Chapter 1

Muscle Pain and Muscle Spindles

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Abstract

Muscle pain is a common symptom associated with, for example, myofascial syndrome, fibromyalgia and polymyalgia rheumatica. Many diseases of the muscle tissue are, however, completely or nearly painless such as polymyositis and inclusion body myositis. Thus, a mere inflammation cannot be the cause of muscle pain. In needle electromyography (EMG), the insertion of a needle electrode causes pain but further advancement is usually painless. However, there are small spots of muscle tissue where sudden pain is elicited with the needle. In EMG, these 'active spots' are observed to produce spontaneous activity in the form of end plate noise and spikes (EPSs). End plate noise is elicited at the neuromuscular junction of α, β or γ motor neuron. EPSs are action potentials of γ or β motor units. Muscle spindles are the main nociceptors in muscle tissue, both in healthy muscle and in diseases with muscle pain by inflammation of the muscle spindles. Multiple possible mechanisms of muscle pain exist. Polymyalgia rheumatica may have interstitial pain and possibly pain associated with muscle spindle capsules. Delayed onset muscle soreness may reflect both interstitial muscle pain caused by minor injuries and pain generated in mildly inflamed muscle spindles.

Keywords: muscle pain, myalgia, myofascial syndrome, fibromyalgia, polymyalgia rheumatica, muscle spindle, nociception, fibrillation, fusimotor, electromyography, end plate activity, intrafusal, C-fibres, soreness, DOMS, trigger point, taut band, muscle afferents

1. Introduction

The generation of muscle pain is enigmatic. There may exist several mechanisms for pain production. Many diseases of the muscle tissue are completely or nearly painless, even if there are inflammatory histopathological findings. Thus, inflammation *per se* may not be reflected as muscle pain, although generally inflammation is considered to be associated with pain. In needle EMG, pain caused by the EMG needle seems to be localised in small spots. The EMG activity in these 'active sites' consists of spontaneous electric activity (SEA), whereas in painless sites there is no spontaneous EMG activity. Trigger points, which are sensitive to manipulation and may be exquisitely painful, are a typical feature of muscle pain syndromes. Trigger points are situated in palpable taut bands of the muscle. The principal aim of this chapter is to discuss whether these localised pain spots may actually be inflamed muscle spindles with nociception.

2. Muscle pain produced by a needle during needle electromyography

Meadows [1] studied muscle pain during needle electromyography. He stated that there are sensory receptors associated with skeletal muscle that may give rise to the sensation of pain as observed after ischaemic exercise, or injection of 5–6% sodium chloride. Another form of muscle pain is encountered during the insertion of a concentric EMG needle electrode. When an EMG needle electrode is inserted into a muscle, transient pain is usually experienced, but once the needle has come to rest, the subject may be unaware of its presence. Meadows studied needle pain with concentric needle electrodes with external diameter of 0.46 and 0.30 mm, respectively, on his own vastus medialis muscles. 'When the needle is slowly advanced through the skin, pain is experienced on piercing the skin and again on piercing the muscle fascia, the latter case having a duller and less well-localized character. Further advancing of the needle is then usually quite painless. However, on infrequent occasions, a variably painful point may be reached during such a steady advance. If the needle is further advanced the pain usually subsides but in a few instances was found to be so intense, that further insertion was not attempted. Occasionally when the needle was critically positioned the slightest pressure on its butt caused intense pain which ceased as soon as the pressure was discontinued. It was sometimes apparent, that the site of such pain spots coincided with an increased resistance to the advancing needle, similar to that felt on encountering the muscle fascia when first entering the muscle. In the region of end plate zone advancing the needle sometimes caused a stab of pain which was associated with a twitch of a small fascicle or sometimes a greater part of the muscle'. He also studied pain produced by electrical stimulation through a concentric needle electrode, with the tip of the needle, positioned immediately adjacent to an extremely painful spot in the muscle. Single pulse of 0.05 ms and <5 V produced delayed discomfort and 10/s stimulation produced severe pain. No visible contraction could be seen. The same stimulation in other areas of the muscle was quite painless. Thus it was concluded that there are 'pain spots' in muscle tissue. However, the histological nature of the receptors was obscure. One point was of interest: when a pain spot was encountered, it was sometimes found that there was an increased resistance to the advancement of the needle at this point, suggesting that the receptors may be associated with intramuscular fascial planes.

3. Electromyography of pain spots, historical aspects

The first description of spontaneous EMG activity in pain spots was given by Jasper and Ballem [2]. They found local action potentials comparable to those described by Snodgrass and Sperry [3], and observed that these potentials were associated with particularly acute pain [2]. They

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conjectured that the needle tip was penetrating a nerve and called these potentials 'nerve potentials'. Kugelberg and Petersén [4] described similar potentials in clinical EMG as 'protracted irregular activity'. 'Such discharge was mostly irregular, might be ordinary motor unit potential as in fasciculation or little amplitude and duration as in fibrillation'. Jones et al. [5] further studied the origin of 'nerve potentials' with electrically injected iron marks at sites of their appearance and found most of these iron dots close to peripheral intramuscular nerve twigs. Buchthal and Rosenfalck [6] observed that miniature end plate potentials (MEPPs), or end plate noise, were often associated with this activity, which they called 'spontaneous diphasic spikes'. Finally, Brown and Varkey [7] proved that 'nerve potentials' were postsynaptic, recorded from muscle fibres. Thereafter, the term 'nerve potentials' was rejected and at present these potentials are called 'end plate spikes' (EPSs). The general consensus was that EPSs were activated by the EMG needle, which causes action potentials, when it touches an intramuscular nerve twig or nerve terminal. Action potentials are recorded postsynaptically with the EMG needle. It was not considered, that an ectopic nerve potential spreads to both directions from the site of its origin [8] and thus a motor unit potential (MUP) or fasciculation potential should be recorded, not an EPS [9]. In addition, experimental studies do not support the hypothesis that irregular sustained action potentials like EPSs be activated by peripheral nerve injury or irritation [10–12]. To discuss the origin of EPSs, we have to look at the physiological properties of the muscle spindle.

4. Structure, vascular supply and innervation of the muscle spindle

Human muscle spindles are 7–10 mm long fusiform fluid-filled capsulated organs with equatorial (A) and polar (B) regions. The capsule of the muscle spindle is a lamellated structure, which prevents the diffusion of extrafusal substances into the intrafusal periaxial space [13]. The mean thickness of the capsule is 1.8 μm in the B region, 4.2 μm in the juxta B and A and 7.6 μm in the A region [14]. The periaxial space is between the outer and inner capsule of the spindle and it is full of highly viscous gel. There is a transcapsular potential of −15 mV, which is partly due to a relatively high [K^+] in the fluid. This may contribute to the excitability of the intrafusal endings. There are three types of intrafusal muscle fibres such as nuclear bag 1, nuclear bag 2 and nuclear chain fibres. One spindle has usually one bag 1 fibre, one bag 2 fibre and 4–7 nuclear chain fibres [13]. The muscle spindles are mainly distributed at the region of nerve entry into the muscle and around the subdivisions of the intramuscular nerves [13]. The distribution is thus different from that of the end plate zone, which usually is a relatively narrow band around muscle belly [15]. The main spindle artery is separated from those supplying extrafusal muscles, and in intrafusal capillaries, there is a blood nervous system barrier in both endoneurial and periaxial spaces [13]. The extrafusal capillaries are different and have efficient perfusion when compared to the intrafusal ones. Removal of substances which accumulate into the gel-filled periaxial space of the muscle spindle is a slow process. The sensory innervation of a muscle spindle consists of primary and secondary endings [13], and also III- and IV-afferents [16–19]. Also, autonomic innervation has been observed [19, 20].

The motor innervation consists of fusimotor (gamma) and skeletofusimotor (beta) nerve axons, both of which also have dynamic and static components. They adjust the responses of the primary and secondary endings to the length and changes in the length of the muscle [21]. Dynamic gamma neurons innervate the bag 1 fibre by a p2 plate ending. Static gamma neurons innervate the bag 2 fibre and chain fibres by the trail endings. Dynamic skeletofusimotor beta neurons innervate the bag 1 fibre and extrafusal slow oxidative type 1 muscle fibres by p1 plate endings. Static beta neurons innervate the long chain fibres and extrafusal fast oxidative type 2 muscle fibres by p1 plate endings [13]. Each spindle receives about 7 motor axons, mean 3.2 beta and 3.8 gamma axons. The bag 1 fibre is almost always separately innervated by dynamic beta and gamma axons. Static beta branches supply exclusively the long chain poles. The bag 2 and chain fibres may receive a completely or variously segregated input in each pole [13].

5. Origin of end plate spikes

Where is the origin of EPSs if they are not nerve potentials or postsynaptic muscle fibre action potentials, activated by peripheral nerve injury? Partanen and Nousiainen [22] suggested that EPSs are action potentials of intrafusal muscle fibres such as small nuclear bag and nuclear chain muscle fibres inside the muscle spindles. EPSs can also be observed in active sites after manoeuvres for activating the gamma and beta motor activity such as passive stretch of the muscle, voluntary effort and repetitive nerve stimulation [9]. If multichannel EMG recordings are used, there are also different propagation patterns of EPSs such as local junction potentials as those observed in nuclear bag fibres [23], propagation for a very short distance as in nuclear chain fibres and propagation like MUPS but with the EPS firing pattern, as in beta (skeletofusimotor) motor units [9, 24, 25]. EPSs were also conjectured to be confined to the end plate zone of a muscle [26]. In fact EPSs can be found far from the end plate zone [9, 27]. It is a misconception that MEPPs are observed solely at the end plate zone, where the extrafusal neuromuscular junctions are situated [26]. Actually, MEPPs which are found far from the end plate zone, are mostly intrafusal representing synaptic activity of motor p2, p1 and trail endings. These MEPPs are often associated with EPSs, that is, gamma and/or beta motor unit potentials. At the end plate zone, MEPPs representing an alpha motor nerve terminal are not associated with EPSs [27, 28]. However, there are also muscle spindles at the end plate zone and thus, also MEPPs with EPSs may be found there.

Each pole of the muscle spindle receives 4–5 different motor axons and each gamma or beta axon innervates several spindles, but in a selective manner [13]. Thus junction and action potentials arise in several different spindles, when gamma and beta motor units are activated. This can also be seen in multichannel needle EMG recording. Synchronously firing EPSs may be found in remote active sites of a muscle, if these sites are innervated with the same gamma motor unit [27]. If EPSs in different remote active sites of a muscle are not innervated by the same gamma motor units, EPS firing is asynchronous. Intramuscular EPSs are not seen in the surface EMG, but MUPs of surface EMG are seen in the intramuscular sites with EPSs [27]. EPSs cannot be activated voluntarily, but voluntarily stopping of this activity is possible [27, 29]. Active spots with EPSs can also be stimulated with the concentric needle electrode, using electric impulses. With such stimulation, a reflex response resembling a myotatic reflex can be recorded [27]. Stimulation of an active spot with very small electric stimuli yields a response on another active spot, and even late responses resembling F-waves. Thus, muscle spindles are electrically active structures in EMG, working in a network of gamma and beta motor units and having specific reflex responses [27].

6. End plate spikes are different from fibrillation potentials

In clinical EMG, EPSs may be confused with fibrillation potentials, which are spontaneous action potentials of muscle fibres, or pieces of muscle fibres, which have lost contact with their motor axons. The development of fibrillation potentials needs time and there may be both rhythmic and irregular fibrillation sequences [30]. However, fibrillation potentials are distinctly different from EPSs both by the wave form and by the firing properties [9]. There is also a rare type of fibrillation-like activity, 'myokymic' fibrillations, which are elicited by so-called 'giant miniature end plate potentials' [31, 32]. The essential difference between EPSs and fibrillation potentials is the fact that denervation causes prolongation of the refractory period of the muscle fibre and thus the fibrillation potential cannot recur as promptly as action potential in a normal muscle fibre [33]. This causes the relatively long minimum inter-potential interval of both rhythmic and irregular fibrillation potentials [31]. On the contrary, EPSs have numerous short intervals less than 30 ms [9].

7. Trigger points, taut bands and pain spots

Muscle pain with trigger points (TrPs) is observed in myofascial syndrome and fibromyalgia. In fibromyalgia, there are also other pain spots outside the muscle tissue [34]. Myofascial syndrome is common in medical practice, but also latent TrPs are common in young, asymptomatic persons [35]. The main symptoms of myofascial syndrome are the presence of palpable taut bands in muscles, spot tenderness with TrPs, referred pain, pain recognition and twitch response [36]. The prevailing hypothesis for TrPs and taut bands in myofascial syndrome is 'the integrated trigger point hypothesis' [36, 37]. In short, muscle overload may cause local ischaemia and hypoxia with energy crisis. This causes increased acidity and acetyl choline leakage from the nerve terminal. This is seen as increased spontaneous electrical activity (SEA) in EMG and it achieves local sarcomere contraction knots in muscle fibres. These are felt as taut bands in the muscle. Ischaemia, energy crisis and contraction metabolites increase the local concentration of inflammatory and pain metabolites leading to the development of painful trigger points. Shah et al. [38] found significantly increased concentrations of [H^+], bradykinin, calcitonin gene-related peptide, substance P, tumour necrosis factor-α, interleukin-1β, serotonin and norepinephrine in active TrPs only. SEA in TrPs was stated to be different from spontaneous activity of normal neuromuscular junctions: the electrical discharges occur with frequencies that are 10–1000 times that of normal miniature end plate potentials [39].

However, in EMG studies, SEA is found in 5–10% of routine insertions of the needle into normal muscle [5, 40], without any evidence of dysfunctional end plates. The most common finding is EPSs with end plate noise in the background [25, 40]. For an electromyographer, it is very difficult to accept that MEPPs or end plate noise can achieve contraction knot in the postsynaptic area of the muscle fibre. These wave forms in EMG are a very common finding in quite normal muscles, without any taut bands or trigger points. The situation may be different in experimental studies, where the function of acetylcholinesterase was blocked [41]. The findings of microdialysis of trigger points [38] can be explained by intrafusal microdialysis: a twitch elicited by insertion of the capillary needle may show a myotatic reflex by the activation of intrafusal 1a-afferents of the given muscle spindle. Taut bands may be the final result of sustained reflex activation of beta motor units by intrafusal II-, III- and IV-afferents [25, 27, 28]. Trigger points comprise inflamed and painful muscle spindles with overactive nociceptive afferents. There are somatic thin nerve axons inside the muscle spindle and in its capsule [19]. Thus, it is also conceivable that pain spots in routine EMG of healthy muscles [1] are in fact muscle spindles. Extrafusal muscle fibres in rigour in taut bands cannot produce action potentials, but they can show end plate noise at the neuromuscular junction. Thus, the finding of Simons et al. [42] in myofascial pain can be explained: they found end plate noise (EPN) without spikes (EPSs) in TrPs of all 11 muscles studied, but EPN was found only at four sites at the end plate zone outside of TrPs. The spikes were also observed, but they occurred unexpectedly: one at TrP site, 12 at end plate zone outside TrPs and two at taut band sites. The plausible explanation is that spikes (action potentials of gamma or beta motor units) were mostly blocked in motor units in rigour in TrPs and taut bands, but were readily found outside of these sites [27]. Another issue is the occurrence of end plate activity inside and outside TrPs. Some studies reported end plate activity in every TrP and total absence of such activity in the control points [43, 44]. However, it was showed, that the difference between TrPs and control points, as to the number of EPSs, may even be non-significant [45]. The exception is the upper trapezius muscle, where EPSs are significantly more numerous in TrPs than in control points [45]. The latter explanation is consistent with the fact that there are inflamed muscle spindles (with EPSs) in TrPs and normal muscle spindles (with EPSs) at the control points [27].

Ojala et al. [45] also found increased prevalence of complex repetitive discharges (CRDs) in 16% of patients with myofascial syndrome. CRDs may reflect ephaptic impulse transmission from II-afferents to gamma- or beta-motor efferents intrafusally. This may happen if the concentration of contraction metabolites, especially [K^+] is increased in the periaxial space of muscle spindles after sustained fusimotor activation [46].

8. Interstitial muscle pain

Muscle pain is not always associated with trigger points and taut bands. Injection of hypertonic saline into the muscle causes pain [1, 47, 48], which evidently is interstitial activating mainly extrafusal pain C-fibres. C-fibres are known to be present in every tissue of the muscle with the exception of capillaries [18]. However, there is also evidence that hypertonic saline increases the sensitivity of muscle spindles to stretch [49], and thus also muscle spindles may be involved in the production of pain. The effect on pain caused by capsaicin injection does not differ from that of hypertonic saline injection [48]. In polymyalgia rheumatica, there is an abrupt onset of proximal pain and stiffness, especially in the neck and shoulder girdle. There are also signs of soft tissue oedema and inflammation. Tenosynovitis and bursitis are common. Polymyalgia rheumatica is also often associated with giant cell arteritis [50]. Trigger

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points and taut bands are not typical for polymyalgia rheumatica, and muscle pain is evidently interstitial. EMG is usually normal, and this also is my experience as an electromyographist. Yet abnormalities consistent with either mild myopathic or neurogenic process have been reported in single patients [51]. There are numerous, but non-specific ultrastructural changes of muscle fibres in polymyalgia rheumatica. The endothelial cells of the capillaries showed no changes [52]. Any investigations on the histopathology of muscle spindles in polymyalgia rheumatica were not found. A tempting hypothesis is that there are inflammatory changes of the spindle capsule ('capsulitis'). The spindle capsule at about the equatorial region is made up of fibrous tissue lamellae which usually number 5–7, and are rather rich in endothelial-like nuclei. Among the lamellae lie several small blood vessels [53] as well as thin somatic nerve axons [18, 19]. The thick capsule on the equatorial area of the muscle spindle [14] may be felt as an increased resistance of the EMG needle resembling fascial planes [1, 27].

9. Delayed onset muscle soreness after exercise

Eccentric muscle contractions cause lesions of the muscle membrane and also ultrastuctural damage of muscle fibres. These kinds of lesions are not observed after concentric muscle efforts [54]. Up to six hypothesised theories have been proposed for the mechanism of delayed onset muscle soreness (DOMS) after exercise: lactic acid, muscle spasm, connective tissue damage, muscle damage, inflammation and the enzyme efflux theories. DOMS develops usually in 24 h after exercise in untrained persons [55]. It may be associated with fasciculations, visible spontaneous intermittent contractions of a portion of muscle. The origin of spontaneous fasciculation potentials is mainly distal [56].

10. Fasciculations as a sign of muscle injury after exercise

We studied the appearance of muscle fasciculations after exercise with stretch-shortening cycle (SSC), with partly eccentric contractions. Nine healthy men, aged 25–50 years, were recruited for the study. Spontaneous fasciculations of the soleus muscle were recorded immediately before and at 11 min after 100 jumps with the ball of the right foot with extended knee joint. Fasciculation potentials were recorded with two concentric needle electrodes (diameter 0.3 mm), interelectrode distance 10 mm. The recording was performed before exercise, and 1–2, 4–5, 6–7 and 10–11 min after exercise with Dantec Keypoint EMG machine and Sony DAT recorder. The needles were removed temporarily, and were not used during the exercise. There was a significant increase of the number of fasciculations, beginning at 4–5 min after the 100 jumps and increasing thereafter (**Table 1**). Statistical analyses were performed using IBM SPSS Statistics for Windows (Version 24.0, IBM Corp., Armonk, NY). The differences between the number of fasciculations before and after the 100 jumps (i.e. 1–2, 4–5, 6–7 and 10–11 min after the jumps) were normally distributed, as assessed by the Shapiro-Wilk test ($p > 0.05$). Therefore, a paired-samples t-test was used to determine whether there was a statistically significant difference in the mean number of fasciculations before and after the 100 jumps; the test was repeated for the four conditions corresponding to 1–2, 4–5, 6–7 and 10–11 min after the jumps. The level of significance was set at $\alpha = 0.05$.

N = 9	Mean	min	max	SD
Before	3.6	0	15	4.6
1–2 min after jumps	3.0	0	13	4.0
4–5 min after jumps	20.9*	1	55	18.3
6–7 min after jumps	34.1*	3	87	24.7
10–11 min after jumps	38.4*	4	88	24.4

*$p < 0.01$ (compared to the number of fasciculations before jumps, paired-samples t-test).

Table 1. Number of fasciculations before and after 100 jumps with the ball of foot.

There was no statistically significant difference in the mean number of fasciculations between the measurements before the jumps (M = 3.56, SD = 4.58) and 1–2 min after the 100 jumps (M = 3.00, SD = 3.94), $t(8) = -1.17$, $p = 0.28$, $r = 0.38$. However, the number of fasciculations was, on average, significantly greater for 4–5 min after the jumps (M = 20.9, SD = 18.3) in comparison to the number before the jumps, $t(8) = 3.58$, $p = 0.007$, $r = 0.78$. The increase in the number of fasciculations was further enhanced 6–7 min after the jumps (M = 34.1, SD = 24.7), $t(8) = 4.08$, $p = 0.004$, $r = 0.82$, and even more so for 10–11 min after the jumps (M = 38.4, SD = 24.4), $t(8) = 4.69$, $p = 0.002$, $r = 0.86$.

We conjectured that the eccentric phase of SSC contractions with minor injury [57] caused some biochemical substances, such as cytokines, creatine kinase and [K^+], to be released. Increased extracellular concentration of these substances, especially [K^+] [58], may elicit spontaneous ectopic potentials in intramuscular motor nerve twigs or nerve terminals, spreading to the corresponding motor units and recorded as fasciculation potentials in needle EMG (author's presentation in Single Fibre and Quantitative EMG Meeting, Nijmegen, The Netherlands, June 6–10, 2004). In this case, fasciculations reflect slight damage of muscle fibres caused by the exercise.

Both low-volume high-intensity interval exercise and continuous exercise cause DOMS. Pressure-pain threshold, pressure-pain tolerance and perceived pain intensity were changed in 24 h after exercise [59]. Tenderness to palpation is unevenly distributed in muscles with DOMS. There are regions that are tender to pressure and some regions that are not. Trigger points, referred pain or taut bands, are not observed (author's unpublished observations). Thus, DOMS may reflect both interstitial muscle pain and painful muscle spindles. A question remains: why is there a 24 h delay before the appearance of soreness? It may take time until the extracellular concentration of K^+, caused by the leakage through muscle membranes with minor injuries, is sufficient to increase the firing of interstitial C nerve axons. On the other hand, exercise is associated with overload of muscle and increased fusimotor activity, which increases the concentration of contraction metabolites in the periaxial space of muscle spindles. Accumulated contraction metabolites may induce increase of inflammation metabolites, cytocines and finally pain metabolites intrafusally. Intrafusal pain C-fibres are sensitised by increased periaxial concentration of [K^+] [60]. Thus, there may be a slight inflammation of muscle spindles, and consequently increased pressure sensitivity and pain generated by the intrafusal C-fibres. The development of pain in this way apparently needs some time.

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11. Final comments

The aim of this chapter is to emphasise the major role of muscle spindles in muscle pain. Inflammatory muscle diseases with major histopathological changes are usually not associated with muscle pain. On the other hand, another disease with minor histopathological changes, the myofascial syndrome, may have severe muscle pain and local tenderness to pressure in TrPs. This fact can be explained by inflammation and pain elicited in the muscle spindles. Painful spots in needle EMG may simply be muscle spindles with nociception. Polymyalgia rheumatica may be associated with interstitial muscle pain. It remains to be studied whether there is also pain caused by inflammation of the muscle spindle capsules. DOMS may express both interstitial pain and muscle spindle pain with mild intrafusal inflammation.

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