American Pain Society
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PUBLISHED BY ELSEVIER

The Journal of Pain, Vol 12, No 12 (December), 2011: pp 1282-1288
Available online at www.sciencedirect.com

Latent Myofascial Trigger Points are Associated With an Increased Antagonistic Muscle Activity During Agonist Muscle Contraction

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Abstract: The aim of this study was to evaluate motor unit activity from a latent myofascial trigger point (MTP) in an antagonist muscle during isometric agonist muscle contraction. Intramuscular activity was recorded with an intramuscular electromyographic (EMG) needle inserted into a latent MTP or a non-MTP in the posterior deltoid muscle at rest and during isometric shoulder flexion performed at 25% of maximum voluntary contraction in 14 healthy subjects. Surface EMGs were recorded from the anterior and posterior deltoid muscles. Maximal pain intensity and referred pain induced by EMG needle insertion were recorded on a visual analogue scale. The results showed that higher local pain was observed following needle insertion into latent MTPs (4.64 ± .48 cm) than non-MTPs (2.35 ± .43 cm, $P < .005$). Referred pain was reported in 6/14 subjects following needle insertion into latent MTPs, but none into the non-MTPs. The intramuscular EMG activity, but not surface EMG activity, in the antagonist muscle was significantly higher at rest and during shoulder flexion at latent MTPs than non-MTPs ($P < .05$). The current study provides the first evidence that increased motor unit excitability is associated with reduced antagonist reciprocal inhibition.

***Perspective:** This study shows that MTPs are associated with reduced efficiency of reciprocal linhibition, which may contribute to the delayed and incomplete muscle relaxation following exercise, disordered fine movement control, and unbalanced muscle activation. Elimination of latent MTPs and/or prevention of latent MTPs from becoming active may improve motor functions.*

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***Key words:** Isometric contraction, intramuscular electromyography, myofascial trigger points, muscle relaxation, reciprocal inhibition.*

Myofascial trigger points (MTPs) are main characteristics of the myofascial pain syndrome,[5,32] the most common muscle pain disorder in clinical practice.[9,14] Active and latent MTPs[8,25,35] present with a tender spot within a muscle taut band, local twitch response upon palpation and/or dry needling, and spontaneous electrical activity with intramuscular needle electromyographic (EMG) examination when the muscle is at rest.[19,20,33,34]

Active MTPs are responsible for pain symptoms in patient populations, while latent MTPs exist without spontaneous pain.[15,31,32,35] Latent MTPs are prevalent in healthy subjects and musculoskeletal pain patients, and may be 1 of the potential sources of sensorimotor dysfunctions in humans. Current evidence shows that latent MTPs contribute to the development of muscle cramps,[11,36] restricted joint range of motion,[16] and muscle weakness and accelerated fatigability.[10] Latent MTPs may cause a reorganized recruitment of muscles that work synergistically to produce an action.[13,22,23] Peripheral factors related to the muscle fibers accommodating the MTP as well as central motor

Received June 27, 2011; Revised August 11, 2011; Accepted September 15, 2011.
This study is supported by Danish Agency for Science, Technology and Innovation: 07-028674-1.
The authors have no conflicts of interest to report.
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1526-5900/$36.00
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doi:10.1016/j.jpain.2011.09.005

control mechanisms[18] have been suggested to be involved in the reorganized muscle activity in musculoskeletal pain conditions. However, whether spinal inhibitory mechanisms are related to the increased motor unit excitability at MTPs is unknown.

Reciprocal inhibition plays an important role in movement control.[22,23] When a muscle receives a nerve impulse to contract, its antagonist simultaneously receives another to relax (Sherrington's reciprocal inhibition). Previous studies have shown the existence of reciprocal inhibition mechanisms in different muscle groups.[2,3,28] It is known that muscle pain may cause increased antagonistic activity[18] during an active movement and hence somehow impair the potency of reciprocal inhibition.[10,13] When the anterior deltoid muscle is isometrically contracted, the posterior deltoid—its antagonist—will be inhibited and will demonstrate reduced muscle activity immediately following agonist contraction.[6]

We hypothesized in the current study that latent MTPs may be associated with a reduced antagonist reciprocal inhibition during agonist muscle contractions. Confirmation of this hypothesis will provide further evidence supporting the role of latent MTPs in driving motor dysfunctions. To test this hypothesis, intramuscular EMG activity from latent MTPs in posterior deltoid muscle was recorded at rest and during isometric anterior deltoid muscle contraction (shoulder flexion) with an aim to evaluate the efficiency of reciprocal inhibition as compared to non-MTPs.

Methods

Subjects

Fourteen healthy subjects (12 males and 2 females: mean age, 26 ± 6.9 years; mean weight, 74.5 ± 9.9 kg; mean height, 178 ± .1 cm), with no signs or symptoms of musculoskeletal pain, volunteered for this study. This study was approved by the local Ethics Committee (N-20100048) and conducted in accordance with the Helsinki Declaration. Informed consent was obtained prior to experiment.

Experimental Protocol

Each subject participated in a 2-trial study in which latent and non-MTP were detected by palpation in the posterior deltoid muscle. This experiment consisted of 2 sessions with an intramuscular EMG needle electrode was inserted into either a latent MTP or a non-MTP in the posterior deltoid muscle on the dominant side. There was a 1-day interval between 2 sessions. The intramuscular EMG needle insertion into the latent MTP or a non-MTP was randomized. Intramuscular EMG activities from posterior deltoid muscle and surface EMG activities from both the anterior and posterior deltoid muscles were recorded at rest and during 25% of maximal voluntary contraction (MVC) force of shoulder flexion. There was a 10-minute interval between trials. Local pain intensity and referred pain from EMG needle insertion were recorded at the end of each session.

The subject was seated in a chair with back support. They were asked to relax their arms in a neutral position; a supporting pillow was placed under the relaxed forearm to form a 45° angle of passive elbow flexion. A force transducer (MC3A; AMTI, Watertown, MA) was closely contacted to the upper surface of the forearm. Shoulder flexion was achieved by the active isometric contraction of the anterior deltoid muscle against the force transducer. The isometric contraction, instead of dynamic contraction, was chosen in the current study due to the potential needle displacement out of the MTP during dynamic contraction of the posterior deltoid muscle.

Palpation

A latent MTP was defined by the presence of a taut muscle band, local twitch response, and most tender spot upon digital palpation. A non-MTP was defined by the absence of latent MTP characteristics.

MVC Recordings

Anterior deltoid muscle contraction force without needle in the muscle was measured using a force transducer mounted in custom-designed setups. Subjects were asked to maximally flex the dominant shoulder for 3 seconds and repeated 3 times with 30 seconds between each repetition. The contraction with maximal force was chosen as the value for the MVC.

EMG Recordings

An intramuscular EMG needle (Ambu Neuroline Concentric, .25 × 45 mm; Ballerup, DK) was inserted into a latent MTP or a non-MTP. A latent MTP was then confirmed by the presence of intramuscular spontaneous electrical activity (SEA) from the intramuscular EMG needle.[33,34,36] To search for the SEA, a concentric EMG needle is used in the current study due to our experience that a concentric EMG needle is more stably localized in the MTP than a monopolar EMG needle during slow ramp muscle contraction. The procedure of searching for the SEA is similar to those reported previously,[33] except that the angle of needle insertion was at an angle of approximately 90° to the skin surface overlying an MTP (targeted to the nodule) for the first track of needle insertion. The second track of needle insertion was at an angle of approximately 80° directed proximally to the first track and the third track of needle insertion was at 80° directed distally to the first track. The angles of needle insertion at 80 to 90° to the skin were adopted in the current study to minimize potential mechanical influence of needle on the electrical activity of the MTP during muscle contraction. Each advance continued until it encountered endplate spikes with the amplitude of at least 50 uV when the muscle is at rest. Conversely, a non-MTP was confirmed by the absence of spontaneous intramuscular electrical activity from the intramuscular EMG needle, which was placed in a non-taut-band muscle outside of the endplate zone (MTP region), 1 to 2 cm away from the site being examined.[33] The recording needle electrode for

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