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肌筋膜激痛點之超顯微構造的研究

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計畫主持人：官大紳
共同主持人：簡基憲，林瑞模

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中文摘要

肌筋膜激痛點是肌筋膜疼痛症候群的特徵，它是存在於骨骼肌內裡緊繃肌帶上之局部性過度激活的痛點。激痛點裡的活動小點所發現到的自發性電位活動(SEAs)，是一種不正常型式的終板電位，它是來自於乙醯膽素的過度釋放。肌筋膜激痛點的病理機轉，可能來自於激化的感覚神經纖維(痛覺感受器)和不正常之終板的刺激，再經由脊髓的整合機制所產生而成。雖然肌筋膜激痛點的電氣生理學的特徵已被釐清，但是肌筋膜激痛點確定的形態學徵象則仍然是不清楚的。而且以前大部分的報告，不是激痛點的診斷不夠明確，就是研究材料多是來自於動物或是人類的屍體身上。

我們提出兩年期的研究計劃，目的在於藉由新進之肌電圖學儀器的幫助來對肌筋膜激痛點做定位，再來找尋、並且界定出肌筋膜激痛點的形態學特徵。在第一年的研究裡，我們要在動物的骨骼肌肉上定位出肌筋膜激痛點來，然後利用光學顯微鏡與電子顯微鏡來找尋出它的形態學特徵。第二年的研究，則是要將我們在第一年所得到的實驗成果，運用到人體的活組織上，以界定出人體肌筋膜激痛點真正的形態學特徵。

關鍵詞：肌筋膜疼痛症候群，肌筋膜激痛點，自發性電位活動，終板雜訊，形態學

ABSTRACT

A myofascial trigger point (MTrP), a characteristic of myofascial pain syndrome, is a localized hyperirritable spot in a palpable taut band of skeletal muscle fibers. Spontaneous electrical activities (SEAs), which can be found in active loci of MTrPs, are abnormal patterns of endplate potentials resulting from excessive leakage of acetylcholine. The pathogenesis of MTrPs is probably related to an integrative mechanism in the spinal cord in response to sensitized sensory nerve fibers (nociceptors) associated with dysfunctional endplates. Although the electrophysiological characteristics of an MTrP have been delineated, the conclusive morphological findings of an MTrP still remain unclear. Most of these previous reports were either lack of definitive MTrP diagnostic criteria, or from animal or cadavers study.
With the aids of new electromyographic (EMG) instrument to localize an MTrP, we designed a 2-year study to define the morphological characteristics of the MTrP. In the first year of the study, we will localize a myofascial trigger spot on skeletal muscle of an animal to look for its morphological characteristics with light microscopy and electron microscopy. In the second year, we will apply the methods used in the first year on the living human tissue to define the really morphological characteristics of human MTrP.

The processes of preparing the animal muscular tissue for histological examination were very complicated. In our preliminary results, we found some “dense-muscle-fibers” figures in our specimen. It might correspond to the “contaction knot” which had been claimed as the characteristic morphology of MTrP by Simons. Through these morphological findings, this study may further enhance our understanding of the pathophysiology of an MTrP.

Key Words: Myofascial pain syndrome (MPS), Myofascial trigger point (MTrP), Spontaneous electrical activity (SEA), Endplate Noise (EPN), Morphology.

Background and Objective

Myofascial trigger point (MTrP) is a highly localized hyper-irritable spot in a palpable taut band of skeletal muscle fibers [Travell and Simons, 1983 & 1992]. Although myofascial pain syndrome (MPS) have been recognized for many decades, its exact diagnostic criteria is still been challenged. Simons concluded that "spot tenderness", “pain recognition," and "taut band" are the most reliable signs and the minimal criteria needed to identify an MTrP, while "referred pain" and "local twitch response" are most useful as confirmatory signs of the MTrP [Simons, 1996]. Hong and Torigoe had developed an animal model and found it was very useful for studying the pathophysiology of MTrP. The most sensitive spot to elicit a local twitch response (LTR) in a taut band in a rabbit was defined as a myofascial trigger spot (MTrS), which is equivalent to the human MTrP [Hong and Torigoe, 1994].

In 1993, Hubbard and Berkoff demonstrated the presence of EMG activities at minute sites ("nidus") in an MTrP region of the upper trapezium muscle [Hubbard and Berkoff, 1993]. They called these EMG activities as "spontaneous EMG activity" Simons and colleagues later defined this continuous low-amplitude activity was as “spontaneous electrical activity (SEA)” to distinguish it from the intermittent spike activity (100 to 600 µV, biphasic, initially negative), which could be recorded only from active MTrPs but not from latent MTrPs [Simons, et al, 1995]. Comparing the SEA patterns to the endplate activities mentioned by electromyographier [Kimura, 1989], there is almost no difference between these two tracings. Based on histological and pharmacological studies, Wiederholt has described an electrical activity similar to SEA in his studies on rabbit skeletal muscle, and he confirmed this activity as “endplate noise (EPN)” [Wiederholt, 1970]. Ito et al demonstrated that this abnormal pattern of
endplate potentials was attributed to excessive release of acetylcholine packets [Ito et al, 1974]. Therefore, SEA is probably one type of endplate potential, and the active loci of an MTrP are closely related to dysfunctional endplates. Since the EPN was exactly similar to SEA recorded from MTrP region, Simons has suggested that the term “EPN” instead of “SEA” should be used in MTrP studies [Simons, 2001].

With the multiple loci theory for the MTrP, Hong later developed the concept of “Basic Unit of an MTrP” [Hong and Simons, 1998]. These SEAs (EPNs) loci are related to dysfunctional endplates (“motor structures”), and they are defined as active loci to distinguish them from the sensitive (LTR) loci, which are "sensory structures". Therefore, a sensitive locus is probably in the immediate vicinity of an active locus, and both structures (sensitive locus and active locus) together may form an MTrP locus, a basic unit of an MTrP [Hong and Simons, 1998]. The MTrP locus, consists of a sensitive locus and an active locus. The sensitive locus, from which an LTR can be elicited, is related to nociceptors. The active locus, where SEA can be recorded, is related to dysfunctional endplates (“motor structures”), and also related to nociceptors. The pathogenesis of MTrPs is probably related to an integrative mechanism in the spinal cord in response to sensitized sensory nerve fibers (nociceptors) associated with dysfunctional endplates.

From the above review, the morphological characteristics for MTrPs are still inconclusive. Most of the reported findings come from animal study, while those come from human studies were either lack of definite diagnostic criteria of MTrPs, or from non-living human body. In this study, we will use current electrophysiological diagnostic criteria to objectively localize the MTrP. Specially-designed recording needle electrode will help us define the active locus of an MTrP more precisely. In the first year of the study, we will use an animal model to delineate the morphological characteristic of an MTrP.

Results and Discussion

With the specially designed EMG needle, we localize the location of MTrS in animal muscles. It was a very complicated process for us to obtain a good animal muscular specimen for our morphological study. We had tried our study protocol on 10 rats but the results showed nothing special. (Fig. 1&2). Later, we did our experiments on the rabbits. In our preliminary results, we found some “dense-muscle-fibers” figures in the MTrS region of rabbit skeletal muscles (Fig. 4~7). It might correspond to the “contaction knot” which had been claimed as the characteristic morphology of MTrP by Simons. However, we need more samples to exclude the possibility of artifact.

In 1976, Simons and Stolov used TrP criteria to examine canine muscles for a tender spot in a palpable taut band comparable to that observed in human patients [Simons and Stolov, 1976]. Some isolated, large, round muscle fibers and some groups of these darkly staining, enlarged, round muscle fibers appeared in cross
sections. In longitudinal sections, the corresponding feature was a number of contraction knots. An individual knot appeared as a segment of muscle fiber with extremely contracted sarcomeres. This contractured segment showed a corresponding increase in diameter of the muscle fiber. This feature may represent one of the first irreversible complications that result from the continued presence of the contraction knot. The muscle fibers containing contraction knots are clearly under increased tension both at the contraction knot and beyond. This sustained tension could produce local mechanical overload of the connective tissue attachment structures in the vicinity where the taut band fibers attach. Taut bands in MTrPs are probably formed as persistent contracture (a state of muscle contractile activity without EMG activity) of muscle fibers due to excessive acetylcholine leakage in the active loci. Simons concluded that the SEAs (EPNs) found in active loci of MTrPs are abnormal patterns of endplate electrical activity resulting from excessive acetylcholine leakage [Simons et al, 1995].

References


