Increased H-reflex response induced by intramuscular electrical stimulation of latent myofascial trigger points

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ABSTRACT

Background: Myofascial trigger points (MTrPs) present with mechanical hyperalgesia and allodynia. No electrophysiological evidence exists as to the excitability of muscle spindle afferents at myofascial trigger points (MTrPs). The purpose of this current study was to explore whether an H-reflex response could be elicited from intramuscular electrical stimulation. If so, to assess the possibility of increased reflex response at MTrPs.

Methods: The H-reflex latency and the conduction velocity were first determined from electrical stimulation of the tibial nerve in 13 healthy subjects. Then an intramuscular monopolar needle electrode was inserted randomly into a latent MTrP or a non-MTrP in the gastrocnemius muscle. Electrical stimuli at different intensities were delivered via the intramuscular recording electrode to the MTrP or non-MTrP.

Results: The average conduction velocity (44.3 ± 1.5 m/s) of the electrical stimulation of tibial nerve was similar (p > 0.05) with the conduction velocity (43.9 ± 1.4 m/s) of intramuscular electrical stimulation. The intramuscular H-reflex at MTrPs was higher in amplitude than non-MTrPs (p < 0.001). The reflex threshold was lower for MTrPs than non-MTrPs (p < 0.001).

Conclusion: The current study provides first electrophysiological evidence that intramuscular electrical stimulation can evoke H-reflex, and that higher H-reflex amplitude and lower H-reflex threshold exist at MTrPs than non-MTrPs respectively, suggesting that muscle spindle afferents may be involved in the pathophysiology of MTrPs.

Myofascial trigger points (MTrPs) are a common cause of the enigmatic musculoskeletal pain and dysfunction. Active MTrPs in taut muscle bands are widely recognised, presenting with local and referred pain, and local twitch responses evoked upon snapping palpation and/or dry needling of the MTrPs.1,2 There also exists a spontaneous needle electromyographic activity at the MTrPs.3 While active MTrPs contribute to local myofascial pain syndromes, many latent MTrPs exist in healthy subjects and these latent MTrPs may induce more pain than non-MTrPs upon chemical stimulation and high levels of static postural and visual stress experienced during computer work result in greater pain ratings from MTrPs in the trapezius.4–6 Although the MTrPs are clinically well-described, their pathophysiology remains unclear. Recent evidence shows that local and referred pain intensity and spontaneous needle electromyographic activity at the MTrPs can be facilitated by increased sympathetic outflow to the muscle.7–8 Concentrations of protons, bradykinin, calcitonin gene-related peptide, substance P, tumour necrosis factor-α, interleukin-1β, serotonin and especially norepinephrine are significantly higher at the MTrPs.9 These results suggest that sympathetic hyperactivity is involved in the pathophysiology of MTrPs. An increased sympathetic outflow has been shown to be associated with increased muscle spindle sensitivity in humans.10 We thus proposed that muscle spindle afferents might be involved in the pathophysiology of MTrPs.

Muscle spindle afferents are myelinated muscle afferents (group I and II). The Hoffmann (H-)-reflex is one of the most studied reflexes in humans and is the electrical analogue of the monosynaptic stretch reflex. The H-reflex is evoked by low-intensity electrical stimulation of the afferent nerve, rather than a mechanical stretch of the muscle spindle, that results in monosynaptic excitation of α-motoneurons. The H-reflex recorded from the triceps surae muscle and induced by tibial nerve stimulation in the popliteal fossa is commonly used as a tool in determining the magnitude and distribution of spindle input to a motoneuronal pool, though H-reflex response is also influenced by the excitability of motoneurons.11–15 Therefore, evaluating the H-reflex responses from MTrPs and non-MTrPs would suggest whether or not there exists a higher excitability of H-reflex pathway at MTrPs than non-MTrPs.

Since MTrPs are localised focally within taut muscle fibres, a technique of direct intramuscular electrical stimulation via intramuscular needle electrode, instead of transcutaneous nerve stimulation, is used for the first time in the current study to stimulate the muscle spindle afferents to induce the H-reflex. Thus, the first aim of this study was to explore whether an H-reflex-like response could be evoked by intramuscular electrical stimulation. For this purpose, we compared the conduction velocities of the reflex responses in the gastrocnemius muscle evoked by the intramuscular stimulation and by standard tibial nerve stimulation. The second aim of the study was to compare the reflex threshold and amplitude induced intramuscularly between the MTrPs and non-MTrPs.

MATERIAL AND METHODS

Subjects

Thirteen healthy subjects (seven men and six women, mean age 26.8 ± 1.7 years, mean weight 65.4 ± 2.3 kg and mean height 168.3 ± 6.4 cm) participated in this study. Subjects were enrolled if latent MTrPs were found in the gastrocnemius muscle: taut band,
focal point muscle tenderness and the local twitch response induced manually. The study, approved by the local ethics committee, was performed in accordance with the Helsinki Declaration and the informed consent was obtained from each subject.

Experimental protocol
The subjects were asked to take a prone position on the examination table with a pillow under the chest. The right ankle joint was comfortably supported by a 10 cm high pillow to relax the gastrocnemius muscle. The latent MTrPs were usually found at the medial and lower portion of the gastrocnemius muscle. H-reflex induced by transcutaneous tibial nerve stimulation in the popliteal fossa was first recorded from the gastrocnemius muscle and the latency of the reflex was calculated. Then a monopolar needle electrode was inserted slowly into an MTrP or a non-MTrP in the gastrocnemius muscle, respectively. During the needle insertion, the intramuscular electromyographic (EMG) activity was monitored visually to aid in the searching for the MTrPs. A point in the muscle with spontaneous EMG activity from intramuscular recording but not from surface recording at rest (figure 1), together with muscle tenderness and local twitch response, was defined as a latent MTrP. A point 1 cm lateral or medial to the trigger point with no intramuscular resting EMG activity (figure 1), no tenderness, and no local twitch response was defined as a non-MTrP. Then the same monopolar recording electrode was connected to an electrical stimulator (Counter Point, Dantec, Skovlunde, Denmark). A constant-current square-wave electrical twitch of 1 ms duration was used to evoke the reflex responses. Two pairs of bipolar surface EMG electrodes (Neuroline 720-01-k, Olsøykkje, Denmark) 2 cm rostral to the needle electrodes (corresponding to the MTrP or non-MTrP) were mounted to record the reflex responses from the gastrocnemius muscle. Ground electrode was positioned at the ankle. The reflex recruitment curves induced by tibial nerve stimulation and intramuscular electrical stimulation were constructed and the conduction velocity of the reflex was calculated. The intramuscular reflex threshold, that is, the least electrical intensity (mA) to induce the reflex was recorded.

H-reflex induced by tibial nerve stimulation
The gastrocnemius H-reflex was measured in the right leg using a bipolar surface electrode (Ag-AgCl) with an intra-electrode distance of 2 cm. The recording electrodes were placed over the gastrocnemius muscle fibres at the middle point of the line connecting popliteal fossa crease and medial malleous. A ground electrode was placed around the ankle. The gastrocnemius H-reflexes were elicited with a longitudinally placed ball cathode and anode along the length of the tibial nerve in the popliteal fossa of the right leg. By adjusting the position of the stimulating electrode (cathode being proximal) and the constant current stimulation intensity, the site with first induction of an H-reflex but not M response at low electrical intensities and then the induction of an M response at higher intensities was chosen as the best electrical stimulation spot. Stimulus intensities (1–20 mA) were increased in steps of 1 mA until the maximal H-reflex was identified, and then the stimulus intensity was increased in steps of 2 mA until the maximal M response (Mmax) was achieved, though with a maximum of 24 mA. Five trials were recorded at each stimulus intensity, with each subject receiving approximately 100 stimulations in total. The EMG signal was amplified (Counter Point, Dantec, Skovlunde, Denmark) with gain at 500 μV/division and filtered (bandpass 30 Hz–5 kHz). EMG was sampled (2 kHz sample frequency), averaged and stored for offline analysis. H-reflex recruitment curves were constructed as the H-reflex peak-peak amplitude as a percentage of the Mmax amplitude corresponding to the applied electrical intensity (figure 2). The individual conduction velocity was estimated as: the distance from popliteal fossa to lumbar (L) 4–5 plus the distance from L 4–5 to the surface electrodes divided by reflex latency. M response threshold was also recorded. A visual check for dorsiflexion was done to ensure that the tibialis anterior was not contracting with each stimulus.

Reflex responses induced by intramuscular stimulation at MTrPs and non-MTrPs
A latent MTrP in a taut muscle band with local twitch response upon manual stimulation was first identified and marked. Then a non-tender point (out of the taut muscle band) 1 cm medial or lateral to the MTrP in the gastrocnemius muscle was identified and marked too. A monopolar needle electrode (Ambu neuroline monopolar, 22 mm × 0.36 mm, the exposed tip as the active stimulating area) was slowly inserted into the MTrP or non-MTrP in the gastrocnemius muscle, separately. Needle movement during each advance was approximately 1.5 mm. The reference surface electrode (Ag-AgCl) was placed 2 cm lateral to the needle electrode. The ground electrode was placed around the ankle. The resting electrical activity of an MTrP or a non-MTrP was recorded for 5 s before electrical stimulation. With same recording needle electrode kept in place, the connecting cable was connected onto the electrical stimulator (Counter Point, Dantek, Skovlunde, Denmark) in order to deliver electrical stimulus via the needle electrode. One new pair of bipolar surface EMG electrodes was mounted 2 cm rostral to the needle electrode corresponding to the MTrP or the non-MTrP. The electrical stimulations were the same as that used for tibial nerve stimulation, except the stimulation intensity up to 20 mA. H-reflex recruitment curves were constructed as the H-reflex peak-peak amplitude as a percentage of the Mmax amplitude corresponding to the applied electrical intensity. The reflex threshold, reflex latency, and conduction velocity were recorded and calculated same as that for tibial nerve stimulation. The latency of M response threshold was also visually monitored from the Counter Point EMG screen and recorded. The conduction velocity was calculated as: (the distance from the point of needle insertion to L 4–5) plus (the distance from L 4–5 to the surface electrodes) divided by reflex latency.

Statistical analysis
The Student t test was used to compare differences in nerve conduction velocity between tibial nerve stimulation and intramuscular stimulation. A paired t test was used to compare differences in the threshold of electrical intensity to elicit the reflex responses intramuscularly between MTrPs and non-MTrPs, and also used to compare differences in M response threshold between tibial nerve stimulation and intramuscular stimulation. A two-way repeated measure analysis of variance was used to analyse differences in the reflex amplitude at different points (MTrP and non-MTrP) and different electrical intensities. In all tests, the level of significance was set at p<0.05. Mean ± SE are reported in the text and figures.

RESULTS
Reflex latency induced by tibial nerve stimulation and by intramuscular electrical stimulation at MTrPs and non-MTrPs
The average distance was 0.57 ± 0.02 m from the popliteal fossa to the L 4–5, 0.67 ± 0.02 m from L 4–5 to the surface recording electrode and 0.69 ± 0.02 m from needle stimulating electrodes to...
the L 4–5. The H-reflex recruitment curve and an example from tibial nerve stimulation are shown in figure 2 and figure 3A. The average H-reflex onset latency was 28 ± 1.1 ms for tibial nerve stimulation, and 31 ± 1.5 ms for intramuscular stimulation at both the MTrP and non-MTrP. The average conduction velocity (44.3 ± 1.5 m/s) of the electrical stimulation of tibial nerve was similar (p>0.05) with the conduction velocity (43.9 ± 1.4 m/s) of intramuscular electrical stimulation.

The M response threshold was 1.5 ± 0.4 mA for the intramuscular stimulation at both MTrPs and non-MTrPs and was significantly lower (p<0.001) than that for the tibial nerve stimulation (9 ± 1.2 mA).

Reflex responses induced by intramuscular electrical stimulation at MTrPs and non-MTrPs
The intramuscular stimulation evoked reflex from both the MTrP and non-MTrPs (figure 3B). The reflex threshold for intramuscular stimulation was significantly lower (t = 5.01, p<0.001) at MTrPs (4.0 ± 0.5 mA) than at non-MTrPs (10.5 ± 1.4 mA).

Two-way repeated measure analysis of variance showed a significant difference (p<0.05) in the reflex amplitude between MTrPs and non-MTrPs, a statistically significant difference (p<0.001) in the reflex amplitude among different levels of electrical stimulation intensity, and a significant interaction between stimulation intensities and the type of points, that is, H-reflex amplitude at MTrPs and non-MTrPs depended on the stimulation intensity. Post-hoc analysis showed a significant increase in reflex amplitude for electrical intensities between 9 mA and 20 mA within the MTrP group, but not within the non-MTrPs. A higher level of the reflex amplitude in the MTrP group than the non-MTrP group was also seen over electrical intensity range of 9 mA until 20 mA as shown in the reflex recruitment curve (figure 4).

DISCUSSION
The results in the present study provide the first evidence that H-reflexes can be induced by intramuscular stimulation and that there exists a lower H-reflex threshold and higher H-reflex amplitude at the MTrPs than non-MTrPs. These results suggest that a greater excitability of the muscle spindle afferents is associated with the MTrPs.

Intramuscular electrical stimulation of muscle spindle afferents
Our results show that the conduction velocity of the reflex pathway by intramuscular stimulation is similar to that of tibial nerve stimulation in poplitical fossa. Tibial nerve stimulation induces H-reflexes, which represent the magnitude and distribution of spindle input to a motoneuronal pool.11 12 Since the reflex conduction velocity is the same for tibial nerve and intramuscular stimulation, the reflex response by intramuscular stimulation resembles the H-reflex evoked by tibial nerve stimulation. Thus, intramuscular H-reflex is due to direct stimulation of muscle spindle afferents. Thus, direct intramuscular stimulation with resultant H-reflex provides a method to observe different responses at MTrPs and non-MTrPs to electrical stimulation.
Involvement of H-reflex pathway at myofascial MTrPs

The lower electrical stimulation intensity applied to induce H-reflex and the higher H-reflex amplitude at the MTrPs than non-MTrPs suggests that H-reflex pathway is involved at myofascial MTrPs. The lower reflex threshold and higher reflex amplitude at myofascial MTrPs could be related to a greater density or excitability of muscle spindle afferents. This result is supported by experimental muscle pain studies which show the increased stretch reflex sensitivity during muscle pain in animals and in humans.14 15 Increased spindle input to the motoneuronal pool may provide an explanation to muscle stiffness, poor movement coordination, spontaneous local muscle twitch and the increased resting or dynamic muscle activities in chronic musculoskeletal pain conditions.41 61 7 No significant changes in H-reflex amplitude during muscle pain in some studies may be due to the differences in muscle activity levels when the H-reflex was induced.18 The increased H-reflex response at latent MTrPs following intramuscular electrical stimulation is consistent with the results of a human study indicating that nociceptive stimulation to the muscle induces increased H-reflex when the muscle is relaxed, but inhibits the H-reflex when the muscle is in contraction possibly due to the fact that when the muscle is at rest, nociceptive muscle stimulation is unable to modify the excitability of Renshaw cells involved in recurrent inhibition.19

Methodological considerations of intramuscular induction of H-reflex

Preliminary results in additional two subjects show that intramuscular H-reflex has spatial characteristics in signal propagation within the muscle, that is, more distant to the stimulation point, more decreased is the reflex amplitude. Therefore, the surface recording electrodes placed at a distance of 2 cm away from the intramuscular stimulation site can better capture the reflexes. Since the intramuscular H-reflex is also evoked at the non-MTrPs, the effect of volume conduction at higher stimulation intensities to the MTrPs can not be excluded though the withdrawal of the stimulating needle about 1.5–2 mm at the MTrPs led to the disappearance of the reflex at lower stimulation intensities. But the effect of volume conduction does not negate the differences in H-reflex pathway between the non-MTrPs and the MTrPs.

It is also worth noting that intramuscular H-reflex has several characteristics that are different from H-reflex induced by tibial nerve stimulation. (1) Intramuscular H-reflex has spatial characteristics in signal propagation within the muscle, that is, more distant to the stimulation point, more decreased is the reflex amplitude. Therefore, the surface recording electrodes placed at a distance of 2 cm away from the intramuscular stimulation site can better capture the reflexes. Since the intramuscular H-reflex is also evoked at the non-MTrPs, the effect of volume conduction at higher stimulation intensities to the MTrPs can not be excluded though the withdrawal of the stimulating needle about 1.5–2 mm at the MTrPs led to the disappearance of the reflex at lower stimulation intensities. But the effect of volume conduction does not negate the differences in H-reflex pathway between the non-MTrPs and the MTrPs.

The mechanisms underlying increased sensitivity of muscle spindle afferents at the MTrPs are still unclear. This could be related to increased inflammatory substances in the MTrPs,9 leading to increased static fusimotor drive to muscle spindles or increased muscle spindle sensitivity.20–22 Alternatively, the increased muscle spindle sensitivity at MTrPs may partly be related anatomically to the segregation of muscle spindles in the muscle and related sensory partitioning phenomenon.22

Figure 3  H-reflex induced by tibial nerve stimulation at the intensity of 9 mA to show the reflex latency at 31 ms (A), and H-reflex induced by intramuscular electrical stimulation at the intensity of 7 mA at both the trigger point and non-trigger point in the gastrocnemius muscle, the induced reflex latency is 32 ms (B).

Figure 4  The reflex recruitment curves from intramuscular electrical stimulation at the trigger point and non-trigger point and peak-peak reflex amplitude was normalised as a percent of maximal M wave (%Mmax). *Indicates significantly higher reflex amplitude over the range of electrical stimulation of 9–20 mA as compared with that at lower intensities within the trigger point group. # indicates significantly higher level of reflex amplitude at the trigger point as compared with the non-trigger point.

A

B

Figure 5  H-reflex induced by intramuscular electrical stimulation at both the trigger point and non-trigger point (A). The induced reflex latency is 32 ms (B).

Figure 6  The reflex recruitment curves from intramuscular electrical stimulation at the trigger point and non-trigger point and peak-peak reflex amplitude was normalised as a percent of maximal M wave (%Mmax). *Indicates significantly higher reflex amplitude over the range of electrical stimulation of 9–20 mA as compared with that at lower intensities within the trigger point group. # indicates significantly higher level of reflex amplitude at the trigger point as compared with the non-trigger point.
Summary points

- Intramuscular electrical stimulation can evoke H-reflex mediated by muscle spindle afferents.
- The excitability of muscle spindle afferents is greater at myofascial trigger points than at normal muscle points.
- Therapies directed at decreasing the excitability of muscle spindle afferents at myofascial trigger points may decrease trigger point sensitivity.

In conclusion, the current study provides first electrophysiological evidence that intramuscular electrical stimulation can evoke H-reflex and that higher H-reflex amplitude and lower H-reflex threshold exist at MTrPs than non-MTrPs, suggesting that muscle spindle afferents may be involved in the pathophysiology of MTrPs.

Competing interests: None.

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