients lightly. In most cases, they will come with a history of being dismissed and perhaps mistreated by your medical colleagues.

FMS is real and may be maintained by the sympathetic nervous system (Martinez-Lavin, Vidal, Barbosa, 2002). The stress of having an invisible and misunderstood medical condition, often compounded by lack of support from family, companions and the medical and insurance world, can add to the burdens. "The most aggressive challenges of the FMS concept have been from legal defenses of insurance carriers motivated by economic concerns. Other forms of critique have presented as psychiatric dogma, uninformed posturing, suspicion of malingering, ignorance of nociceptive physiology, and occasionally have resulted from honest misunderstanding" (Rau, Russell 2000).

Fibromyalgia, a neuroendocrine illness, and chronic myofascial pain, a neuromuscular illness, are often confused. Many doctors lack a thorough knowledge of myofascial trigger points, and this often leads to inappropriate treatment. Raynaud's syndrome is often present with FMS (Bennett 1991), and nerve, blood and lymph vessel entrapment by myofascial TrPs can compound the symptoms (Simons, Travell, Simons 1999). The symptoms are signs of impaired circulation, as well as enhanced sensitivity. There may be increased neurogenic inflammation (Littlejohn, Weinstein and Helme 1987), sensory dysfunction (Kosek, Ekholm and Hansson 1996), and altered sympathetic nerve activity (Elam, Johansson and Wallin 1992) in FMS. There can also be orthostatic sympathetic derangement (Martinez-Lavin, Hermosillo, Mendoza, et al. 1997). Multiple FMS symptoms may respond to central acting medications (Suzuki, Dickenson 2002), and the research in this area is very vigorous.

It is important to check the whole patient, as symptoms may develop that are due to combinations of therapies. For instance, toxic optic neuropathy with visual acuity loss, dyschromatopsia, altered light adaption, and evolving bilateral cecocentral scotomas can develop if a patient is on a combination of Zoloft (given for FMS symptoms), melatonin (taken to help sleep) and is on a high protein diet (for Metabolic Syndrome, insulin resistance or reactive hypoglycemia) (Lehman, Johnson 1999). A complete history is most important in these patients.

Myofascial TrPs can mimic so many different conditions, such as visceral pain in the absence of visceral pathology (Gerwin, 2002). To doctors untrained in myofascial referred pain patterns, this may cause patients to be misdiagnosed with somatiform illness. Proprioception dysfunction can be associated with any myofascial TrPs. Some people have reported becoming dizzy to the point of falling just from looking at patterns of light and dark. Some patients even vomit from this and must avoid fabric stores and take care using escalators because of their tread pattern. Patterns of light and dark from trees by the side of the road can cause a petit-mal fugue state, depending on the lighting. It is not uncommon for people with myofascial TrPs to trip over their own feet, bite their tongue, or have disturbance of judging weight held in the hand. Some of this is due to the proprioception disturbances so well documented by Travell and Simons. There may also be a proprioceptive component in FMS. People with FMS also may have indications of a movement disorder and sensory disturbances (Burgunder, 1998). Other physicians may not know about this.

Myofascial TrPs can cause nerve and vascular entrapments. TrPs cause dysfunction before they cause pain and must be suspected whenever there is pain at the end of decreased range of motion. They cause specific muscle weakness. Many TrPs can cause grip failure. Knees, ankles and hips can buckle. A list of common diagnoses can be found in "Travell and Simons Myofascial Pain and Dysfunction: The Trigger Point Manual, vol I ed 2 (Simons, Travell and Simons, 1999, p 37). Thorough knowledge of TrPs and their individual pain patterns and associated symptoms are a necessity for differential diagnosis in most cases. There are many cases of muscle dysfunction that have persisted needlessly for over 20 years, because the TrPs were not diagnosed and properly treated. In some cases, the contracted muscles have pulled bones out of alignment, and degeneration of bone has resulted, not from "aging", but from iatrogenic causes. Early attention to perpetuating factors and treatment of the TrPs can cause sometimes astounding and rapid relief.

Carpal tunnel syndrome is a description, not a diagnosis. The cause must still be identified before it can be corrected. Scaleni, brachialis, brachioradialis, radial wrist extensors, palmaris longus, flexor carpi radialis, pronator teres, opponens pollicis and adductor pollicis TrPs symptoms mimic CTS (Simons, Travell and Simons 1999, p 688). TrPs in the subscapularis muscle can refer pain in a wrist band pattern. One study of over 90 cases of cumulative trauma disorders found that when fibrous adhesions and resulting faulty biomechanics were treated manually, normal function was restored, with relief of symptoms (Leahy and Mock III. 1992). Those researchers found that if a nerve is irritated at one point, it is more susceptible to compression and irritation at other points, so there may be many areas of nerve entrapment if detection and treatment is not timely.

There has been some media attention (and confusion) concerning Chiari Malformation, spinal stenosis, FMS and CFIDS. On March 10, 2000, Barbara

Walters interviewed some doctors who perform this surgery. During the interview, one neurosurgeon mentioned that even heavy coughing could cause narrowing of the spinal canal. It is a fact that any procedure that hyperextends the neck can also cause this narrowing. Medical literature shows that reductions of 1.5 mm or less in the diameter of the spinal canal can come from simple *changes in posture*, such as a rotation in the pelvis (Harrison, Cailliet, Harrison et al. 1999). Levator scapulae and scalene TrPs may be involved in cervical narrowing. It is common to have several areas of spinal rotation in CMP. The connection between Chiari I malformation and orthostatic hypotension is not well supported by research (Garland, Robertson 2001). Patients must be evaluated carefully, and *appropriate* and *thorough* noninvasive treatments tried before such surgery is even considered, and they should *never* be considered for general symptoms of FMS or CMP.

I have heard of countless cases where vertebrae have been fused due to degeneration, only to have the discs above and/or below degenerate, requiring more spinal fusion. This can be repeated and repeated on the same patient, who often becomes increasingly more disabled. If muscles are contracted due to TrPs, they can pull bones out of alignment. Muscle function groups become weakened due to the presence of TrPs, and you cannot strengthen a muscle with a TrP. Strengthening exercises, commonly given, simply make the TrP worse. Then there is more chance of bulging disc and/or degeneration. Dealing with the disc or the vertebrae does nothing to reduce the strain from the muscles. You must deal with the TrPs properly, or surgery may cause even more strain, resulting in more contracture and future problems, as well as “failed back surgery syndrome”. TrPs are more likely to occur in certain muscles in the presence of cervical disc lesions at specific levels (Hsueh, Yu, Kuan et al. 1998). Attention to the TrPs may prevent the need for surgery if the soft tissue problem is caught in time. Even those patients requiring surgery still need attention to the TrPs, before and after surgery, as well as attention to perpetuating factors. This requires that doctors be trained in the diagnosis and proper treatment of TrPs. All too often they inject steroids, which may cover the problem by temporarily relieving the pain, but do nothing for the cause. Myofascial TrP injection training is available, as well as hands-on training in the diagnosis and treatment of myofascial TrPs (see www.painpoints.com).

Eyelid twitching can be due to myofascial TrPs. Check for periorbital TrPs, extrinsic eye muscle TrPs, sternocleidomastoid, the temporalis, and the trapezius TrPs for possible contributors. You may also find other head TrPs. Other muscles twitching can become bothersome. Sometimes there can be a continuous twitch of many muscles. In other cases, one or two muscles will fire off now and then. This may be intensified by mineral insufficiency and/or neurotransmitter dysregulation. Fasciculations and waves of twitches can be caused by low-level TrPs. This has been described by patients as having the nerves plugged in to twinkling Christmas lights. Other people have severe twitches that disrupt their functioning. These can become painful cramping. Check for vitamin and mineral insufficiencies as possible perpetuating factors.

Headaches may be modified considerably by TrP therapy (Simons, Travell, Simons 1999; Krabak, Borg-Stein, Oas, et al. 1996; Graff-Radford, Jaeger and Reeves 1986). There may be abnormal electromyographic results due to nerve entrapment by TrPs, and even abnormal EEGs, although these vary, like the symptoms, from hour to hour and day to day. Please use only surface EEGs. Remember that there is considerable pain amplification in FMS, with amplified and extended wind-up (Staud, Vierck, Cannon et al. 2001), so be as gentle as possible. An examination may boost pain levels for an extended period of time (weeks or longer), causing FMS flare.

There seems to be alterations in the microstructure of sleep in FMS (Drewes, Kielson, Taagholt et al. 1995). Sleep restriction alters the hypothalamic-pituitary-adrenal response to stress (Meerlo, Koehl, van der Borght et al. 2002). Some morning stiffness may be due to the immobility of the night, as well as to the lack of restorative sleep. It isn't so much the amount of sleep we get as the poor quality of sleep (Branco, Atalaia and Paiva 1994; Drewes, Gade, Nielsen, et al. 1995; Horne and Shackell 1991). Sometimes diphenhydramine is enough to help sleep, but it does cause insomnia in a percentage of people. There may be many factors contributing to your patient's lack of restorative sleep, and it is vital that this be addressed as soon as possible. Your patient must feel rested when he or she wakes up. Sleep starts can be common in FMS. Bruxism and restless legs are common in both FMS and CMP.

Cognitive deficits have been well documented as part of FMS (Park, Glass, Minear et al. 2001). They are often called "fibrofog", and can be more disabling and troubling than the pain. Difficulty distinguishing right from left and/or difficulty finding places or following directions is common. Short-term memory problems and confusional states are common. There may be difficulty with multitasking, or performing a number of steps in sequence. This is common in chronic pain states (Gritchnik, Ferrante 1991; Grigsby, Rosenberg, Busenbark 1995). Difficulty getting out known words, especially nouns and pronouns, is part of the "cognitive deficits" package we often get with FMS. In addition, TrPs in speech muscles can create slow, "halted" speech patterns, or garbled sounds. This in no way indicates a lack of intelligence. It does cause a level of frustration and misunderstanding that can add to the patient's burdens. Some researchers have found that FMS causes slowed psychomotor speed in tasks that require sustained effort (Landro, Stiles and Sletvold, 1997). Also, patients may forget things they need to minimize symptoms, and that, plus the additional stress brought about by fibrofog, can lead to a flare.

Chronic pain and injury can cause changes that lead to biochemical trauma and neural plasticity. These changes can take a while to develop. Many cases of FMS and CMP start with whiplash injury, and these effects can add to cognitive deficits. Whiplash can cause later deficits in attention, concentration and memory (Kischka, Ettlin, Heim et al. 1991). Emotional symptoms as well as brain function may be

affected whenever the cervical spine has been injured (Radanov, Bicik, Dvorak et al.1999). It doesn't take an auto accident to supply the force required to do damage. Some "psychological" brain-disconnects after whiplash are a consequence of the whiplash, and not psychological at all (Radanov, Begre, Sturzenegger et al. 1996).

Fibromyalgia and chronic myofascial pain are real, and there is a wealth of research available to help you diagnose them and treat them effectively. You can make a profound difference in the quality of life of many of these patients, and many can regain a measure of function that they thought was lost forever.

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