



What is known about Electrophysiological Muscle Functions of Patients with Fibromyalgia Syndrome?

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Abstract

Fibromyalgia syndrome (FM) is a disorder characterized by widespread pain, in particular, muscular pain. It is known that central pain processing in FM is deregulated. This results in pain augmentation, a phenomenon termed central sensitization. In addition, there is growing evidence of muscle dysfunction in FM, especially in respect to electrophysiological muscle phenomena.

This paper presents what is known from the literature about electrophysiological muscle functions in patients with FM. Two important phenomena have been shown in the last decade in patients with FM: a) an increased muscle fiber conduction velocity (CV) in their non-painful and non-tender point-related muscles and, b) a tendency to develop myofascial tender points (MTPs). MTPs are very painful spots that are irritable on physical examination (much pain, with referred sensations), and electrically hyper-reactive, as they reveal local vigorous activity with needle electromyography. Studies have shown that MTPs, as a source of pain, may play a crucial role in initiation and maintenance of central sensitization.

The Conclusions are, 1) there is already much known about electrophysiological dysfunction in muscles of patients with FM. 2) The main feature is one of overall muscle hyperactivity/hyperexcitability. 3) A question arises as to whether both electrical phenomena, that is the increased CV and the tendency to produce MTPs in FM, have a common underlying factor. 4) If so, which factor is it? Is the factor intrinsic (i.e. the muscle itself), or extrinsic (i.e. related to a disturbance in the central motor regulation, a neurohormonal influence, or chronic low-level mental stress)?

Keywords: Fibromyalgia; Syndrome; Electrophysiological muscle; Electromyography; Muscles; Conduction velocity

Introduction

Fibromyalgia syndrome (FM) is a disorder characterized by spontaneous chronic widespread and particularly muscular-pain, accompanied by other symptoms, such as fatigue and sleep disturbance [1-3]. It is known that the central processing of nociceptive peripheral input in FM is disturbed [4-7]. This results in pain augmentation, a phenomenon termed central sensitization [8]. In addition, there is evidence of disturbance in the muscle functions of patients with FM. Since the muscle fiber is a structural, mechanical and bio-energetic entity driven by a balanced bioelectrical system, the electrophysiological muscle functions are crucial for a sound working muscle.

This article presents, based on the literature, evidence of electrophysiological muscle disturbances in patients with FM.

Electrophysiological Findings in the Muscles of Patients with Fibromyalgia Syndrome

Since no specific structural aberrations have been found in the muscles of patients with FM [9-11], the interest of researchers since the end of the 1980s has been directed towards electrophysiological muscle aspects. The findings of various studies are presented in Table 1.

Electrical muscle phenomena are measured by means of electromyography (EMG). An EMG investigation has two forms: a) an invasive, needle EMG that is used mainly in a clinical setting and, b) noninvasive, surface EMG (sEMG) that is used only in research. Needle EMG measures electrical activity very closely to the structures. It elicits changes at the levels of the motor unit, muscle fiber and motor endplate. sEMG picks up a broader spatial surface. A one-channel sEMG is able to measure quantitatively the amount of activity produced and to compare activities across various muscles. An array-electrode sEMG (being two or more channels) measures the speed of propagated action potential along muscle membranes. A multichannel sEMG works at the motor unit level and is able to track the recruitment and discharge frequencies of individual motor units.

Quantitative sEMG Studies

Various quantitative sEMG studies have shown differences in patterns and intensities of muscle activity in patients with FM when compared with control individuals. These differences apply especially to postural muscles, such as the trapezius. This finding is of interest since the trapezius is the most often affected/painful muscle in FM. A 2001 study by Elert and coworkers that used sEMG found that patients with primary FM produced more activity in their (postural, trapezius and infraspinatus) muscles than healthy controls; whereas their muscle performance was lower [12]. Another study from the same group demonstrated that FM patients had difficulty with relaxing their

muscles between motor tasks [13]. Research by Banskevisius and coworkers demonstrated that pain induced by chronic mental stress was correlated with the muscle activity of the postural muscle-the trapezius, but not non-postural muscles-in this case, the head muscles [14]. The authors concluded that the trapezius muscle EMG response may be part of a general stress response that causes pain independently of motor activity in overall muscles.

Muscle	Method	Findings	Authors
Trapezius, painful	Multichannel surface EMG ¹ , isometric contractions with 0, 1, 2 and 4 kg load	Increased CV ²	Gerdle et al. [15]
Biceps brachii, non-painful	Surface EMG, array electrode, 30 s isometric contractions at 60% MVC ³	Increased CV	Casale et al. [16]
Biceps brachii, non-painful	Surface EMG, array electrode, 4 s static contractions at 0-10% MVC	Increased CV	Klaver-Krol et al. [17]
Biceps brachii, non-painful	Surface EMG, array electrode, prolonged dynamic contractions at 0-20% MVC	Increased CV	Klaver-Krol et al. [18]
Opponens pollicis, non-painful	Surface EMG, ischemic and post-ischemic conditions	“Tetany” features: spontaneous long-lasting repetitive EMG burst	Vitali et al. [25]
Trapezius	Needle EMG inserted into the MTP ⁴	Characteristic MTP activity	Hubbard et al. [30]
Various muscles	Needle EMG into the MTP	Characteristic MTP activity	Ge et al. [28]

¹EMG: Electromyography; ²CV: Muscle Fiber Conduction Velocity; ³MVC: Maximum Voluntary Contraction Force; ⁴MTP: Myofascial Trigger Point

Table 1: Muscle electrophysiological abnormalities in patients with fibromyalgia syndrome.

Measurements of Muscle Fiber Conduction Velocity

A study in 2008 by Gerdle and colleagues measured for the first time (as far we know) a muscle fiber conduction velocity (CV) in the muscles of patients with FM [15]. It was found, by using multichannel sEMG that the CV of the trapezius muscles was in the patients significantly higher than in controls. Since the trapezius is very often a painful muscle, the authors attributed this CV increase to possible alterations in histopathology and microcirculation.

However, later research demonstrated that the CV was also increased in the non-painful biceps brachii muscles of FM patients [16-18]. Thus, the CV increases no longer could be explained just by local phenomena. The results pointed to a more general feature of the musculature. The CV increases occurred under various experimental conditions: i.e. in static and non-static isometric contractions, in dynamic exercise, and at a range of force levels. Thus, the study by Casale and colleagues found increased CV in the biceps brachii of patients with FM during fatiguing repetitive isometric contractions at force of 60% of maximum voluntary contraction (MVC) [16]. Our two 2012 studies demonstrated an increased CV in the biceps brachii muscle of female patients with primary FM, when compared with

carefully selected age-, length-, and body mass-matched controls. In one study, short static contractions were applied, unloaded, and loaded up to 10% of MVC [18]. In another study, the contractions were dynamic, prolonged up to fatigue, and ranged in the force level from unloaded to 20% MVC [17]. These dynamic tests were performed in a so-called ‘position’ set-up. Here, the subjects were asked to move their lower arm in a standardized way between two exactly-defined positions. To maintain a position in static tests, or to move between different positions in dynamic tests require a high level of central control [19,20]. Therefore, the position tests may provoke possible abnormalities in the muscle control of patients with FM. The patients showed a curious CV pattern during the fatiguing experiment. Normally, CV declines during fatigue (and it is regarded as a sign of EMG fatigue). As FM patients commonly complain of fatigue, one might have expected that the CV decline would have occurred earlier or more clearly. Contrary to expectations, the CV decline was significantly smaller than found amongst the controls, while the endurance time did not differ between groups.

In the 2012 study, that used static contractions, besides increased CV, we also found a strong correlation between the CV and the number of tender points (TPs) found at the bodies of the patients. TPs were examined by manual palpation of 18 body sites, as defined in the 1990 American College of Rheumatology criteria for FM [2]. The correlation suggests that CV and widespread muscular tenderness may have a common underlying background in FM. This problem needs further investigation.

The underlying mechanism of the CV increases is unknown. There are two possibilities: the disturbance can lie in the muscle membrane, or in the motor unit recruitment. If the disturbance is localized in the membrane, this indicates that the membrane is hyperactive, its discharge threshold is lower and its action potential is propagated faster (the CV is higher) [21,22]. If the disturbance lies in the recruitment, this means that the larger motoneurons in the spinal cord that govern thick, fast propagating and anaerobic muscle fibers (type II fibers) would be recruited earlier, and so contribute to higher CV [23,24]. Normally, the fast muscle fibers are gradually recruited with increasing force. The question as to whether either the muscle membrane or motor unit recruitment (i.e. the central regulation) is responsible for increased CV in FM remains unsolved.

Muscle Hyper-Excitability, Hyper-Reactivity Measured by Needle EMG

An early, interesting finding obtained by means of needle EMG was that by Vitali and colleagues. They observed signs of “neuromuscular hyperexcitability” in the patients with primary FM [25]. When measured under post-ischemic conditions induced by a tourniquet, the lower arm muscles of the patients with FM showed spontaneous long-lasting EMG activity, whereas those of control subjects with rheumatoid arthritis did not.

Patients with FM develop the so-called Myofascial Trigger Points (MTPs) more often than people within a “normal” population. The MTPs are hyperirritable spots located within a taut band of skeletal muscle which, when compressed, cause referred pain, local tenderness, and autonomic changes [26]. MTPs occur acutely or sub-acutely, spontaneously or with an immediate cause [27]. MTPs can be active or latent. They are found by manual palpation of the muscle. A gentle palpation is performed across the direction of the muscle fibers to identify a region of tenderness and “the taut band” [26]. After a firm

palpation for several seconds, the typical distribution of the referred pain will be elicited. If a patient's spontaneous pain is reproduced by pressure, the MTP is designated as an active MTP [28]. If pain symptoms are not reproduced, the MTP is considered as latent. Clinically, only active MTPs are relevant, as the latent MTPs are present in many muscles of every individual [29]. The MTPs can be investigated by means of a needle EMG, and it is the only electrophysiological method to document the existence of MTPs [28]. In 1993, a study by Hubbard and colleagues demonstrated that, when a needle is inserted directly into MTPs of the trapezius muscle of FM patients and controls, both patients and controls, in their active and latent MTPs produce spontaneous muscle activity [30]. The amplitude of this spontaneous muscle activity was significantly higher in the active than in the latent MTPs. Patients had a greater number of active MTPs, and higher spontaneous activity amplitudes than controls. Also, the amplitude was strong correlated with the muscle tenderness [30]. This MTP muscle activity is different from the motor unit activity or insertion activity, and is much higher than the endplate activity. Hubbard and colleagues proposed that MTPs are sympathetically activated contractions of the intrafusal muscle fibers (the fibers that regulate muscle tension) [30,31]. Alternatively, the MTPs could be segmental myofibril contractions/cramps of the "normal" extrafusal muscle fibers. MTPs can be visualized by the use of ultrasound technology, which displays them as focal hypoechogenic elliptic areas 0.5-1 mm² in size [32]. With the simultaneous use of color Doppler variance imaging and vibration, a technique called vibration sonoelastography, one observes reduced vibration amplitude. This indicates a local stiff nodule, which may correspond with minuscule local contraction.

Muscle Electrophysiological Phenomena and Pain

Several studies in the last decade showed the relevance of muscle pain, and, particularly MTPs, in initiating and maintaining central sensitization [28,33,34]. A property of an active MTP is that it renders a referred pain along with hyperalgesia (lower threshold for pain stimuli) and allodynia (innocuous stimuli, such as touch, are perceived as being unpleasant and/or painful). Both hyperalgesia and allodynia are not confined to the MTP location, but extend to distant locations [11]. This is the core of the central sensitization: noxious impulse input from peripheral structures is increased (or inhibited too little) in the central pain processing circuits and this leads to the pain augmentation.

Conclusion

Much is already known about electrophysiological functions of muscles in patients with fibromyalgia.

The main feature is hyperactivity/hyperexcitability of the muscles. This applies not only to the painful musculature, but also to non-painful muscles in FM.

A question arises as to whether the increased CV found in clinically healthy, non-painful muscles of patients with FM, and the tendency of the patients to produce myofascial trigger points, have a common underlying factor. If so, what is the common factor? Is it an intrinsic muscle (e.g. membrane) property, or is it an extrinsic factor, such as a disturbance in the central motor regulation, neurohormonal influences, or a chronic low-level mental stress?

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