

Summaries of the Padua symposium on myofascial pain, fibromyalgia, and fascial pain disorders, June 2023, *Aula Falloppio* at the Human Anatomy Institute of the University of Padova, Italy

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Abstract

The International Symposium on Myofascial Pain, Fibromyalgia, and Fascial Pain Disorders was held at the University of Padua, Padua, Italy in June of 2023. This report presents a summary of the presentations from scientists and clinicians from around the world who presented to the symposium. The purpose of this symposium and resultant paper is improve health professional's recognition and understanding of the clinical characteristics, co-morbidities, mechanisms, and treatment strategies for these common conditions to better understand and manage their pain, dysfunction, and quality life.

Key Words: Padua Italy; symposium; myofascial pain; myopain; fibromyalgia; soft tissue pain; abstracts.

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Chronic pain conditions are the top reason for seeking care, the top cause of disability and addiction, and top driver of health care costs, costing more than cancer, heart disease, and diabetes.¹⁻³ The personal impact in terms of suffering, dysfunction, disability, depression, addiction, loss of work, conflict and other consequences is incalculable. Everyone, at some point in their lives, has experienced muscle pain associated with trauma or repetitive strain. Myopain conditions including myofascial pain, fibromyalgia and other fascial disorders are the most common cause of chronic pain and associated with more visits to health professionals than any other pain condition.¹⁻³ Yet, there is a lack of awareness, understanding, and successful management of these soft tissue pain conditions in clinical practice by all types of health professionals.

For this reason, an International Symposium on Myofascial Pain, Fibromyalgia, and Fascial Pain Disorders was held at the University of Padova, Padua, Italy the 16 and 17 June, 2023. This report presents a summary of the presentations from scientists and clinicians from around the world who presented to the symposium. The purpose of this symposium and resultant paper is improve health professional's recognition and understanding of the clinical characteristics, co-

morbidities, mechanisms, and treatment strategies for these common conditions to better understand and manage their pain, dysfunction, and quality life.

Clinical characteristics of Myofascial Pain, Fibromyalgia, and related fascial pain conditions

Myofascial pain (MFP) and fibromyalgia (FM) and other fascial pain disorders are most common of chronic pain conditions. These two disorders have many similar characteristics and may represent two ends of a continuous spectrum. MFP is a regional muscle pain disorders characterized by localized muscle tenderness and pain while FM is a widespread pain condition characterized by soft tissue tenderness, fatigue, and sleep disturbance. In both cases, the pain is described as a "chronic dull aching pain" and is central to the diagnosis of both disorders. MFP is the most common cause of persistent regional pain such as back pain, shoulder pain, tension type headaches and jaw pain, while FM is one of the most common causes of widespread pain in the body. Two studies of pain clinic populations have revealed that MFP was cited as the most common cause of pain responsible for 54.6% of a chronic head and neck pain population,⁴ and 85% of a back pain population.⁵ In addition, a study of prevalence of MFP in a general



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internal medicine practice found that among those patients that presented with pain, 29.6% were found to have MFP as the cause of the pain.⁶ Symptoms of FM are also prevalent in the general population with an estimated 5% exhibiting FM regardless of economic background or ethnicity.^{7,8} MFP is also prevalent in FM patients and often overlooked as a common cause of persistent regional pain in most people with FM. The summary discusses the diagnosis and characteristics of myofascial pain and fibromyalgia.

Myofascial Pain

Myofascial Pain (MFP) is a regional pain disorders and distinguished from the widespread muscular pain associated with Fibromyalgia Syndrome (FM).⁹⁻¹⁴ FM resembles MFP but is broader and more systemic in its pain patterns and tenderness. FM consists of widespread pain and tenderness on palpation. The clinical characteristics of MFP include trigger points (TrP) in muscle bands, pain in a zone of reference, occasional associated symptoms and the presence of contributing factors (Table 1). A TrP is defined as localized deep tenderness in a taut band of skeletal muscle that is responsible for the pain in the zone of reference, and if treated, will resolve the resultant pain. The zone of reference is defined as the area of perceived pain referred by the irritable TrP. The pain is usually located over the

Table 1. *Clinical characteristics of myofascial pain*

Trigger points

Tenderness in the muscles on palpation of the trigger point;
Consistent points of tenderness in taut muscle bands
Palpation alters pain locally or distally;

Pain in Zone of reference

Consistent patterns of referral from trigger points;
Constant dull ache and fluctuates in intensity:
Alleviation with massage, anesthetic, and needling of trigger point.

Table 2. *Clinical characteristics of Fibromyalgia as defined by the American College of Rheumatology*

1. History of widespread pain. Pain is considered widespread when all of the following are present: pain in the left side of the body,
2. Pain in the right side of the body,
3. Pain above the waist, pain below the waist.
4. Axial skeletal pain (cervical spine/ anterior chest or thoracic spine/ lumbar pain)
In this definition shoulder and hip pain is considered as pain for each involved side. Low back pain is considered lower segment pain.

TrP or spreads out from the TrP to a distant site. There are generally no neurologic deficits associated with the disorder unless a nerve entrapment syndrome with weakness and diminished sensation coincides with the muscle TrPs. Blood and urine studies are generally normal unless the pain is caused by a concomitant disorder. Imaging studies, including radiographs and magnetic resonance imaging, do not reveal any pathological changes in the muscle or connective tissue. The affected muscles in MFP may also display an increased fatiguability, stiffness, subjective weakness, pain in movement, and slightly restricted range of motion.¹³⁻¹⁶ The muscles are painful when stretched, causing the patient to protect the muscle through poor posture and sustained contraction. Range of motion in patients with MFP and no joint abnormalities, demonstrated a slightly diminished range of motion (approximately 10%) with pain in full range of motion. This slight restriction may perpetuate the TrP and develop other TrPs in the same muscle and agonist muscles. As mentioned earlier, this can cause multiple TrPs with overlapping areas of pain referral and changes in pain patterns as TrPs are inactivated. The onset of MFP pain is often related to and/or generated by repetitive strain to muscles from posture, tensing habits, and excessive or prolonged use of the muscle.

MFP is frequently overlooked as a diagnosis because it is often accompanied by signs and symptoms in addition to pain, such as coincidental pathology conditions and behavioral and psychosocial problems with few objective measures. Although routine clinical electromyographic (EMG) studies show no significant abnormalities associated with TrPs, some specialized EMG studies of trigger points reveal differences. For example, needle insertion into the TrP can produce a burst of electrical activity and local muscle twitch response when specifically snapping the tense muscle band containing a MFP TrP. Diagnostic and therapeutic injections of local anesthetic into the active TrP will reduce or eliminate the referred pain and the tenderness for both diagnostic and therapeutic. Treatments such as spray and stretch, dry needling, exercise, or massage directed at the muscle with the TrP also predictably reduces tenderness and referred pain. These findings provide evidence of a broad range of objective characteristics in the diagnosis of MFP. The signs and symptoms of MFP may appear to mimic many other conditions including joint disorders including arthritis, migraine headaches, neuralgias, temporal arteritis, causalgia, temporomandibular joint disorders, spinal disk disease, sinusitis, and other pathologies causing confusion in diagnosis.

Fibromyalgia

Fibromyalgia (FM) is also a chronic pain condition characterized by more widespread and persistent non-inflammatory musculoskeletal pain.¹⁷⁻²¹ FM pain is relatively stable and consistent in contrast to MFP, which can vary in intensity and location depending on which muscles are involved. Patients with FM may also have pain in the low back, neck, shoulders, and hips (Table 2). These areas frequently affect MFP reflecting the overlap between the two disorders. Studies demonstrate that the pain in FM is considerably more severe over a larger body area than patients who have non-localized chronic pain conditions. In 2010-11, the American College of Rheumatology confirmed diagnostic criteria based exclusively on Widespread Pain Index (WPI) and the Symptom Severity (SS) Scale. The WPI comprises a list of painful areas that patients report. The SS evaluates the severity of fatigue, waking unrefreshed, and cognitive symptoms and a checklist of symptoms including fatigue, insomnia, morning stiffness, depression, anxiety, and cognitive problems (forgetfulness, concentration difficulties, mental slowness, memory and attention problems, and others. Patients have to state whether or not they have these symptoms. To diagnose FM, one of these two conditions must be fulfilled: a $WPI \geq 7$ and $SS \geq 5$, or a WPI between 3 and 6 and $SS \geq 9$. Similar to the 1990 criteria, it is mandatory that symptoms be present for at least 3 months. Chronic widespread pain (CWP) (defined as pain in the left side of the body, pain in the right side of the body, pain above the waist, pain below the waist, and axial skeletal pain [cervical spine or anterior chest or thoracic spine or low back]). The authors

of the revised 2016 criteria also consider the spatial distribution of painful sites including pain in 4 of 5 regions, called “generalized pain” to distinguish it from the 1990 definition of “widespread pain.”

The prevalence of FM in the general population ranges from 3.7% to 20% with the majority are female between the ages of 30 and 60 years old.⁷⁻⁸ Characteristics that occur in the majority of FM patients include chronic fatigue, stiffness, and sleep disturbance. A variety of associated symptoms also occur in FM patients including irritable bowel, headaches, psychological distress, Raynaud’s phenomena, swelling, paresthesias, and functional disabilities. Central nervous system modulating factors such as stress, sleep disorders, and depression play some role in FM.

List of acronyms

CWP - chronic widespread pain
EMG - electromyographic
FM - fibromyalgia
MFP - myofascial pain
SS - symptom severity
TrP - trigger points
WPI - widespread pain index

Contributions of Authors

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Section 1.0 Characteristics of myofascial pain, fibromyalgia, and fascial pain conditions

1.1. Diagnosis of Fascia in Myopain Syndrome

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Aims: Diagnosis and management of musculoskeletal pain is a major clinical challenge. Following this need, our group has developed a study with the aim to prove the efficacy of an innovative magnetic resonance technique, called T1 ρ , to quantify possible alterations in subject suffering of elbow pain.

Background and Methods: In this pilot study, five patients were recruited presenting chronic elbow pain (>3 months), with an age between 30 and 70 years old. Patients underwent two T1 ρ -mapping evaluations, one before and one after a series of Manual treatments. After the first MRI evaluation, a Disability of the Arm, Shoulder and Hand (DASH) questionnaire was administered to quantify the symptoms and pain intensity. Patients underwent three sessions of manual therapy, once a week for 40 min each.

Findings and recommendations: A statistically significant difference was found between bound and unbound water concentration before and after treatment. Unbound water is related with self-aggregate hyaluronan that generate a high viscous extracellular matrix with consequence stiffness and over stimulation of the free nerve ending. This preliminary data suggest that application of the manual method seems to decrease the concentration of unbound water inside the deep fascia improving the patient symptoms collected with the DASH scale. These results could also explain the change in viscosity perceived by many practitioners during the manual treatment thanks the restoration of the normal property of the loose connective tissue. Being able to identify an altered deep fascial area may better guide therapies, contributing to improve the management of musculoskeletal pain syndrome.

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1.2 The Case for Frozen Shoulder

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Aims: Frozen shoulder from adhesive capsulitis is a commonly encountered clinical complaint. It affects 2-5% of adults over the age of 40, is more common in females (60/40) and generally on the non-dominant side. The natural history occurs over 30 months with three overlapping phases of freezing (I), frozen (II) and thawing (III). Night pain is the common feature of phase I and all phases present with a globally restricted restriction in active range of motion (AROM) and passive range of motion (PROM) of more than 50% in all directions. The presenting clinical picture is a patient with flexion (biceps) and internal rotation (subscapularis) in a type of 'shoulder sling' holding posture/pattern. No treatments have been proven to either reduce the intensity or lessen the duration of symptoms over and above the natural history.

Background and Methods: Two studies were presented examining a myofascial trigger point (MTP) manipulative approach to improving frozen shoulder: The Niel-Asher Technique (NAT). First, results from a randomized placebo controlled clinical trial published in the *British Journal of Rheumatology* in which NAT performed 6 times over 8 weeks demonstrated a significant improvement in AROM when compared to hospital Physical Therapy and Placebo (P=0.002) and Strength and Power (P=0.047), whilst the Shoulder Pain and Disability Index (SPADI) did not show statistical significance (P=0.07) it represented an 80% improvement in reported SPADI. The second study on NAT was a prospective multi-centre study of 163 patient published in the *International Journal of Osteopathic Medicine* (IJOM).

“There was a large and statistically significant reduction in VAS pain scores between baseline and post-treatment assessments. There was a significant improvement in AROM abduction and flexion across time irrespective of the phase of illness (acute / stiff / resolving)”.

Findings and recommendations: NAT is a counterintuitive predictable physical medicine treatment, applying deliberate sequential Trigger Point techniques such as Inhibition Compression (ICT) and Deep Stroking Massage (DSM). It reduces touch to a discreet set of inputs mostly Involving Agonists and Antagonists in a five-steps algorithm. Each step is deliberate to utilize MTP treatment pain to release the shoulder Holding pattern and decrease the pain (which originates in the long head of biceps (bicipital groove). Stimulating MTP's to create layered spinal reflex changes including;

- Khonstamm reflex
- Isometric and Isotonic soft tissue inputs
- Reciprocal Inhibition
- Post Isometric relaxation

NAT is 100% effective for 95% of patients with a mean of 7 treatment sessions. It represents a good opportunity for trigger point and myofascial research since adhesive capsulitis lends itself to a range of objective outcomes.

Key words: frozen shoulder, adhesive capsulitis, myofascial pain, trigger point, range of motion

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1.3 Persistent Post Traumatic Headache: Implications of Physical Findings on the Physiology of Headache Disorders.

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Aims: Pressing head and neck muscles in the Myofascia l can provoke all pain elements of migraine without aura, chronic migraine, and tension-type headaches. (1,2)

Persistent Post Traumatic Headache (PPTH) has similar pain phenotypes as these headaches.(3,4) This study investigates whether the MFE can provoke PPTH pain phenotypes and discusses the implications of these findings.

Background and Methods: The author performed an MFE on 29 patients with PPTH. 22/29 were female. The average age was 49.3 years (range 24-72 years). The average time between injury and evaluation was 20.2 months (range 1-168 months). 5/29 had primary headaches (migraine, cluster, or tension-type) before injury.

Findings and Recommendations: The 29 subjects had the pain phenotypes of migraine in 12 of TTH in 16, and of both in 1. The MFE reproduced the subject's headache pains in 28 of 29 (96.5%). The subject whose headache could not be reproduced by the MFE suffered a skull fracture during his injury. The findings show the MFE can reproduce the pain phenotypes of migraine without aura, chronic migraine, and tension-type headaches. The functional and structural anatomies of migraine without aura and chronic migraine are different from those of PPTH with the same phenotype.(5,6) Since each can be provoked by the same MFE, the functional or connectivity pathways conducting information from the different cortical and cerebellar areas should be similar in order to have similar phenotypes.

Key words: myofascial examination, migraine, Post Traumatic Headache

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1.4 Myofascial pain: Sensitization and Headache

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Aims: To discuss the relationship and mechanisms between headache and myofascial pain.

Background and Methods: Primary headaches are one of the most disabling conditions worldwide, with migraine being the first cause of disability in the age between 15-49, when people are expected to be most active (1). Migraine is a complex cyclic neurological disorder, affecting more females than males (3:1), and is considered a disorder of sensory processing (as it includes not only pain, but also photophobia, phonophobia, osmophobia, nausea among others) (2). Pain is related to the activation of the trigeminovascular system, which is strictly related to the trigemino-cervical complex (3). This complex located at the brainstem level, receives afferents from the upper cervical spine (C1-C2-C3 nerve roots) and the trigeminal nerve, explaining how structures innervated from upper cervical spine roots may refer pain to trigeminal innervated regions (4). When this system becomes sensitized (for example through long-lasting peripheral nociceptive input) it may facilitate pain mechanisms, as well as central sensitization comes into the game. Indeed, central sensitization usually needs a certain amount of ongoing peripheral nociception, as it has been confirmed by studies in which the anesthetization of Myofascial Trigger Points (MTrPs) provoked a reduction of widespread facilitation of pain (i.e. which is related to central sensitization) (5-6).

Several studies suggested that migraine patients have a higher prevalence of musculoskeletal impairments (such as MTrPs) as well as a greater chance of having neck pain (7-9). However the relationship between the neck region and migraine is still source of debate. We recently published a study in which we found that musculoskeletal impairments are present in all phases of the migraine cycle, independent from the presence of concomitant neck pain, and associated with higher disability, frequency, and attacks duration (10). However, in another recent study, we found that migraine patients with concomitant neck pain are worst affected by the disease, having higher frequency, disability, use of symptomatic medication, and with more premonitory symptoms, as compared to migraine patients without concomitant neck pain (11).

Findings and Recommendations: Neck pain alone is not enough to justify physical therapy intervention in migraine patients, but specific musculoskeletal impairments must be assessed and considered, as they may worsen the clinical picture. On the other hand the presence of neck pain is also related to a worse scenario, so a proper investigation of neck pain and neck impairments should guide clinicians into a multidisciplinary management when the neck is involved into migraine-related aspects. Clinicians should perform an adequate assessment of the neck (both pain and impairments), and eventually include its management when the physical assessment and the clinical reasoning confirm that it plays a role.

Key words: Myofascial pain, Sensitization, Headache

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1.5 Long Covid Musculoskeletal Pain.

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Aims: To provide evidence that musculoskeletal pain can be prominent long-term problem in people recovering from a COVID-19 infection ('Long-COVID MSK pain').

Background and Methods: Many viral infections cause myalgia and for the SARS-CoV-2 infection it was a very prevalent problem for both those managing the disease at home and those who were hospitalized. During the fall of 2020 it became more and more evident that many people/patients recovering from COVID-19 continued to suffer from long-COVID MSK pain. Many groups continued to monitor the prevalence of long-COVID pain, to categorize the pain characteristics, and follow the course of the MSK pain problems over the coming years. From a pain research point-of-view the pandemic with 768.237.788 (WHO Coronavirus Dashboard, July 2023) infected worldwide, there is a unique opportunity to 1) explore why some develop long-COVID MSK pain whereas others experience no long-term pain sequela and 2) identify important predictors for the development of MSK. Our assumption is that understanding predictors for developing long-COVID MSK pain can be applicable for the understanding and possible prevention of MSK pain across other conditions.

Findings and Recommendations: Many studies have been published on the prevalence of long-COVID MSK pain after recovery from the virus either at home or at hospital. So far, the studies have been conducted and prevalence determined at different time points over a 3-year period. As expected from other studies on long-term sequelae after a viral infection

the residual symptoms are generally declining over time but often with a small group who will continue having some remaining problems. At this stage this seems also to be the case for long-COVID MSK pain. The findings across studies indicate that myalgia/musculoskeletal is widely present during the infection for both those recovering from hospital (ranging from approx. 35-65%) (1). The presence of myalgia at hospital admission was associated with pre-existing history of musculoskeletal pain (OR 1.62) and myalgia at the acute phase was associated with long-COVID MSK pain (2).

The prevalence of long-COVID MSK pain varies from 0.3% to 65.2% depending on the timing after the infection, the health care disciplines involved and if assessments are related to localized or generalized, widespread MSK pain (3). Female gender and higher BMI are potential risk factors (3), the virus variant where most experience widespread pain after the historical Wuhan virus (4), days at hospital (5). "De novo" (new-onset) pain has been found 75 % among those with post-COVID MSK pain and 25% experienced an increase in previous symptoms (exacerbated COVID-related pain) (5).

The ability to phenotype long-COVID pain into nociceptive, neuropathic, nociplastic or mixed type, is suggested as the first step to better planning of tailored treatment programs (6).

Long-COVID MSK pain is a prevalent problem affecting a residual group of patients after a SARS-CoV-2 viral infection and they need special attention and care. The predictors for developing long-COVID MSK pain can provide now fundamental knowledge generally applicable to the field for MSK pain.

Key words: Covid, Musculoskeletal Pain, Myalgia

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Key words: Long Covid, musculoskeletal pain, SARS, pain

1.6 Myofascial and Articular Treatment of Adolescent Idiopathic Scoliosis

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Aims: This is a report on a study of subjects with Adolescent Idiopathic Scoliosis (AIS) to assess the influence of myofascial dysfunction, fascial constriction, and related joint dysfunction on development and progression of AIS.

Background and Methods: Subjects were randomly assigned to a control group and an active treatment group. The control group received standard care. The active treatment group received a myofascial and articular manual treatment regimen twice per month for 6 months, along with an individualized home exercise program. Arch supports were provided for use throughout the study. Heel lifts and/or ischial lifts were dispensed as indicated for temporary use until the pelvis balanced standing and seated. Results of this care for the active treatment group were monitored during each treatment session. Selection criteria for the study included: age 10-15 years of age, curvature of 15 to 30 degrees, no other significant disorder contributing to spinal curvature, and subjects who had not completed bone growth. Subjects who had been prescribed braces continued with the bracing throughout the study. The intervention regimen was standardized by being performed by a single provider with the requisite training and experience to employ this novel treatment approach. The care was provided primarily at the Clinical and Translational Science Center at the University of New Mexico, although some treatment sessions were performed in a private practice office in Northern New Mexico.

Measurements were performed at the beginning of the study participation and 6 months later at Carrie Tingley Children's Hospital Orthopaedic Clinic, University of New Mexico to assess and compare the results of care for each subject in the control group and active treatment group. Measurements included x-rays with measurement of Cobb angles, scoliometer readings to measure the degree of rib or paraspinal prominence, and subjects filled out a visual analog pain questionnaire and a quality-of-life questionnaire: SRS-22, at the beginning and end of the 6 months of participation. The aim of the study was to determine whether this novel treatment approach might hold promise, when added to standard care, to reduce or reverse progression of curvature, to improve Quality of Life, and to decrease pain.

Findings and Recommendations: Due to the recruitment difficulties associated with the COVID pandemic, the size of the control and active treatment groups (9 and 10 subjects respectively completed study participation) was limited and this in turn limited the number of statistically significant results, with a few significant findings in: the rating of pain, and in results of the SRS-22 questionnaire. Otherwise, trends in a positive direction were observed in SRS-22 subscales, and some aspects of the pain questionnaire. Also, there were highly unusual decreases of spinal curvatures (Cobb angle measurements) in 40% of subjects who completed active treatment: 13 degrees of decrease in 2 subjects, 10 degrees of decrease in 1 subject, and 9 degrees of decrease in another subject.

If structural changes in the spine accounted for the development of scoliotic curvatures, it should not have been possible to produce these decreases of scoliotic curvatures, because the active treatment intervention was designed to alter connective tissue and muscular influences on the spine but not the shape and form of the spinal elements themselves. Furthermore, the structure of the study and interventions allowed for systematic clinical observations that shed light on the etiology of AIS and these observations may bear on how best to successfully implement myofascial and articular care in the future to reduce or reverse progression of AIS, so that further research can incorporate the useful aspects of this care, when appropriate, and further explore this approach for care of AIS. When we consider the current state of research into AIS, the current status of fascia science, and the findings of this research study, it may be most productive to consider AIS as a complex connective tissue disorder in which fascial dysfunction plays an important role.

Keywords: Adolescent Idiopathic Scoliosis, myofascial dysfunction, fascial dysfunction, muscle imbalance, ligamentous laxity, imbalance of pelvis, over-pronation

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1.7 Co-morbidities in fibromyalgia: Can we join the dots? Examining relationships between functional gastrointestinal disorders and comorbidities in women with fibromyalgia.

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Aims: Fibromyalgia is a chronic, idiopathic pain condition. It is associated with increased rates of several co-morbidities, with studies to date primarily focused on coexistent affective disorders, concomitant musculoskeletal diagnoses, headache disorders, and irritable bowel syndrome. While results invariably find increased prevalence of the examined disorders, scant attention has been paid to the constellation of problems commonly affecting people with fibromyalgia. The aim of this study was to compare functional gastrointestinal disorders and co-morbidities between women with fibromyalgia and healthy controls.

Background and Methods: Non-pregnant, non-breastfeeding adult women meeting fibromyalgia criteria, without diabetes or coexistent inflammatory conditions were recruited for a microbiome-focused study (the FIDGIT Study - an investigation into symptoms of Fibromyalgia, Digestive function, and the microbiome of the Gastrointestinal Tract) over 12 months to December 2022 in Auckland, New Zealand. Data collected included validated surveys for the disorders of gut brain interaction (DGBI), headache, depression, anxiety, plus co-morbid presentations (tabulated according to ICD-10), oral health and medication use. These were compared to age and sex- matched controls without fibromyalgia.

Results and Recommendations: In 113 women with fibromyalgia and 55 healthy controls a high prevalence of irritable bowel syndrome (IBS) was noted, along with other DGBI occurring 5-fold higher in cases, compared to controls. Overall, in addition to fibromyalgia, cases had almost ten times as many concomitant disorders compared to healthy controls ($P < 0.001$), with medication use approximately 8-fold higher. A strong relationship was noted between the burden of DGBI and scores related to fibromyalgia, oral health, sleep, mental health, and headache.

Conclusion: Digestive dysfunction in women with fibromyalgia extends beyond IBS, with intimations that the degree of digestive dysfunction is predictive of severity of fibromyalgia, related symptoms, and concomitant co-morbidities. Our study aims to explore this further in the context of the gut microbiome and its function.

Keywords: Fibromyalgia, co-morbidities, irritable bowel syndrome

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Section 2.0 Understanding the Etiology and Mechanisms of Myopain Conditions

Introduction: The etiology and mechanisms associated with myopain conditions including myofascial pain and fibromyalgia (FM) are important to understand to improve management and long-term outcomes. Although acute muscle injuries can be caused by trauma or repetitive strain, there are many etiologies that shed light on broad-based mechanisms of myopain conditions. Localized progressive increases in oxidative metabolism and metabolic distress at motor endplates, particularly in type I muscle fibers are associated with maintain high static muscle tone from sustained poor posture. Tenderness at local trigger points and pain in the muscle, as mediated by type III and IV muscle nociceptors, can be activated by locally released noxious substances such as potassium, histamine, kinins, or prostaglandins. If multiple peripheral and central risk factors further strain the muscles, this increases peripheral and central sensitization due to neuroplasticity can result in more complex chronic myopain conditions such as FM. *Risk factors* such as behavioral factors (eg, repetitive strain, muscle tension, postural habits), emotional risk factors (eg, anxiety, depression), co-morbid conditions (eg, headache, back pain, neck pain, jaw pain, FM), and cognitive factors (eg, poor understanding of etiology, unrealistic expectations, somatization, coping strategies, catastrophizing) have been found to be important in the progression from acute to chronic pain. Diverse factors can also alleviate the pain such as stretching, relaxation, good postures, temperature change, and massage.

2.1 A New Unified Theory of Trigger Point Formation: Failure of Pre- and Post-Synaptic Feedback Control Mechanisms.

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Aims: The origin of the myofascial trigger point (TrP), an anomalous locus in muscle, has never been well-described. A new trigger point hypothesis (the new hypothesis) presented here addresses this lack detailed mechanisms.

Background and Methods: The new hypothesis was developed based on the concept that existing myoprotective feedback mechanisms that respond to muscle overactivity, low levels of adenosine triphosphate, (ATP) or a low pH, fail to protect muscle in certain circumstances, such as with intense muscle activity. This results in an abnormal accumulation of intracellular Ca^{2+} , persistent actin-myosin cross bridging, and activation of the nociceptive system, resulting in the formation of a trigger point. The relevant protective feedback mechanisms include pre- and post-synaptic sympathetic nervous system modulation, modulators of acetylcholine release at the neuromuscular junction, and mutations/variants or post-translational functional alterations in either of two ion channel-opathies, the ryanodine receptor and the potassium-ATP ion channel, both of which exist in multiple mutation states that up- or down-regulate ion channel function.

Results: These concepts are central to the origin of at least some TrPs. The failure of protective feedback mechanisms and/or of certain ion channelopathies are new concepts in relation to the mechanism of myofascial trigger points.

Keywords: myofascial trigger point, mechanism, myoprotective feedback

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2.2. Emerging Insights into the Pathophysiologic Mechanisms of Myofascial Trigger Points and Myofascial Pain

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Aims: To elucidate the ongoing debate surrounding the pathogenesis of Myofascial Trigger Points (MTrP) and Myofascial Pain Syndrome (MPS), and introduce the Neurogenic Hypothesis as a plausible explanation for the unique clinical manifestations associated with MPS. Overview of the current consensus of the pathogenesis of MPS and Myofascial Trigger Points (MTrP) is presented highlighting key clinical observations that are unresolvable using the current local injury model. Exploration of the Neurogenic Hypothesis of Myofascial Pain and the potential causal role of neurogenic inflammation in the pathophysiology of MTrP and MPS.

Background and Methods: MPS is a highly prevalent condition of muscle pain characterized by the presence of palpable hyper-irritable contraction knots within the muscles known as myofascial trigger points (MTrP). Despite the prevalence of MPS, there is still significant controversy within the scientific community surrounding its precise definition and pathogenesis. In particular, debate still exists about whether MPS should be viewed as a distinct disease entity or simply a clinical syndrome. Examination of the connection between degenerative spine disease and MPS using experimental animal models is also important. Furthermore, it is still unclear whether the MTrP is the primary pathology driving the pathophysiology of MPS or represents a secondary physical sign associated with the clinical manifestation of MPS. Clinicians and researchers largely agree on the idea that acute or chronic muscle overload injury to the myotendinous unit is the initial cause of trigger points.

Nonetheless, this hypothesis is unable to explain a number of clinical observations associated with MTrPs and MPS. Accumulating clinical evidence suggests that MTrP may manifest without a clear history of muscle injury. A growing body of research, for instance, demonstrates the comorbidity of MPS with a variety of non-musculoskeletal conditions, including chronic pelvic pain, prostatitis, cystitis, irritable bowel syndrome, and even psychological stress. MTrP and MPS are also frequently observed with chronic musculoskeletal disorders such as osteoarthritis, migraines, and fibromyalgia in the absence of frank injury, suggesting a multifactorial etiology for MTrP and MPS. Yet another significant challenge to the local injury theory is that interrogating the MTrP with manual pressure or needle puncture fails to produce the classic nociceptive withdrawal reflex that is typically elicited from injured tissues. In contrast to the sharp, well demarcated noxious pain typically associated with tissue injury, patients describe trigger point pain as non-noxious and refer to it as "good pain."

Findings and Recommendations: The Neurogenic Hypothesis of Myofascial Pain is an emerging theory proposing that neurogenic inflammation may play an important causal role in the pathogenesis of MTrP and MPS. According to this theory, a persistent primary pathology within visceral or somatic tissues evokes central sensitization, leading to neurogenic inflammatory responses within affected muscles residing within the common neuromeric field of the primary pathology [1]. This hypothesis provides biological plausibility for the clinical observations that are yet unresolved using the local injury hypothesis. Additional emerging research indicates that degenerative spine disease (osteoarthritis) may play a key role in the pathogenesis of MPS. Animal models of experimentally induced spinal facet OA show elevated levels of pro-inflammatory neuropeptides (SubP, CGRP)[2] and increased collagen expression[3] within neurosegmentally linked myotomes. These collective results suggest that targeting spinal OA could be a valuable therapeutic strategy in the ongoing management and prevention of MPS. Significant uncertainty still exists whether MPS is a distinct disease entity or a clinical syndrome. Clinical observations highlight that MTrP and MPS can manifest

without obvious muscle injury. One of these is its documented comorbidity with several non- musculoskeletal conditions. In contrast to pain elicited by local tissue injury, MTrP evoke non-noxious pain, that is often referred to by patients as “good pain”. Furthermore, manual pressure to a MTrP locus does not evoke a pain withdrawal reflex commonly observed with tissue damage.

The Neurogenic Hypothesis [1] suggests that neurogenic inflammation, evoked by a primary pathology within the common neuromeric field but distinct from the affected muscle(s), may lead to MPS. This hypothesis offers biological plausibility for clinical observations unresolvable by the local injury hypothesis. Research on animal models has shown potential links between spinal osteoarthritis and the pathogenesis of MPS [2,3], emphasizing the role of pro-inflammatory neuropeptides and increased collagen expression.

The pathogenesis and characterization of MPS remain areas of active debate. The Neurogenic Hypothesis presents a unique perspective, offering a biological basis for many clinical observations that previously lacked explanation. Furthermore, the potential connection between degenerative spine disease and MPS could further elucidate the pathogenesis of MTrP and MPS, and pave the way for innovative therapeutic interventions.

Keywords: myofascial trigger point, Neurogenic Hypothesis, pro-inflammatory peptides, neurogenic inflammation

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2.3 Innervation of Superficial and Deep Fascia.

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Aims: The aim of this presentation is to review the features and innervation of superficial and deep fascia.

Background and Methods: For many years, the fasciae have been considered only as a “white envelope for the muscles” with very little attention given to their macroscopic and histological anatomy. Recent research has made it clear that we can recognize different fasciae elements including superficial and deep fascia each one with specific features. The superficial fascia is rich in elastic fibers, adaptable and strongly connected with the skin. It envelops and protects the superficial vessels and nerves and probably plays a key role in lymphatic drainage. The deep fasciae are distinguished in two big groups: the aponeurotic fasciae, that work as a bridge connecting different muscles, and the epimysial fasciae, specific for each muscle. The deep fascia are formed by collagen fibers organized in layers, and each layer is separated by the closer one by loose connective tissue, rich in hyaluronan. The collagen fibers define the mechanical behavior of fasciae, the hyaluronan defines the tissue hydration and the ability of glide.

It is important to recognize that each of these elements could be altered by trauma, bad posture, immobilization. Altered (restricted, densified) fascia is responsible for chronic stiffness, decreased strength and abnormal movement patterns (loss of motor direction of bodily segments). The fasciae are also very well innervated, both with sensitive and autonomic innervation, so much that it can be considered a sensory organ. We can distinguished three different types of innervation inside the fasciae:

1. Free nerve ending forming a network. This network is totally embedded in the fasciae, and able to perceive every change in the fascial tension. These receptors have a key role in proprioception and in the perception of the motor directions, but also they could be able to perceive pain.
2. Autonomic fibers. These fibers are around 35% in the superficial fascia, a little less in the deep fasciae. They are present above all around the main vessels, and consequently they are responsible of the regulation of the blood flow inside the fasciae, but also in the middle of the connective tissue, and consequently they could be involved in the fibrotic process of fascial tissue
3. Muscle spindles. These spindles are embedded in the perimysium, and their capsule is nothing more than a doubling of the perimysium around the infrafusal fibers. Muscle spindles inform the Central Nervous System (CNS) of the continually changing status of muscle tone, movement, loss of normal elasticity, position of body parts, absolute length of muscle and rate of change (velocity) of the length of the muscle. In order for a muscle spindle to function it must be able to lengthen, shorten and glide to allow its annulospiral and flower spray organs to be stretched to report accurate information to the CNS.

We also demonstrated as aging increase the thickness of the MS capsule, decreasing the sensibility of the musculoskeletal system to the muscular tonus variation, and consequently this can explain why aged people have a worst proprioception. Besides, the same alterations were found in the multifidus muscle of sheep with back pain.

Findings and Recommendations. Fascia could be considered a key element in peripheral motor coordination and proprioception. This knowledge contributes to our understanding of the biomechanical behaviour of the fasciae, their role in acute and chronic myofascial pain.

Keywords: Fascia, peripheral motor coordination, proprioception, Muscle spindles, utonomic fibers, Free nerve Endings

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2.4 Dry Needling for Spasticity and Scars

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Aims: The objectives of this presentation were to review central nervous system changes after peripheral dry needling to reduce spasticity, and to explore the mechanisms of fascial / scar tissue dry needling.

Background and Methods: Dry needling to reduce spasticity is a promising new approach especially within the practice of physiotherapy. Spasticity, a common finding in patients with multiple sclerosis (65%), spinal cord injury (65%), and stroke (40-60%), features “disordered sensory-motor control, resulting from an upper motor neuron lesion and presenting as intermittent or sustained involuntary activation of muscles” (1-3). Muscle contractures secondary to spasticity commonly demonstrate an increase in the number of cross-bridges, increased muscle stiffness and collagen tissue, and a decrease in muscle fiber length (4). It is known that dry needling damages the neuromuscular junction, which may at least partially explain the positive effects of dry needling on spasticity (5).

In a recent case report, we reported the use of functional magnetic resonance imaging (fMRI) to assess the effects of DN on brain activity of a stroke patient with spasticity. Hemodynamic changes were assessed using Blood Oxygen Level Dependent (BOLD) imaging (5). At the peripheral (local) level, dry needling of the adductor pollicis muscle improved the wrist flexor spasticity and hand function. In the brain, the hand activation area in the motor cortex of the brain improved following dry needling with an increased intensity of BOLD activity in the affected primary motor cortex, the

primary somatosensory cortex, the supplementary motor cortex area, and the unaffected primary motor cortex (6). In a follow-up study (unpublished), a similar protocol was implemented with ten male subjects with spasticity following a stroke. A fMRI was done while performing a motor block design twice before, and immediately after dry needling and hemodynamic changes were assessed using Blood Oxygen Level Dependent (BOLD) imaging to recognize statistically significant voxels with activation during a block paradigm. Imaging was performed at the National Brain Mapping Laboratory (NBML) in Tehran, Iran. The fMRI images were acquired as T2-weighted using a 3.0T Siemens Magnetom MRI system (Siemens, MAGNETOM Prisma). A structural image was acquired using a T1 weighted, MPRAGE sequence with $1 \times 1 \times 1$ mm³ isotropic voxels (repetition time = 1800.0 ms; echo time = 3.53 ms; flip angle 7°, field of view, 244 × 244; matrix = 244 × 244). All fMRI scans were performed using a 45 slice (3 mm) axial plane, gradient echo planar image acquisition, with 2×2 mm² in plane resolution (repetition time = 3000 ms; echo time = 30.0 ms; flip angle = 90° field of view, 192 × 192 mm; matrix = 64 × 64).

Results and Recommendations: The result of the case series were similar to the single patient case report. An increase in activity size was observed in 100% of the subjects in the postcentral region of the affected hemisphere with a 67.73% increase in the maximum amount of activity intensity in the post-central region of the affected hemisphere.

Needling approaches, including dry needling, fu's subcutaneous needling, and acupuncture, have been described previously to improve painful and range of motion limiting scars (5-11). Abnormal scars can be a cause of myofascial pain (12). Empirically, the scar needling approach we developed is remarkably effective and often no more than just a few treatments are indicated. During the lecture, I shared several videos of patients and fascia/scar needling on a cadaver. In a recent case report of the physiotherapeutic assessment and management of a 33-year-old man with erectile dysfunction, Peyronie's disease, and impaired penile sensations following reconstructive surgeries of the penis (13). Significant changes were observed after only five treatment sessions that included dry needling (DN) and needling (14) of the scar and stretching exercises of the penis (13).

In another case report of a 30-year-old female, who underwent an elective total hip replacement following a motor vehicle accident, the patient presented with complaints of persistent burning pain and limited hip range of motion being worse on the right. (1). The patient was treated with six sessions of dry needling over a three-week period combined with infrared radiation for 20 minutes (1). Scar tissue dry needling had a positive effect on the characteristics and appearance of the scar, the patient's pain level, and the hip's range of motion following hip replacement surgery. Functionally, the patient increased her ambulation distance without claudication and improved her single-leg balance based on the Timed Up-and-Go test result (1). The exact mechanisms of scar tissue needling are not known. A study examining the effects of scar tissue needling on tissue compliance, fascial mobility, and pain in progress (15)

Keywords: myofascial trigger point, Dry Needling, Spasticity, Fascial scar needling

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2.4 Historical Review of Myofascial Trigger Point Biopsies and Report of our Findings

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Aim: A historical review of biopsies of muscle pain was presented.

Background and Methods: The review included biopsy studies from human muscles with conditions not diagnosed as myofascial trigger points, such as myogelosis (1), non-articular rheumatism (2), interstitial myofibrositis (3) and fibromyalgia (4). In addition, a review was presented from muscles of human subjects suffering from myalgia (5, 6) or diagnosed as Myofascial Trigger Points (7, 8), including the description of the methodology employed by our team to obtain biopsies from MTrPs of living human subjects (unpublished data). We also review biopsy results from animal studies, such as dog (9), rat (10), mouse (11), and from myofascial trigger point models in rodents (12, 13). Finally, we also reviewed biopsy results from biopsies of human cadavers (unpublished data).
and Recommendations: This data was synthesized and included within the presentation.

Keywords: myofascial trigger point, myogelosis, biopsy studies

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Section 3: Comprehensive Management and Prevention of Myopain Conditions

Myopain pain conditions including myofascial pain and fibromyalgia are among the most prevalent chronic pain conditions, the top reason for health care visits, and driver of health care costs. The personal cost of chronic pain can be devastating resulting in loss of function, disability, depression, addiction and suicide. Everyone, at some point in their lives, has experienced acute muscle pain associated with overuse or repetitive strain. However, when acute pain becomes chronic, patients and their healthcare providers can become confused and overlook myofascial pain and the underlying risk factors that drive myofascial pain. More than half of the persons seeking care for pain conditions still have pain 5 years later, and up to 20% develop long-term disability. Yet few studies identify risk factors and protective factors and how they either lead to or can prevent chronic pain.

The Institute of Medicine (IOM) (2011) and the United States National Pain Strategy stated that studies on early management and preventing chronic pain was among the highest research priority.

3.1 Transformative Care for Myopain conditions: Integrating Self-care Training with Treatment.

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Aim: The aim of transformative care model is to integrate self-care training with treatment as part of routine treatment of myopain conditions to improve long-term outcomes (1-9). This model shifts the paradigms of care from passive doctor-centered model to active patient-centered model that educates, engages, and empowers patients to reduce risk factors that lead to delayed recovery and chronic pain and enhance protective factors that prevent pain (Table 3). Transformative care includes 1) treatment with trigger point counter-stimulation and 2) Self-care training in exercises and reducing postural and repetitive muscle strain.

Background and Methods: President, Myopain Seminars

Diplomate, Academy of Integrative Pain Management Treatment of MFP includes repeated counter stimulation of the muscle trigger/ tender points to desensitize the tenderness and improve the pain. There are many methods suggested for providing repetitive stimulation to tender muscles. Massage, acupressure, and ultrasound provide noninvasive mechanical disruption to inactivate the TrPs. Moist heat applications, ice pack, spray and stretch, and diathermy provide skin and muscle temperature change as a form of counter-stimulation. Transcutaneous electrical nerve stimulation, electroacupuncture, and direct current stimulation provide electric currents to stimulate the muscles and TrPs. Acupuncture, Trigger point injections of local anesthetic and dry needling provide direct mechanical or chemical alteration of TrPs.

Training in self-care is essential to implement patient-centered strategies for Myopain conditions includes exercise, reducing postural repetitive muscle strain.

- *Stretching, strengthening and aerobic exercises.* The most useful muscle rehabilitation exercise techniques include muscle stretching, strengthening exercises, and cardiovascular fitness. In patients with both MFP and other muscle disorders, a home program of active and passive muscle stretching exercises will reduce the muscle tenderness while postural exercises will reduce its susceptibility to flare-ups by physical strain. Strengthening and cardiovascular fitness exercises will improve circulation, strength, and durability of the muscles (1).
- *Reduce Postural and Repetitive Muscles Strain.* One of the common causes of failure in managing MFP and FM is failure to identify and reduce risk factors that may perpetuate muscle strain, restriction and tension. In general, a muscle is more predisposed to developing problems if it is held in sustained contraction in the normal position and, especially, if it is in an abnormally shortened position through poor postural habits. Postural habits causing sustained muscle tension can occur with habits such as: a receptionist cradling a phone between the head and shoulder for hours each day, a laborer lifting with lumbar strain, a student studying with the head forward for hours at a time, or oral parafunctional habits such as bruxism, clenching, gum-chewing, or tongue thrust. Static postural problems include unilateral short leg, small hemipelvis, occlusal discrepancies, and scoliosis. In a study of postural problems in 164 head and neck MFP patients, poor sitting/standing posture was identified in 96% of the patients, forward head in 84.7%, rounded shoulders in 82.3%, lower tongue position in 67.7%, abnormal lordosis in 46.3%, scoliosis in 15.9% and leg length discrepancy in 14.0% (8). In improving posture, specific skeletal conditions such as structural

asymmetry or weakness of certain muscles need to be considered. Examples of strategies to *reduce postural problems* including a foot lift for a unilateral leg length discrepancy, a pelvic lift for a small hemipelvis, and proper height of arm rests in chairs for short upper arms. Postural exercises can also be implemented and designed to teach the patient mental reminders to hold the body in a balanced relaxed position and use the body with positions that afford the best mechanical advantage. Correcting poor habits through education and long-term reinforcement is essential to preventing a reduced TrP from returning. Biofeedback, mindfulness, meditation, hypnosis, stress management counseling, psychotherapy, anti-anxiety medications, antidepressants, and even placebos have been reported to be effective in treating MFP and other muscle disorders.

Findings and Recommendations: Transformative care model is designed to integrate self-care training with treatment as part of routine treatment of myofascial pain conditions to improve long-term outcomes. This patient-centered model educates, engages, and empowers patients to reduce risk factors that lead to delayed recovery and chronic pain and enhance protective factors that prevent pain. Additional opportunities for further learning about transformative care approach is also offered in a massive open on-line course (MOOC) at www.coursera.org.

Table 3. Shifting the doctor/patient paradigms involves each member of the team following the same concepts by conveying the same messages implicit in their dialogue with the patient.

<u>Paradigm</u>	<u>Statement to patient</u>
Self Responsibility	You have the most influence on improving the problem
Self-care	You will need to make daily changes in order to improve your condition
Education	We can teach you how to make the changes
Coaching	We will coach you in making the changes
Long-term change	It will take at least 6 months for the changes to have an effect
Strong provider-patient relationship	We will support you as you make the changes
Patient motivation	Do you want to make the changes

Keywords: myofascial pain, treatment, transformative care, training, self-care

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3.2 Patient compliance with the use of MedicApp® in subjects with myofascial Temporomandibular Disorders.

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Aim: Temporomandibular disorders (TMD) are a group of highly prevalent musculoskeletal conditions affecting the temporomandibular joint, masticatory muscles, and all associated structures (1,2). They may present with orofacial pain and/or sounds during function indicating muscle impairments, disc derangements or degenerative disorders. Conservative treatment is currently recommended for the management of TMD (3) and manual therapy has emerged as one of the most effective therapeutic options (4-7). Telephone applications (Apps) are increasingly being used to manage various activities of daily living, including telemedicine (8). MedicApp® (LIO Srl, Brescia, Italy) is an interactive smartphone and computer application that provides videos on tele-rehabilitation, interfacing the therapist with the patient in a smart form, and allows the clinician to monitor the patient's compliance. At the same time, the patients can check whether they are performing the assigned exercises correctly by following the video displayed on the bottom half of the smartphone screen (self-monitoring), while the app records through the smartphone's front-facing camera.

The aim of this pilot study was to assess patient adherence (compliance) and satisfaction after 2 months treatment with MedicApp®.

Background and Methods: The study was conducted on adult patients with a diagnosis of myalgia or myofascial pain according to the Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) (9). Patients had to be self-sufficient, able to communicate and understand the instructions and capable to use the smartphone. Subjects receiving drugs altering pain perception were excluded. Before and after the treatment, the degree of spontaneous maximum mouth opening (in millimeters) and of pain intensity on a 1-10 millimeters VAS scale were recorded to evaluate improvement. The assigned treatment protocol included masseter and temporalis muscle massage, passive stretching exercises, and proprioceptive training (4-7). Patients were asked to complete a dedicated questionnaire at the end of the study to assess adherence (compliance) and satisfaction.

Results: The sample included 9 patients with myalgia and 6 with myofascial pain. All patients stated to have performed the exercises according to the protocol, so the compliance was very high. Half of the patients (53,3%) reported that they did not use the videos all the time, but to have used them 90% of the time and the 83% of the patients very often controlled if they were doing the exercises correctly. The VAS scores at the end of the 2 months treatment were significantly decreased ($P < 0.001$) and the maximum mouth opening was significantly increased from $43,07 \pm 7,96$ mm to $46,53 \pm 6,80$ mm ($p = 0.001$).

The MedicApp® proved to be a useful treatment tool that helped patients to feel more confident in performing the exercises. In addition, the clinicians' monitoring helped to motivate the subjects. In conclusion, MedicApp® showed good results in terms of adherence to the prescribed exercises, patient satisfaction, and pain control, encouraging further research into its effectiveness for the management of masticatory muscle pain.

Keywords: Patient compliance, MedicApp®, myalgia, Temporomandibular Disorders

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3.3 New Insights into the Electromyography of Chronic Muscle Spasm and Response to EMG Chemo-Denervation Treatment

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Aim: Chronic muscle spasm is characterized by the presence of spontaneous electrical activity (SEA). It has also been shown that chronic muscle spasm leads to the development of hibernating skeletal muscle with loss of mitochondria and muscle fibers. The Coletti Method for EMG guided chemodenervation (CMECD) includes a combination of phenoxybenzamine, lidocaine and dexamethasone to improve this SEA and resolution of the chronic pain caused by the chronic muscle spasm (1-4). The aim of this presentation is to present the the success rates of needle CMECD in patients with pain duration of greater than 1 year.

Background and Methods: Prior publications have shown that prolonged suppression of the SEA with the use of the CMECD procedure will resolve the chronic muscle pain. A demonstration of the CMECD procedure and detailed information on the necessary equipment, supplies and access to phenoxybenzamine medication were presented. A new electromyographic finding in chronic muscle spasm was the presence of motor unit potentials (MUPs) that had not been previously demonstrated. These motor unit potentials are responsive to adrenaline which is secreted in the presence of ischemia. This ties into the proposed ischemic model of chronic muscle spasm that has been previously put forth in a same named article published last year in the European Journal of Translational Myology. The action of the CMECD injection on the electrical activity of muscles in chronic spasm was discussed and demonstrated. Suppression of typical SEA was first noted on treatment with later suppression of the MUPs just prior to full electrical suppression. Theoretical explanations for this two-fold stage of suppression were proposed. An unexpected treatment effect was discussed wherein voluntary activity following initial treatment did not produce MUPs that were readily produced with voluntary activity prior to treatment. The potential additive role of MUPs in sustaining chronic muscle activity was discussed as it related to the prolonged effect of the CMECD procedure. Encouragement was provided to the participants to undertake the CMECD procedure. Preliminary assessment of the success of such treatments for longstanding chronic muscle spasm can be provided by patient surveys of such treatment. Ninety-three sequential patients were treated with this technique and surveyed by mail. Forty-two patients responded.

Results and Recommendations: Of the respondents, 31 (74%) reported multiple years of pain duration. Of those, 50% reported complete relief of pain with CMECD (81% of which reported relief of pain for greater than 3 months) and 27.4% reported moderate relief of pain (44% of which reported pain relief for greater than 3 months). The average duration of pain when specified was 5 years and the longest was 15 years. A single treated patient, not in this survey, reported near complete pain relief and return of function after 35 years.

Truly longstanding chronic muscle spasm and pain can be successfully treated in a significant portion of patients with stable outcomes utilizing the technique of needle CMECD. In this unselected patient population with longstanding chronic pain, results support further clinical research to establish the utility of this treatment modality.

Keywords: Electromyography, Muscle Spasm, EMG Chemo-Denervation

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3.4 Fascial Plane Blocks Targeting Fascia in Myofascial Pain Patients

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Aims: The importance of fascia in myofascial pain syndrome seems to be clear although what part fascia plays in patients suffering from this syndrome is still under debate. All innervation of the muscles courses through the fascia, and performing a fascial plane block (usually under ultrasound guidance) will abolish the pain, at least temporarily. But what part, if any, does the fascia contribute to the pain experienced?

Background and Methods: In the last few years, we undertook two separate studies to try and cast some light on these questions. The first study, published in 2022, examined the differential Sensitization of Muscle versus Fascia in Individuals with Low Back Pain (1). We discovered that although fascia is sensitive and elicits a pain response to stimulation, the main pain generator seems to be the muscle itself.

The second study, in prepublication form, shows that targeting of fascia with ultrasound-guided fascia plane blocks confers an improvement in the patients' syndrome, albeit for a limited amount of time.

In this brief summary, we present the methods and data of these studies for further discussion from our distinguished group of clinicians and researchers.

In the study examining the differential innervation of muscle versus fascia in patients with low back pain (1) we utilized ultrasound guided acupuncture needles to examine for pain tenderness at the subcutaneous, deep fascia and intramuscular level of 20 patients suffering from myofascial quadratus lumborum low back pain versus 20 healthy control subjects. We found that the subcutaneous tissue level is non sensitive to pain in both groups whereas the level of fascia and intramuscular levels are pain sensitive in those suffering from low back pain. In comparison to the fascial level, the intramuscular level was much more pain sensitive.

In the second study (unpublished results), we followed the course of 30 patients suffering from myofascial pain after undergoing ultrasound guided fascial plane block. The patients suffered from abdominal wall (22%), chest wall (18.5), shoulder pain (18.5), low back pain (18.5%), and other myofascial pain syndromes (22.5%). The Injectate was lidocaine 0.5% with methylprednisolone 40 mg in 20ml saline. Average pain intensity decreased from 5.81 (± 2.08) to 4.95 (± 2.65) on a Visual Analogue Scale (VAS) for Pain (0-10) at 4-6 weeks post intervention ($P=0.77$). The Brief Pain Inventory (BPI) questionnaire showed a reduction from 40.9 (± 19.2) to 29.2 (± 18.1) at 4-6 weeks ($p<0.16$). There were no side effects and the procedure was safe and tolerable.

Findings and Recommendations: In patients suffering from myofascial pain syndrome, the muscle and its fascia are involved. In this study, the muscles were more tender than the fascia. Interfascial plane blocks reduce perceived pain intensity in the short- and medium-term in patients suffering from myofascial pain.

Key words: Myofascial pain syndrome, Fascial plane block, sensitization

References

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3.5 Conceptual study on the probable nocebo effect of ordering imaging on the development of chronic pain.

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Aims: Everyone has acute pain from time to time (with extremely few pathological exceptions). Even though chronic pain is very common and increasingly frequent, most people do not develop chronic pain. Why, in 30% of the population

the pain lingers from acute to chronic pain is an intriguing question of utmost importance, as the answer may lead to appropriate treatment options. We propose that feelings such as pain can be practiced by repetitive activities.

Methods and Background: Pain is an unpleasant sensory and emotional experience. The most important classification of pain in clinical practice divides pain into “acute” and “chronic” even though duration is not necessarily the most important factor. More important is the mechanism behind the pain. Acute pain is frequently associated with tissue damage, while the mechanism behind chronic pain is less clear, but tissue damage is often not implicated. Most researchers relate chronic pain to a fault in central processing of sensations, and evidence has been suggested that this happens both in the brain and the spinal cord. Most researchers assume that chronic pain starts as acute pain. Some claim that pain, particularly chronic, is related to perceived danger. While learning from practice is a behavior well-known for centuries, neuroscience research has elucidated some of the central and spinal mechanisms under the concept of neuroplasticity.

Results and Recommendations: We propose that inadvertently, some people develop chronic pain by practice, i.e. through repetitive maladaptive behavior, and furthermore, some clinicians even more inadvertently, help their patient practice pain but subjecting them to repeated imaging. The massive propaganda of commercialized / industrialized medicine and modern imaging is partially responsible for this (both on part of the patients and the clinicians).

The lack of training in pain theory and medicine in most medical schools is also responsible for enabling contributing behaviors of clinicians to increase the pain epidemic, rather than decrease it. Now that modern medicine has finished most of its role in developed countries in preventing the major contributors to years of life lost due to premature mortality, lack of appropriate constraints together with industrial pressure and clinician ignorance may be contributing to years of life lived with disability.

Keywords: myofascial pain, nocebo effect, ordering imaging, chronic pain