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Myofascial syndrome and pain: A neurophysiological approach

Juhani V. Partanen [a,*], Tuula A. Ojala [b], Jari P.A. Arokoski [c]

[a] *University and University Hospital of Helsinki, Department of Clinical Neurophysiology, Jorvi Hospital, P.O. Box 800, FIN-00029 HUS, Finland*
[b] *University Hospital of Kuopio, Department of Clinical Neurophysiology, FIN-70200 Kuopio, Finland*
[c] *University Hospital of Kuopio, Department of Physical Medicine and Rehabilitation, FIN-70200 Kuopio, Finland*

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Abstract

It has been debated whether muscle spindles have a role in myofascial pain or not. We present a number of arguments for the former hypothesis. It was hypothesized that firing of intrafusal muscle fibres, i.e. fusimotor activity can be observed as "end plate spikes" (EPSs) in electromyography (EMG). The EPSs may be found in local active spots of muscle, often associated with miniature end plate potentials (MEPPs). Insertion of EMG needle electrodes into an active spot is painful, indicating nociception in the muscle spindle. Myofascial syndrome patients have taut bands with active trigger points (TrPs) in painful muscles.

End plate activity (EPSs and MEPPs) is a significantly more common finding in TrPs of myofascial pain than in control points of the muscle, indicating the presence of muscle spindles. However, some control sites may show EPSs of normal muscle spindles. Increased amount of inflammatory metabolites have been observed in active TrPs.

Muscle spindle is a capsulated gel-filled container, where inflammatory and contraction metabolites may be heavily concentrated during sustained fusimotor activation. Thus the intrafusal chemosensitive pain mediating III- and IV-afferents are sensitized and activated. Intrafusal inflammation causes further reflex activation of the fusimotor and skeletofusimotor systems via sensitized III- and IV-afferents. The taut band itself may be a contracture (rigor) of local skeletofusimotor (beta) units caused by sustained reflex drive by the given muscle spindles. In EMG this may be seen as complex repetitive discharges. We conclude that TrPs of myofascial pain are related to painful muscle spindles in taut bands.

Keywords: Myofascial syndrome; Gamma and beta motor units; Muscle spindle; Muscle III- and IV-afferent; Electromyography

1. Introduction

Myofascial syndrome is a muscular pain syndrome [1] with regional symptoms [2]. It is common with a prevalence rate of 30% in the internal medicine practice [3] and cause much disability and inability to work [4]. Typical clinical findings in symptomatic muscles are taut bands with active trigger points (TrPs) of myofascial pain [2]. Latent TrPs are common even in non-symptomatic individuals. They have been detected in the shoulder girdle musculature in nearly half of a group of young, asymptomatic military personnel [5].

The active TrP is defined to cause spontaneous pain at rest, with an increase in pain on contraction or stretching of the muscle involved. The latent TrP is a focal area of tenderness and tightness in a muscle that does not result in spontaneous pain [6]. The main characteristic criteria of the myofascial syndrome are as follows: 1. regional pain complaint, 2. pain or paraesthesia in the typical distribution of the TrP, 3. a taut band in the muscle, 4. exquisite tenderness found in that taut band, 5. a local twitch response within the band of muscle on plucking palpation across the fibres, and 6. a restricted range of motion in the affected muscle [2,7–11]. There are maps describing the typical locations of TrPs and referred pain areas [12]. Mapping of the infraspinatus muscle showed that most, but not all active TrPs are at midfiber region in the painful side [13]. Functional complaints include decreased work tolerance, fatigue, and weakness [14]. Myofascial syndrome may be activated by recent muscle injury or chronic overload (repetitive strain) of muscles [6]. Works which need repetitive movements demanding precision or repetitive light

* Corresponding author.
E-mail address: juhani.v.partanen@hus.fi (J.V. Partanen).

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lifting may cause myofascial pain especially in the shoulder and neck region [4]. It has also been suggested that anxiety, muscle wasting by malignant disease or neurological disorders, muscle ischemia, visceral disease, radiculopathic compression of motor nerves or climatic causes may be possible reasons for myofascial pain [2].

2. Hypotheses on the origin of myofascial pain

Kellgren [15] was the first to study human muscular pain experimentally by injecting hypertonic saline into muscles, and he also turned his attention to patients with muscle pain. The reason for muscular pain syndromes has, however, been a matter of debate for a longer time. Muscular strain is one of the reasons for myofascial syndrome [2]. There are surprisingly sparse histological alterations either in the muscle or in the TrPs, although mitochondrial changes have been described [16]. Thus muscle trauma or lesion does not seem to be the primary cause [1]. There may be an increase of reticular or elastic fibres which are absent in normal muscle [17]. Local muscle pain might be related to local, temporary hypoxia causing a limited energy crisis within the muscle fibres [16,18]. Reduced blood flow was observed in *Trapezius myalgia* and it correlated with the intensity of pain [16]. Muscle biopsies of tender points show that there were abnormalities in high energy phosphate metabolites, but there were no abnormalities in the non-tender muscle [19]. An "integrated hypothesis" was presented; muscular ischaemia, energy crisis, and increased leakage of acetylcholine into neuromuscular junction cause TrPs in taut bands [12].

There is a sustained electrically silent contracture (rigor) of muscle fibres in taut bands [20]. A role of fusimotor reflexes evoked by muscular III- and IV-afferent activation in the evolution of muscle pain and stiffness has also been suggested [21–23]. However, no electrophysiological signs of spasticity in these patients have been confirmed [9,24]. The muscle contraction of a local twitch response following a direct mechanical stimulation of a myofascial TrP appears to occur only within the taut band with latency consistent with a polysynaptic reflex [25]. Recently, using the microdialysis method an increase of a number of inflammatory and algesic metabolites and decrease of pH has been observed in active myofascial TrPs with local twitch response, compared to normal muscle [26,27]. This explains pain and pressure sensitivity by inflammation in TrPs.

3. Nociception of a muscle is mainly in "active spots" with end plate activity

3.1. Hypothesis for the origins of end plate activity

Every electromyographer sometimes observes end plate activity in the muscle studied. These minuscule sites are called "active spots" or "active areas" of the muscle [28]. End plate activity arises in two forms: 1. end plate noise or miniature end plate potentials (MEPPs) [28–31] and 2. end plate spikes (EPSs), which have a characteristic irregular firing pattern [29,32,33]. MEPPs and several sequences of EPSs are usually activated together [34]. The firing pattern [35–39] and wave form [38] of EPSs is distinctly different from other potentials in electromyography (EMG). If a concentric needle electrode is inserted through an active spot, the cannula potentials representing EPSs are seen as sputtering positive potentials in EMG. These potentials are converted to EPSs as the electrode is withdrawn [40].

In needle EMG the insertion of needle through skin and fascia is felt as sharp transient pain, but further advancement is painless [41]. However, there are local spots in the muscle which often are exquisitely painful when inserted with an EMG needle. The hallmark of these spots is end plate activity in EMG [29,32,34,41] (Fig. 1). Occasionally a brief twitch of a strip of a muscle is seen as the recording needle enters an active area [28], i.e. a local myotatic response may be seen in the active spot.

MEPPs are considered to be local postsynaptic non-propagated potentials caused by spontaneous vesicular exocytosis of acetylcholine from the nerve terminal membrane into the neuromuscular synaptic cleft [31]. The initiating factor in EPS generation is believed to be the needle electrode "irritating" mechanically the terminal motor nerve or endplate connected to a muscle fibre. The EPSs are then recorded postsynaptically with the same needle electrode [30,42,43].

However, there is some concern with the prevailing hypothesis of the origin of EPSs. Experienced neurophysiologists have not observed prolonged injury discharges in nerve fibres. If a peripheral nerve is cut across, the muscle is seen to twitch but contraction does not continue. This is evidence that large myelinated motor axons generate nerve impulses on section, but the discharge is brief [44]. Insertion of microelectrode into nerve fascicles may cause spontaneous activity, brief bursts of two to five spikes. Short-term damage to the nerve fibre may result in the generation of spontaneous repetitive discharges. They appear mainly as rhythmic single potentials or rhythmic bursts, "crazy units" [45], i.e. with firing patterns different from that of EPSs [38]. Spontaneously active units could not be influenced by mechanical stimulation within the fascicular innervation territory, nor could units recorded from motor fascicles be influenced by voluntary effort [45], contrary to the behaviour of EPSs [33,34,38]. Ectopic potentials of peripheral branches of motor axons spread to both directions from the site of origin, causing motor unit potentials or fasciculations, not EPSs. There is no experimental data pointing out that mechanical manipulation of a nerve terminal might cause sustained postsynaptic action potentials in the muscle fibre, even if increased concentration of potassium ions accelerates the MEPP frequency [46].

Another theory was presented for EPSs: they are action potentials of intrafusal nuclear chain and nuclear bag muscle fibres [34]. This theory was based on observed EPS reactions