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Spontaneous electrical activities at myofascial trigger points at different stages of recovery from injury in a rat model

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ABSTRACT

Background Spontaneous electrical activity (SEA) is a feature of myofascial trigger points (MTrPs), which can either be latent or active. However, SEA at different stages of recovery from MTrPs remains unclear.

Objective To investigate the temporal changes in the nature of SEA after generation of MTrPs in a rat model.

Methods 32 rats were divided into four groups: 24 rats were assigned to experimental groups (EGs), which underwent the MTrP modelling intervention and 8 were allocated to a control group (CG). All EG rats received a blunt strike to the left vastus medialis combined with eccentric exercise for 8 weeks. After modelling, the EG rats were subdivided into three groups with total recovery times of 4, 8 and 12 weeks (EG-4w, EG-8w and EG-12w, respectively). Taut bands (TBs) with and without the presence of active MTrPs were identified in the left hind limb muscles of all rats, verified by SEA and further examined with electromyography recordings. Myoelectrical signals were also categorised into one of five types.

Results CG rats had fewer TBs than EG rats and EGs showed variable frequencies of SEA. SEA frequencies were higher in EG-4w than in EG-8w and EG-12w groups (240.57±72.9 vs 168.14 ±64.5 and 151.63±65.4, respectively, $p<0.05$) and were significantly greater in all EGs than in the CG (55.75±21.9). Relative to CG rats, amplitudes and durations of electrical potentials in the EG were only increased in the EG-8w and EG-12w groups. Types IV and V myoelectrical signals were never seen in latent MTrPs and type V signals did not occur in EG-4w rats.

Conclusions Increasing recovery periods following a MTrP modelling intervention in rats are characterised by different frequencies and amplitudes of SEA from TBs.

Trial registration number 2014012.

INTRODUCTION

Myofascial trigger points (MTrPs) are the primary cause of local myofascial pain and are defined as hyperirritable points located in the taut bands (TBs) of skeletal muscles.[1] When compressed, MTrPs produce recognisable patterns of pain that is typically referred.[2] MTrPs often develop after muscle tissue injury and are commonly seen in acute and chronic pain and orthopaedic conditions.[3 4] Acute pain is often experienced during exercise or while taking part in sports when acute MTrPs are present.[5] MTrPs have also been implicated in pain related to hip osteoarthritis,[6] cervical disc lesions[7] and temporomandibular dysfunction,[8] as well as dysmenorrhea[9] and pelvic pain.[10] Myofascial pain syndrome is probably underdiagnosed in patients with chronic pain.

Two of the more popular conceptual models of myofascial pain syndrome described in the literature are the MTrP and radiculopathy models.[1 4 11] Active MTrPs can spontaneously trigger pain in the local area, or produce referred pain or paraesthesia at distant sites.[1] Additional symptoms of MTrPs include muscle weakness, limited range of motion and autonomic dysfunction. Latent MTrPs do not trigger local or referred pain without stimulation, but may still alter patterns of muscle activation and limit the range of movement.[1 12] The MTrP is believed to be a disorder of the neuromuscular junction secondary to an excessive release of acetylcholine (ACh) from motor endplates. The release of ACh initiates spontaneous electrical activity (SEA) in muscle fibres, which is characterised by electromyographic

discharges at MTrP sites. These include low-amplitude discharges (10–50 μV) and intermittent high-amplitude discharges (up to 500 μV) in painful MTrPs.[1] Most researchers consider these discharges to be endplate potentials (waveforms that begin at an upward voltage value and then decline); however, others believe that these high-amplitude potentials are discharges from muscle spindles.[13]

Our previous study in an active MTrP model demonstrated an abnormal pattern to the endplate potential—the waveform initially declined and then bounced back, which is the opposite of the pattern seen in normal muscle fibres.[14] However, the nature of SEA was not investigated under resting conditions after modelling. In order to understand the electrophysiological features in muscle fibres with active MTrPs, we aimed to classify and analyse the electrical potentials in SEA under resting conditions and at different stages of recovery after MTrP modelling. We hypothesised that SEA frequencies and signal types would differ at the various stages (acute vs chronic) and between latent and active MTrPs.

METHODS

All experiments were conducted in accordance with the regulations for the administration of affairs concerning experimental animals (approved by the State Council on 31 October 1988 and promulgated by Decree No 2 of the State Science and Technology Commission on 14 November 1988 in China). The individual study protocol was approved by the Shanghai University of Sport Science research ethics committee (permission no 2013012, licence number SCXK 2007-0003).

A total of 32 male adult Sprague–Dawley rats (mean age 7 weeks, weighing 220–260 g) were randomly divided into two groups in a 3:1 ratio. Eight rats were assigned to a control group (CG) and 24 to an experimental group (EG). EG rats subsequently underwent a MTrP modelling intervention consisting of a blunt strike followed by eccentric exercise at weekly intervals for a total of 8 weeks, as previously described.[14 15] All rats were housed in polypropylene cages with a 12 h/12 h light–dark cycle and kept in a temperature-controlled room (20–25°C) at relative humidity 40–70%. Food and water were freely provided.

Modelling intervention

All rats were anaesthetised with an injection of 4 mL/kg 10% chloral hydrate into the abdominal cavity, then fixed on a board with a homemade striking device on the first day of every week.[14] The site of the proximal vastus medialis (VM) of the left hind limb was marked for rats in both groups, but only EG rats were hit at the marked position by a stick dropped from a height of 20 cm with a kinetic energy of 2.352 J once a week to induce muscle contusion. On the second day of each week, all EG rats were made to run on a treadmill (DSPT-202, China) at a −16° downward angle at a speed of 16 m/min for 90 min. Rats were allowed to rest for the remaining days of the week. The interventions were repeated at weekly intervals for a total of 8 weeks. After completing the modelling intervention, the 24 EG rats were randomly divided into three subgroups with allocated recovery times of 4, 8 and 12 weeks, respectively.

Examined with electromyography recording

EG rats were anaesthetised with an injection of 4 mL/kg 10% chloral hydrate into the abdominal cavity and fixed on a board at the end of their allotted recovery period. CG rats were assessed last, after the third EG cohort. The VM and adductor femoris (AF) muscles of the left hind limbs were surgically exposed and cleared of overlying skin and fascia. Palpation of the muscle for the presence of TBs was carried out by two experienced clinicians. TBs were counted and marked in the muscles of the left hind limb. Three fine needle electrodes (Φ0.3 mm) were connected to, and examined with, an electromyography (EMG) device (Z2J-NB-NCC08, NuoCheng, Shanghai) to record myoelectrical signals at confirmed MTrPs. The first (reference) electrode was inserted in the tail of the rat and the second was inserted into the TB under investigation. If a local twitch response was seen then that TB was considered to be a possible MTrP. For confirmation, a third electrode was inserted longitudinally into the TB about 3–5 mm away from the other electrode. If SEA was detected then it was considered to represent a genuine (active) MTrP. If not, the TB was excluded. Subsequently EMGs of the confirmed MTrPs were recorded for 5 min and analysed by group. The EMG of the normal muscle fibres (no TB) at the left hind limb in the CG was recorded as a control.

EMG analysis and statistics

Two research assistants blinded to group allocation separately analysed all the EMG data. Frequencies and amplitudes of SEA were measured after rectification of the EMG in CG rats (normal muscle fibres plus fibres with latent MTrPs) and EG rats (fibres with active MTrPs at three different stages of recovery). Myoelectrical signals from MTrPs were classified into one of five types according to their morphology (figure 1) as follows: biphasic waves that begin with an upward stroke that deflects downwards (type I); triphasic waves that also start upwards (type II); reverse biphasic waves that begin with a downward stroke that reflects upwards (type III); reverse biphasic waves that start downward but are followed by a slow negative backward wave (type IV); doublet fasciculation potentials (type V). The relative proportions of these signal types were compared between the four study groups. Data were analysed by paired t test and one-way analysis of variance followed by post hoc Tukey's test using the Statistical Package for the Social

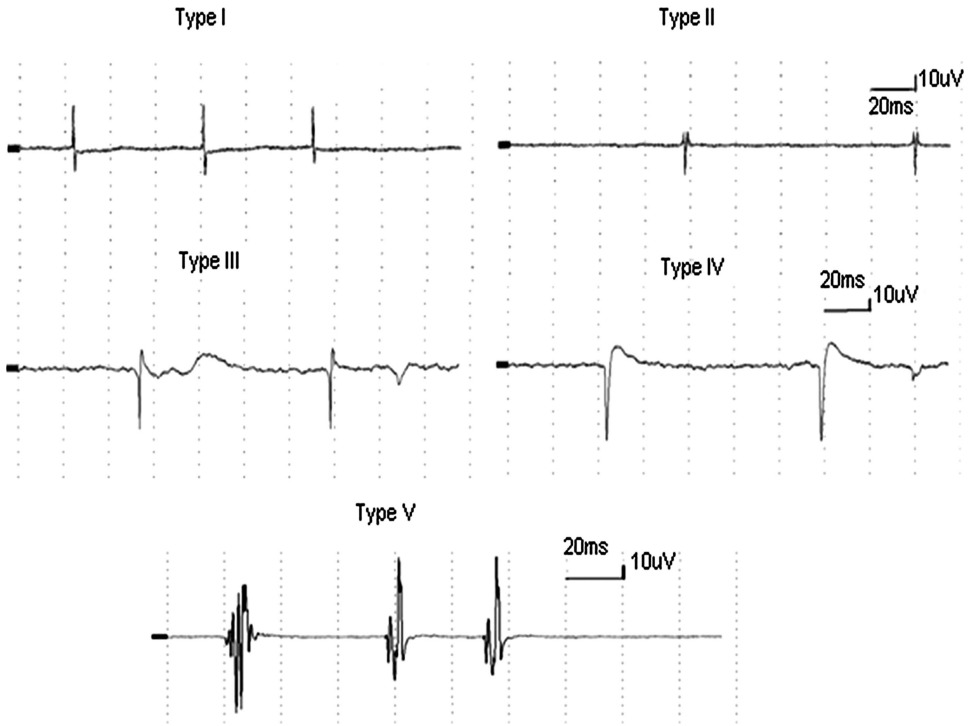

Figure 1 Representative electromyography (EMG) recordings acquired from muscle fibres under resting conditions for approximately 5 min in the control group (CG) and experimental groups (EGs) with 4, 8 and 12-weeks recovery periods (EG-4w, EG-8w and EG-12w groups, respectively): (A) normal muscle fibres of CG showing absence of EMG activity; (B) taut bands (TBs) of CG showing sparse EMG activity; (C) TBs of EG-4w illustrating dense EMG activity; (D) TBs of EG-8w demonstrating intermediate EMG activity; (E) TBs of EG-12w also showing intermediate EMG activity.

Sciences (SPSS) V.17.0 (SPSS Inc, Illinois, USA). p Values <0.05 and <0.01 were considered to represent statistically significant and highly significant differences, respectively.

RESULTS

A greater number of TBs were identified in the VM and AF muscles of the left hind limb in all three EGs than in the CG (table 1). No EMG activity was seen in the normal muscle fibres of CG rats (figure 2A); however, SEA recordings were obtained under resting conditions in TBs of both CG rats (latent MTrPs) and EG rats (active MTrPs) at the three different stages of recovery (figure 2B–E). Overall, SEA frequencies from EG rats were significantly higher than those from CG rats, but were notably lower in those with recovery periods of 8 and 12 weeks compared with 4 weeks. Furthermore, SEA amplitudes were significantly higher for EG rats with recovery periods of 8 and 12 weeks than for those with a recovery period of 4 weeks and for CG rats.

Table 1 Numbers of TBs in the left VM and AF muscles and frequencies and amplitudes of EMG signals by EG

Group	n	TB (n)	Mean±SD	Frequency	Amplitude (μV)
CG	8	8	0.64±0.43	55.75±21.87	26.06±16.67
EG-4w	8	24	3.00±1.07**	240.57±72.88**†	33.08±18.40
EG-8w	8	21	2.63±0.74**	168.14±64.53**	93.26±33.18**
EG-12w	8	18	2.57±0.54**	151.63±65.37**	88.27±31.68*

*p<0.05, **p<0.01 compared with CG.
†p<0.05 compared with both EG-8w and EG-12w groups.
AF, adductor femoris; CG, control group; EG, experimental group(s); EMG, examined with electromyography; TB, taut band; VM, vastus medialis.

Table 2 shows the mean amplitude and duration of the myoelectrical signals from MTrPs in all groups presented by morphological type (I–V). Type I and II signals were significantly shorter in duration and lower in amplitude than those in types III–V. Type V signals demonstrated the highest amplitude and longest duration of all.

Moreover, distributions of the different types of signal varied between latent and active MTrPs and by stage of recovery (figure 3). Types I to III were seen in all four groups and type I accounted for over two-thirds of the EMG signals found in the CG TBs (latent MTrPs), representing a significantly greater proportion ($p<0.01$). By contrast, the proportions of type I and II signals (predominantly type I) appeared lower in EG TBs (active MTrPs), particularly in the groups with longer recovery periods. Conversely, the prevalence of type III and IV signals appeared to increase as the recovery time was extended for EG rats, although only the frequency of type III at 12 weeks was significantly greater ($p<0.05$). Type IV signals were only seen in the EG and type V signals only emerged at 8- and 12-weeks after the modelling intervention. The prevalence of type V signals was

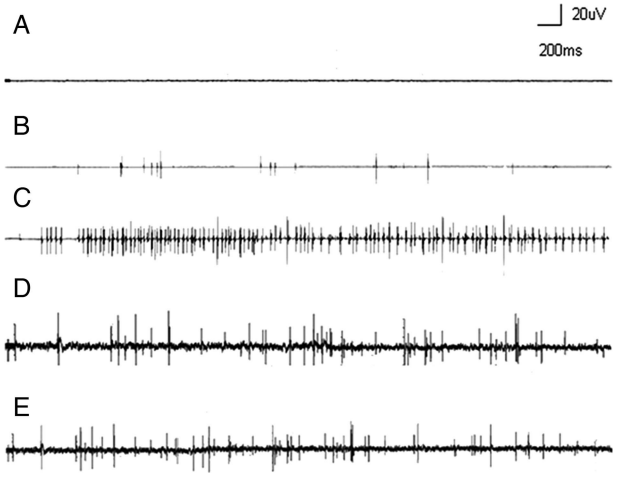

Figure 2 Examples of five different types of myoelectrical signals from muscle fibres of control and/or experimental groups.

greater at 8 weeks than at 12 weeks (15 vs 7%, $p<0.05$). Type IV and V signals were completely absent in the latent TrPs of CG rats.

DISCUSSION

The results show that MTrPs at different stages of recovery have variable SEA frequencies, which are presumably dependent upon MTrP activity. Five types of myoelectrical signals were identified in SEAs of different amplitudes and durations. Type IV signals were present in all EGs; however, type V signals occurred only at the 8- and 12-week stages of recovery.

The VM muscle of the rat is large enough to hit with a striking device.[14 15] The striking position is similar to the site of VM MTrPs in the human body, which refer pain to the front of the patella and anteromedial knee and sometimes the patellar fascia.[1] Single strikes or eccentric exercise alone do not achieve definite and persistent MTrPs in the VM of rats,[14] therefore it was necessary to use a double insult combining strikes to the VM with eccentric exercises to reliably induce trigger points in the muscle. Moreover, the kinetic energy of 2.352 J used in the strike causes skeletal muscle contusion with intact skin[14 15] and a previous study showed that after a repeated loading exercise to the finger, an acute MTrP occurred but vanished within 7 days owing to the lack of continuous intervention.[16] Accordingly a repeated intervention (eight times over 2 months) was used in this study. Typically, a repeated contusion to muscle causes a protective contraction of muscle fibres, resulting in TBs in the VM and AF that are large enough to be palpated by an experienced clinical specialist.[14] Using a repeated blunt mechanical injury in this animal model, a high quality of myoelectrical activity was obtained and all MTrPs were verified by EMG.

Table 2 Amplitude and duration of different types of EMG signals

	Mean±SD	
Type	Amplitude (μV)	Duration (ms)
I	24.04±3.52	4.21±1.21
II	28.84±4.92	5.89±2.13
III	132.84±40.84**	11.40±4.28*
IV	168.30±38.02**	13.84±4.43*
V	216.26±68.47**	23.62±6.21*

*p<0.05 and **p<0.01; compared with both type I and type II groups.
EMG, examined with electromyography.

Although a local twitch response and SEA were obtained from all TBs under different conditions, the frequencies of SEAs varied according to MTrP status. Furthermore, all TBs in EGs had relatively high SEA frequencies compared with those in the CG. We speculate that the lower prevalence and SEA frequencies in CG TBs suggest that these are likely to be latent MTrPs, in contrast to the active MTrPs induced by the modelling intervention in the three EGs. Although it is likely that these are asymptomatic, we cannot be 100% certain given the preclinical nature of this study, and further studies in humans are needed to examine SEA differences between active and latent MTrPs.

TBs in EG rats with 4-week recovery period had the highest SEA frequency of all, which may reflect active injury or inflammation of the muscles.[15 17] In EG rats with recovery periods of 8 and 12 weeks, SEA frequencies were decreased but remained higher than in the CG, which may indicate advanced recovery from acute inflammation. It may be presumed that the different SEA frequencies seen in this study indicate variable MTrP activation within the affected muscles.

Previous studies on needle electromyography of MTrPs have shown that the minute loci at MTrP muscle fibres produce low-amplitude, noise-like electrical activities and intermittent high-amplitude spikes, which are characteristic of SEA.[1 18] This high electrical frequency was primarily seen in EG rats after a 4-week recovery period, suggesting that this type of SEA reflects acute injury and subsequent inflammation with active MTrPs. Tissue inflammation has previously been seen in a MTrP model after a 4-week recovery period.[15 19] In EG rats with 8-week and 12-week recovery periods, SEA frequency decreased relative to a 4-week recovery period, but remained higher than in controls. This may reflect resolution of tissue inflammation and formation of chronic MTrPs.

Analysis of the SEA waveforms yielded different types of myoelectrical signals under different conditions. The source of high-amplitude SEA has been extensively debated. Initially, Hubbard and Berkoff speculated that the high spikes originated from the intrafusal fibres of muscle spindles located near MTrPs.[20] Subsequently, Simons *et al*[1] considered a previous study by Liley[21] and hypothesised that SEA

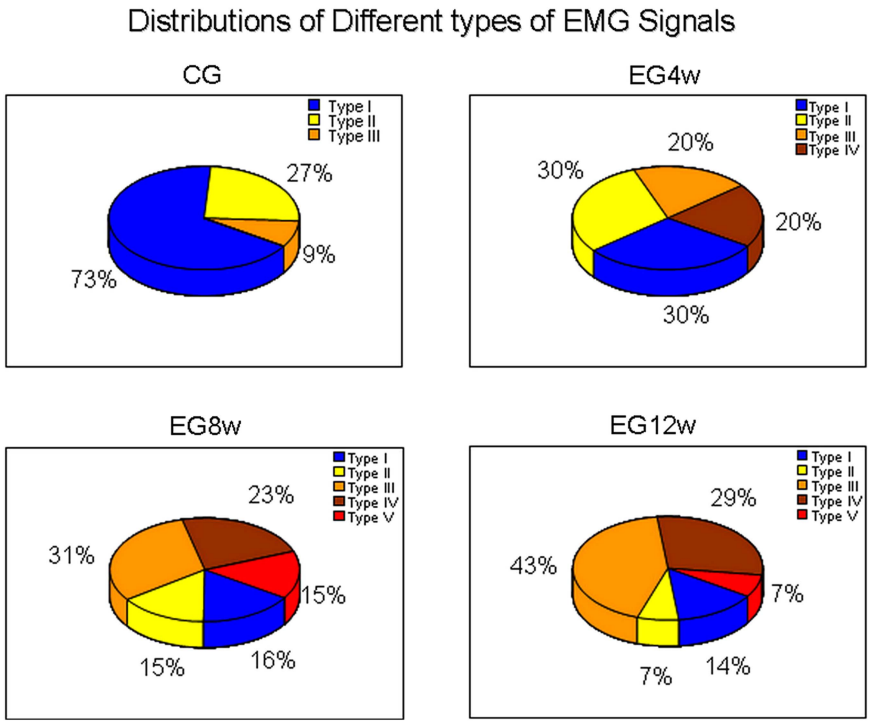

Figure 3 Pie chart depicting average distributions (mean) of five different electromyography (EMG) signal types in muscle fibres of the control group (CG) and experimental groups (EGs) with 4, 8 and 12-weeks recovery periods (EG-4w, EG-8w and EG-12w groups, respectively).

originated from the motor endplates and was defined as endplate noise. In support of this hypothesis, a needle EMG study showed that endplate noise was more prevalent in MTrPs than in adjacent sites.[22] This 'motor endplate' hypothesis was further tested by Kuan *et al*,[23] who injected botulinum toxin into MTrPs to block ACh release into the synaptic cleft and found that it reduced SEA. Lastly, the electrophysiological findings have been correlated with histological changes[24] and local biochemical alterations[25] (eg, inflammatory mediators, neuropeptides, catecholamines and cytokines). However, to our knowledge, no researchers have previously reported the high amplitude of electrical signals or considered that these signals often manifest as an abnormal waveform (such as types III, IV or V).

In our study, types I, II and III myoelectrical signals occurred in all TBs in the CG and EGs and type IV were additionally prevalent in EG rats. In our previous study, the myoelectrical signals of active MTrPs were similar to those of types III and IV in this study,[14] which we consider to be abnormal myoelectrical potentials related to the pathophysiological recovery process of muscle fibres from injury.[14] However, type IV myoelectrical signals might have originated from other sources as proposed by Hubbard and Berkoff.[20] Finally, type V signals only occurred in the EG rats with 8- and 12-week recovery periods and these may relate to intrafusal fibres with sympathetic innervation.

Usually, SEA is a feature of MTrPs or a characteristic of myogenic and neurogenic injury or inflammation;[22 25] however, to our knowledge no other study has reported the different types of myoelectrical signals. Based on cellular physiology, the electrical signals of types I and II are action potentials caused by Na^+ influx and K^+ outflow from cells. However, types III and IV are reverse waveforms and must therefore represent discharges from another source. Type V is a fibrillation potential that is believed to reflect central hypersensitivity or chronic inflammation of the neuromuscular system.[22 25] It remains unclear why types IV and V did not occur in MTrPs of CG and type V was not seen at the 4-week recovery stage. Further research is required to explore the reasons for this in this animal model of MTrPs.

In conclusion, SEA frequency and amplitude vary at different stages of recovery after injury in this rat model of MTrPs and also relative to TBs in control rats. These may represent acute versus chronic and latent versus active MTrP differences, respectively. The high-amplitude spikes of some myoelectrical signals after MTrP modelling may reflect abnormal discharges from sources other than the motor endplate.

Contributors Q-MH thought of the idea and wrote the paper. J-JL and Q-MR conducted the animal experiments and collectected the data. LL analysed the results and revised the paper.

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Competing interests None declared.

Provenance and peer review Not commissioned; externally peer reviewed.

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