Rehabilitation Practice and Science

Volume 29
Issue 2 *Taiwan Journal of Physical Medicine and Rehabilitation (TJPMR)*

Article 1

6-1-2001

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Recommended Citation

Kuan, Ta-Shen; Chen, Shu-Min; Chen, Jo-Tong; Chien, Chi-Hsien; and Hong, Chang-Zern (2001) "The Effect of Transection of Innervating Nerve on Spontaneous Electrical Activity of Myofascial Trigger Spot in Rabbit Skeletal Muscle," *Rehabilitation Practice and Science*: Vol. 29: Iss. 2, Article 1.
DOI: https://doi.org/10.6315/3005-3846.2125
Available at: https://rps.researchcommons.org/journal/vol29/iss2/1

The Effect of Transection of Innervating Nerve on Spontaneous Electrical Activity of Myofascial Trigger Spot in Rabbit Skeletal Muscle

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Myofascial pain syndrome is characterized by the existence of myofascial trigger point (MTrP). Local twitch response (LTR) and spontaneous electrical activity (SEA) are two important objective characteristics of MTrP. The basic unit of an MTrP is the MTrP locus, which contains a sensory component (sensitive locus or LTR locus) and a motor component (active locus or SEA locus). In an animal study, rabbit LTR was only temporarily suppressed during the spinal shock period after transection of the spinal cord at a level high above the lower motoneurons of that muscle. It appeared that LTR is mainly mediated through the spinal cord and supraspinal structures are not essential. SEA recorded from an MTrP is abnormal endplate potentials due to excessive release of acetylcholine (ACh). However, SEA is not controlled or influenced by the spinal or supraspinal circuits because there was no significant change in the prevalence of SEA after spinal cord transection. Fourteen New Zealand rabbits were studied to assess the effect of innervating nerve transection on the SEA in a myofascial trigger spot (MTrS – equivalent to human MTrP). The prevalence of SEA in rabbit biceps femoris was monitored before sciatic nerve transection, immediately after the transection, post-transection 1st day, 2nd day, 1st week, 2nd week, 3rd week and 1st month. SEA was persistent for only one day after sciatic nerve transection and then disappeared on the 2nd day after nerve transection. It remained disappeared till the end of our investigation (one month). It appears that humoral mechanism (ACh release) play a major role in the occurrence of SEA, while neural mechanism (direct nerve impulses) is not important. This study further support that SEA is a dysfunctional endplate potentials due to excessive release of ACh. (J Rehab Med Assoc ROC 2001; 29(2): 65 – 75)

Key words: myofascial pain syndrome, myofascial trigger point, myofascial trigger spot, spontaneous electrical activity, local twitch response

INTRODUCTION

One of the most common muscle pain problems noted in clinical practice is myofascial pain syndrome (MPS). It is characterized by the existence of painful

Submitted date: 7 October 2000. Revised date: 17 November 2000. Accepted date: 24 November 2000.
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myofascial trigger point (MTrP), which is a highly localized hyper-irritable spot in a palpable taut band of skeletal muscle fibers[1,2] . Two important characteristics of MTrP are referred pain and local twitch response (LTR). A patient with an active myofascial trigger point in a particular muscle frequently describes a typical and characteristic referred pain pattern (in the referred zone) for that specific muscle. When a trigger point is mechanically stimulated by snapping palpation or needling, LTR can be elicited, either by palpation or by vision, as a brisk contraction of a group of muscle fibers (usually, a taut band containing a trigger point)[3]. 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[4].

Although there had been no definitely anatomic evidence of MTrP, recent electromyographic study on both human subjects and animals provided us a new concept on the pathophysiology of the MTrP. A useful animal model for studying pathophysiology of MTrP was developed by Hong and Torigoe[5]. They identified taut bands, which were similar to those in human muscle, by finger palpation in rabbit biceps femoris muscle. When a certain sensitive site in the taut band was stimulated mechanically with a blunt metal probe (snapping or tapping) or by a needle, LTRs were observed. Rabbit LTRs are similar to human LTRs both in the characteristic of visible muscle twitching and in electromyographic (EMG) recording. The most sensitive spot to elicit an LTR in a taut band of rabbit skeletal muscle was defined as a myofascial trigger spot (MTrS), which is equivalent to the human MTrP in many aspects[5]. In human, the minute sensitive site from which an LTR can be elicited by needle stimulation has been defined as a sensitive locus in an MTrP region[6].

Hubbard and Berkoff demonstrated the presence of "spontaneous electromyographic (EMG) activity" at minute sites ("nidus") in an MTrP region of the upper trapezium muscle and no such activity at adjacent non-tender sites[7]. They described the spontaneous activities as two types: a low amplitude constant background activity of about 50μV, and an intermittent higher amplitude activity of 100-700μV. Simons and colleagues later found similar spontaneous and continuous low-amplitude action potentials (10 to 50 μV, occasionally up to 80 μV) from human MTrPs[8,9,10]. They defined this continuous low-amplitude activity as spontaneous electrical activity (SEA) to distinguish it from the intermittent spike activity (100 to 600 μV, biphasic, initially negative) that could be recorded only from active MTrPs but not from latent MTrPs. The minute locus from which SEA can be recorded is now defined as an *active locus* of an MTrP[4]. Hubbard and Berkoff hypothesize that the source of the electrical activity is abnormal muscle spindles[7,11]. However, Simons et al recruited many strong evidences and concluded that the site of this electrical activity was at or very near a motor endplate [3,4,8,12].

Either SEA or LTRs, or both, can be observed at different loci in an MTrP region during the search for SEA, and both SEA and LTR are often associated with a sharp pain sensation that is similar to the patient's usual complaint. Therefore, a sensitive locus (LTR locus) is probably in the immediate vicinity of an active locus (SEA locus), and both structures together may form an *MTrP locus,* a basic unit of an MTrP[13,14]. Hong et al studied the EMG activity of LTRs in rabbit skeletal muscle and found it almost disappeared after lidocaine block or transection of the innervating nerve[5]. They also found that rabbit LTRs were unobtainable immediately after spinal cord transection at a level high above the lower motor neurons of that muscle. However, after the spinal shock period (approximately 2.5 hours), rabbit LTRs were nearly completely recovered [15]. It is obvious that LTRs in rabbit skeletal muscle are mainly mediated through the spinal cord, and supraspinal structures are not essential. Hong and Yu later used this animal model to assess the effects of transection of spinal cord and peripheral nerve on SEA[16]. In contrast to LTRs, there was no significant change in the prevalence and amplitude of SEA up to 60 minutes after spinal cord transection and 30 minutes after subsequent nerve transection. It was concluded that the occurrence of SEA is a local motor endplate phenomenon, and it is not mediated through the spinal or supraspinal circuits[16].

From the above studies, we know that LTR is controlled directly by neural (spinal cord) mechanism, while SEA is not directly (or immediately) influenced by

neural impulse. However, the results only revealed short-term effects of nerve transection (up to 30 minutes) on SEA, which could be considered as "neural" mechanism from nerve impulse. Long-term effects of nerve transection on SEA, viewed as "humoral" mechanism concerning about neural transmitters delivered from the nerve terminals, had not been investigated. This study was designed to see if neural transmitters (humoral mechanism) persistently influences on SEA in an MTrS region of rabbit skeletal muscle after transection of the innervating nerve. This study may help us further understand the neurological mechanism of SEAs, which would contribute to the comprehensive pathophysiology of MTrP.

MATERIALS AND METHODS

General Design

One myofascial trigger spot (MTrS) in rabbit biceps femoris was randomly selected and was searched for SEA electromyographically before transection of the sciatic nerve. After obtaining pre-transection data, the sciatic nerve on the same side of the selected MTrS was transected. SEA was searched in this MTrS after the nerve transection with defined intervals (immediately after transection, post transection first day, post transection second day, post transection first week, post transection second week, post transection third week, post transection first month) to see if and when SEA was diminished or disappeared.

Animal Preparation

Fourteen New Zealand rabbits (weight 3 ~ 5 Kg) were studied. These rabbits were anesthetized initially with an intramuscular injection of Ketamine 0.05 mg/GBW [17]. Subsequent intravenous injections of Pentothal at 0.0lg/ml were given every 20-30 minutes to maintain the anesthetic level. These rabbits were fixed on the examination table and were placed on a heating pad to maintain a constant body temperature. The anesthetic level was controlled so that most of the spinal reflexes were preserved. These animals were also monitored for heart rate and respiration to avoid overdose of anesthetics. The skin of the lateral thigh was incised to expose the biceps femoris muscle and it is separated anteriorly from the quadriceps muscle to expose the sciatic nerve. This also made it possible to slip a finger beneath the muscle for pincer palpation of the muscle fibers when searching for taut bands.

Identification of a Myofascial Trigger Spot

The biceps femoris muscle was grasped between the fingers and was palpated by gently rubbing to find a taut band. The fibers of the taut band were unmistakably firmer in consistency than the surrounding muscle such that the band could be snapped between the fingers and was felt like a clearly delineated "rope" of muscle fibers roughly 2-3 mm or more in diameter. The location along the band where snapping palpation produced the most vigorous localized twitch response was the myofascial trigger spot (MTrS).

Electromyographic Recordings

A 2-channel NICOLET Viking IV EMG unit was used for this study. The low-cut frequency filter was set at 100 Hz and the high-cut at 1,000 Hz. The gain was generally set at 20 μV per division for recordings. At the usual sweep speed of 10 ms per divisions, one screen presented 100 ms of record. Intramuscular electrical activity was recorded using 25-mm, disposable, monopolar Teflon-coated EMG needle electrodes. The needle electrode used to search SEA (active recording electrode) was connected to channel 1 of the preamplifier box of the EMG unit, and the control needle electrode, which was inserted into a normal muscle tissue (non-taut band, non-MTrS), was connected to channel 2. A clip used for the surface electrode was attached onto the nearby skin. It served as the common reference electrode by connecting it to both channels through a "Y" connector (Figure 1). The ground electrode was clipped to another site of nearby skin. Room temperature was maintained at $2l \pm 1\,^{0}C$.

Search for a Locus of Spontaneous Electrical Activity

The active recording electrode was inserted parallel to the direction of the muscle fibers into the region of the MTrS at an angle of approximately 45^{0} ~ 60^{0} to the surface of the muscle. After the initial insertion to a point just short of the depth of the MTrS, the needle was

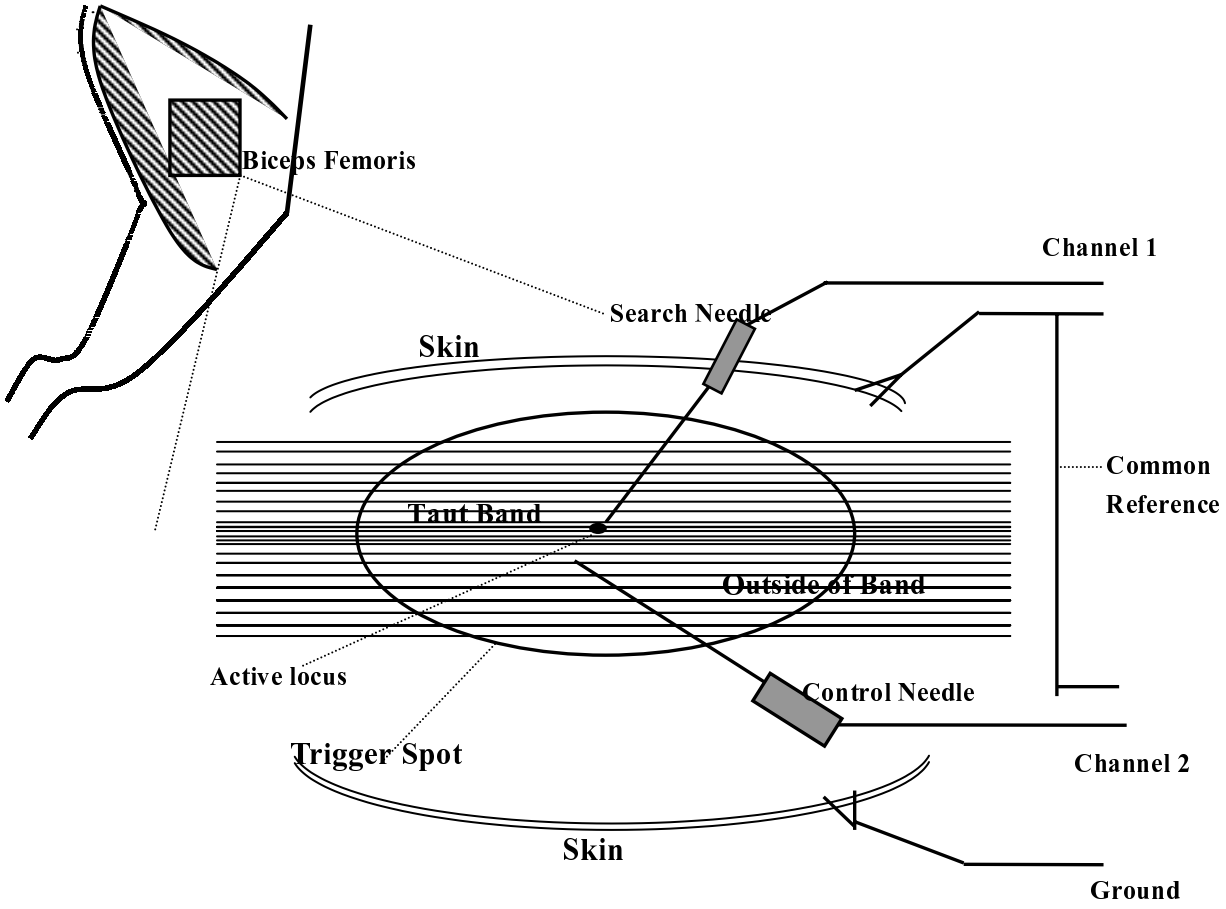

Figure 1. Arrangement of electromyographic electrodes for SEA recording.

advanced very slowly. Each advance was made through the least possible distance (usually 1-2 mm for one advancement) by simultaneously rotating the needle to facilitate smooth entry through the muscle tissue. This would prevent the needle from "grabbing" the tissue and releasing the tissue suddenly to advance in a large jump. After 8 advancements of the needle (one track), the needle was pulled out and was reinserted into next track in the muscle, 1 mm posterior to the previous track. There are 8 tracks investigated in one MTrS region. Large advances should be avoided because of the minute size of an active locus and the likelihood of inducing a rabbit LTR instead of finding a locus of SEA.

When the needle approaches an active locus, the continuous distant electrical activity was heard. A site was an active locus when SEA was identified if: 1) noise-like potentials persisted continuously for more than three screens (300 ms); 2) the potentials had an amplitude of > 10 μV (which was more than twice the instrumentation noise level of 4 μV that was observed in control recordings taken at the beginning and upon completion of each track); and 3) the adjacent control channel was not recording potentials greater than instrumentation noise level. Once the active locus was localized, the needle remained there without further movement, and the SEA was stored for later analysis. Then, the investigator continued searching for another SEA. If no SEA appeared during an advance of the needle, at the end of the advance, the examiner added very gentle side-pressure to the needle sequentially in four directions, forward and back, right and left, with regard to the direction of muscle fibers to see if SEA could be found.

Transection of Sciatic Nerve

Before nerve transection, the sciatic nerve was identified and separated from the surrounding soft tissues.

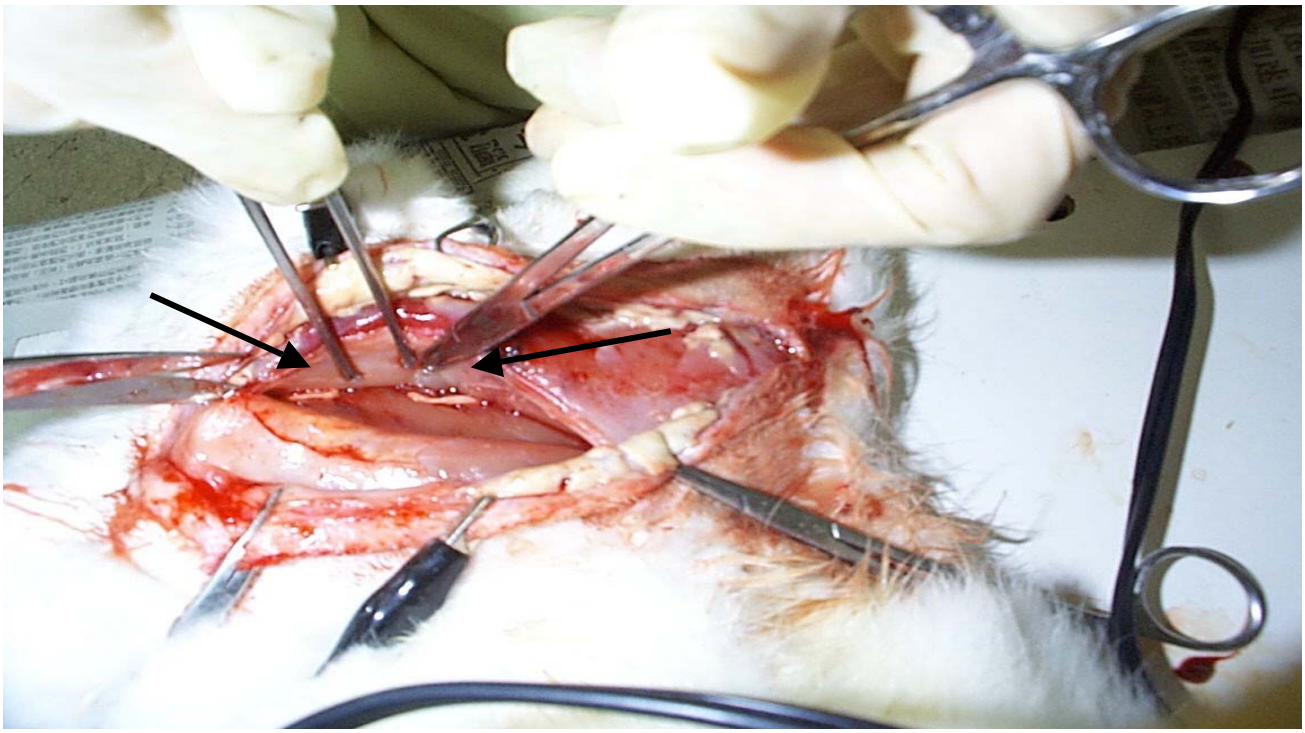

Figure 2. The sciatic nerve (arrow) was cut at the level just above the trifurcation.

After collection of the pre-transection prevalence of SEA, the sciatic nerve was cut with surgical scissors at the level just above the trifurcation (branches to peroneal, tibial and sural nerves) (Fig. 2).

Data Collection

The prevalence of SEA was monitored immediately after the sciatic nerve transection and on defined post-transection period (post transection first day, post transection second day, post transection first week, post transection second week, post transection third week, post transection first month). After each post-transection SEA searching, the skin of lateral thigh was sutured and the rabbit was kept alive for further SEA study.

Control Study

The same procedures of SEA searching were also performed on the other side biceps femoris where the sciatic nerve was exposed but not transected. Similarly, SEA prevalence of the control side was measured on the same day when SEA prevalence of the experimental side was assessed at the defined time points (immediately after transection, post transection 1st day, post transection 2nd day, post transection 1st week, post transection 2nd week, post transection 3rd week, post transection 1st month). The prevalence of SEA on the control side was served as control data to rule out the effect of needling on the occurrence of SEA in an MTrS region.

Data Analysis

The maximal amplitude of a set of SEA (in one screen) with a stable baseline is measured to assess the action potential activity. To measure the maximal amplitude, all the peaks (both negative and positive) of individual identifiable potentials are connected to form an envelop to wrap the SEA of one EMG screen. The maximal height of the envelope is measured as the maximal amplitude of the SEA. Three to five maximal amplitudes of SEAs in an MTrS region were averaged as the mean value of the maximal amplitude of SEA. ANOVA was applied to compare the mean values of experimental data with the control data.

RESULTS

Totally fourteen rabbits were studied. Due to sensory

loss of the lower limb following the transection of sciatic nerve, eight rabbits bit their injured legs to cause leg amputation. Three rabbits (rabbit-3, rabbit-4, and rabbit-8) were found dead during the course of our study.

The prevalence of SEA in the rabbit biceps femoris immediately after sciatic nerve transection was not significantly different from that before transection. Similar result persisted for only one day after the transection of its innervating nerve. SEA disappeared completely on the 2nd day of nerve transection and could not be recorded at all through the rest of this study (to the end of our investigation up to one month after nerve transection) (Table 1). Two rabbits (rabbit-5 and rabbit-6) were even monitored for up to two months and, still, there was no SEA found. Some hyperactive (high-amplitude) electromyographic signals (but not spikes), which occasionally made themselves very difficult to distinguish from SEA, were recorded during the later course of our investigation. These signals, not typical of SEA, were considered to be denervation discharges (diffuse fibrillations), since these signals were never observed in the control side. The prevalence of SEA in the contralateral side where the sciatic nerve was preserved revealed no significant difference during our study course.

One important finding was that the mean value of maximal amplitude of SEA on the nerve-transected side (while SEAs were present) was not significantly different from that collected at different times in the same animal. It was not significantly different from the mean value of maximal amplitude of SEA on the nerve-preserved side (Table 2).

DISCUSSION

Myofascial pain syndrome (MPS) had long been criticized for its lacking of definite scientific characteristics of MTrP. Gerwin had strongly criticized the reliability of MTrP examination and emphasized that it is essential to have hands-on training to achieve a reliable MTrP examination[18]. However, based on recent research studies, mainly electrophysiological, on both animal and human subjects, the pathophysiology of a MTrP becomes much more cleare [3,4,13].

Table 1. Prevalence of spontaneous electrical activity (SEA) in rabbit myofascial trigger spot (MTrS) before and after the transection of the sciatic nerve.

	Before Transaction	Post Transaction Immediately	Post Transaction 1st day	Post Transaction 2nd day	Post Transaction 1st week	Post Transaction 2nd week	Post Transaction 3rd week	Post Transaction 1st month
Rabbit 1	12 / [11]	4 / [--]	10 / [--]	0 / [12]	0*/ [12]	0* / [5]	0*/ [5]	
Rabbit 2	9 / [9]		8 / [7]	0 / [12]	0 / [8]	0* / [5]	0*/ [5]	0*/ [6]
Rabbit 3	10 / [9]	8 / [--]	7 / [6]	0 / [10]	0 / [11]	X		
Rabbit 4	12 / [9]	7 / [--]	7 / [7]	0 / [13]	X			
Rabbit 5	8 / [7]	6 / [--]	5 / [11]	0 / [10]	0 / [12]	0*/ [7]	0 / [4]	0 / [5]
Rabbit 6	7 / [7]	10 / [--]	6 /[10]	0 / [11]	0 / [11]	0*/ [5]	0 / [10]	0 / [7]
Rabbit 7	10 / [3]	9 / [--]	10 / [6]	0 / [5]		0*/ [7]	0*/ [5]	0*/ [6]
Rabbit 8	8 / [7]	5 / [--]	5 / [6]	X				
Rabbit 9	13 / [3]	12 / [--]	4 / [4]	0 / [4]	0 / [3]	0‡/ [3]	0 / [8]	0 / [3]
Rabbit 10	8 / [5]	6 / [--]	6 / [5]	0 / [4]	0 / [5]	0*/ [4]	0 / [7]	0 / [3]
Rabbit 11	9 / [6]	6 / [--]	4 / [9]	0 / [10]	0 / [5]	0 / [10]	0 / [4]	0 / [4]
Rabbit 12	7 / [4]	6 / [--]	6 / [6]	0 / [0]	0 / [0]	0 / [11]	0 / [7]	0 / [7]
Rabbit 13	9 / [6]	10 / [--]	10 / [7]	0 / [10]	0 / [7]	0 / [5]	0 / [7]	0 / [10]
Rabbit 14	9 / [7]	10 / [--]	10 / [5]	0 / [10]	0 / [8]	0 / [9]	0 / [9]	0 / [8]

Data in [ ]: SEA prevalence in the contralateral side where the sciatic nerve was preserved as control data.

* : Hyperactive electromyographic signals similar to SEA (diffuse fibrillations).

X: Rabbit expired.

Table 2. Mean maximal amplitude (μV) of spontaneous electrical activity (SEA) in rabbit myofascial trigger spot (MTrS) before and after the transection of the sciatic nerve.

	Before Transaction	Post Transaction Immediately	Post Transaction 1st day	Post Transaction 2nd day	Post Transaction 1st week	Post Transaction 2nd week	Post Transaction 3rd week	Post Transaction 1st month
Rabbit 1	1.98 ± 0.33	2.23 ± 1.26	1.40 ± 0.30	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	
	(1.48 ± 0.41)			(2.40 ± 1.13)	(1.63 ± 0.13)	(1.30 ± 0.30)	(1.25 ± 0.21)	
Rabbit 2	1.60 ± 0.29		1.10 ± 0.18	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
	(1.28 ± 0.53)		(1.35 ± 0.24)	(1.40 ± 0.17)	(2.05 ± 0.47)	(1.40 ± 0.17)	(1.07 ± 0.15)	(1.13 ± 0.49)
Rabbit 3	1.28 ± 0.54	1.20 ± 0.50	1.23 ± 0.51	0.00 ± 0.00	0.00 ± 0.00	X		
	(1.23 ± 0.31)		(1.30 ± 0.41)	(1.48 ± 0.30)	(1.33 ± 0.40)			
Rabbit 4	1.28 ± 0.33	0.75 ± 0.07	1.73 ± 1.13	0.00 ± 0.00	X			
	(1.05 ± 0.49)		(1.15 ± 0.21)	(1.00 ± 0.53)				
Rabbit 5	1.05 ± 0.35	1.24 ± 0.45	1.10 ± 0.35	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
	(1.53 ± 0.45)		(1.10 ± 0.08)	(1.30 ± 0.48)	(1.17 ± 0.38)	(1.17 ± 0.40)	(2.15 ± 1.00)	(2.20 ± 0.59)
Rabbit 6	1.30 ± 0.39	1.00 ± 0.17	1.08 ± 0.15	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
	(1.10 ± 0.17)		(1.28 ± 0.10)	(1.25 ± 0.54)	(1.10 ± 0.20)	(1.47 ± 0.47)	(1.80 ± 0.71)	(2.63 ± 1.61)
Rabbit 7	1.88 ± 0.26	2.30 ± 0.54	1.90 ± 0.85	0.00 ± 0.00		0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
	(2.20 ± 0.42)		(2.40 ± 0.52)	(2.97 ± 0.68)		(2.55 ± 0.87)	(3.08 ± 0.39)	(3.47 ± 0.35)
Rabbit 8	1.98 ± 0.34	1.90 ± 0.28	1.65 ± 0.06	X				
	(1.83 ± 0.45)		(1.47 ± 0.25)					
Rabbit 9	2.65 ± 0.73	2.68 ± 1.61	2.50 ± 1.74	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
	(3.23 ± 1.34)		(2.77 ± 0.90)	(3.83 ± 1.19)	(3.20 ± 1.25)	(1.70 ± 0.30)	(2.65 ± 0.49)	(2.20 ± 1.56)
Rabbit 10	3.25 ± 1.24	2.20 ± 0.50	2.13 ± 0.84	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
	(2.85 ± 1.06)		(2.33 ± 0.74)	(3.07 ± 0.64)	(2.93 ± 1.78)	(2.30 ± 1.80)	(2.13 ± 1.10)	(2.40 ± 1.13)
Rabbit 11	1.47 ± 0.15	1.63 ± 0.15	1.00 ± 0.14	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
	(1.23 ± 0.52)		(1.63 ± 0.42)	(1.80 ± 0.79)	(2.03 ± 0.42)	(1.65 ± 0.47)	(1.90 ± 0.71)	(1.10 ± 0.14)
Rabbit 12	1.17 ± 0.40	1.43 ± 0.43	1.13 ± 0.49	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
	(1.35 ± 0.21)		(1.20 ± 0.14)	(1.97 ± 0.46)	(1.37 ± 0.15)	(1.25 ± 0.31)	(1.20 ± 0.14)	(1.55 ± 0.10)
Rabbit 13	1.85 ± 0.62	1.60 ± 0.35	1.53 ± 0.40	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
	(1.35 ± 0.17)		(1.53 ± 0.23)	(2.03 ± 0.55)	(2.10 ± 0.37)	(1.90 ± 0.12)	(1.60 ± 0.37)	(2.05 ± 0.26)
Rabbit 14	1.35 ± 0.39	1.50 ± 0.25	1.57 ± 0.49	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00	0.00 ± 0.00
	(1.50 ± 0.28)		(1.40 ± 0.35)	(2.13 ± 0.15)	(1.43 ± 0.32)	(1.58 ± 0.32)	(2.23 ± 0.21)	(1.90 ± 0.77)

註：Data in ( ) under experimental values: Mean values in the contralateral side where the sciatic nerve was preserved as control data.

X: Rabbit expired.

The scientific basis of MTrP was first supported by the studies on LTR on both human subjects and animals. LTR is considered as a valuable objective sign of MTrP[1,2]. The minute sensitive site from which an LTR can be elicited by snapping or needle stimulation has been defined as a *sensitive locus* in an MTrP region[6]. Hong et al has demonstrated a sensory nerve fiber near the LTR locus in a primitive histological study[19]. It is very likely that the LTR locus is a sensitized nociceptor (or sensitized nociceptors). Therefore, a LTR locus

(sensitive locus) is the sensory component of the MTrP locus[13,14]. These sensitive loci are widely distributed in the whole muscle, but more concentrated in the endplate zone based on clinical observation during MTrP injection and an algometer study[20]. An animal study on the rat biceps femoris muscles (mapping LTR loci) has also supported that[21]. When the needle is placed on a sensitive locus, the nociceptors in this locus can be activated and both local pain and referred pain can be elicited. LTRs could be elicited in one MTrP region when the needle was inserted rapidly into multiple sites of that region[6,22,23].

Recent human and rabbit studies also have shown that SEA can be recorded from multiple minute sites in an MTrP (human) or an MTrS (rabbit) region[7,8,9,12]. It had been noted that more SEAs were recorded from an MTrP region than from a non-MTrP region in both human and animal studies[8,12]. By applying mechanical stimuli to the endplate region to convert the normal (discrete negative monophasic) endplate potentials into abnormal continuous noise-like action potentials (similar to the SEA), Liley confirmed these electrical potentials (SEA) as abnormal endplate potentials[24]. In Wiederholt's histological and pharmacological studies on rabbit skeletal muscle, he described an electrical activity similar to SEA and confirmed this activity as "endplate noise" [25]. Ito et al demonstrated that this abnormal pattern of endplate potentials was attributed to excessive release of ACh packets[26]. Comparing the SEA patterns to the endplate activities mentioned by electromyographer[27], there is almost no difference between these two tracings. All of these studies supported Simons' conclusion that the origin of SEA was at or very near a motor endplate[3,4,8,12]. Hong et al used iron deposition technique (by applying DC current, 50 to 100 μV, for 90 sec) at the active locus of MTrS where SEA was recorded and found that small nerve fibers (probably nociceptive nerve endings) were in the vicinity of iron-stain spots (the site of recording needle tip)[28]. Therefore the active locus in an MTrS region is also probably related to nociceptors. Simons concluded that the SEAs found in active loci of MTrPs are abnormal patterns of endplate electrical activity resulting from excessive ACh leakage[8].

Hong has proposed a multiple loci theory for MTrP[6,22]. There are multiple small sensitive loci in an MTrP region. The basic unit of an MTrP, an MTrP locus, consists of a sensitive locus (LTR locus) and an active locus (SEA locus)[13,14]. 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. Since referred pain can also be elicited in "normal" muscle tissue (high pressure, however, is required to elicit it), sensitive loci are nonspecific pain sites and are not necessary found at the MTrP region[20]. It is possible that the sensitive loci are widely distributed in the entire muscle but are concentrated in the endplate zone[20,21]. The sensitive loci not accompanied with active loci are probably non-specific sensitized nociceptors not related to the taut band. They can be caused by other factors such as local trauma or inflammation. Only when the sensitive loci accumulated at the vicinity of active loci, an MTrP can be formed. On the other hand, active loci can be found only in the taut band (or precursor of taut band), and the taut band should contains active loci, although taut band may contains no MTrP. Therefore, in the pathophysiological sense, an MTrP is better defined by the existence of active loci rather than sensitive loci[13].

Based on animal studies, LTR is mediated through the spinal cord in response to the stimulation of a sensitive locus that is in the immediate vicinity of an active locus[5]. Hong and Yu have found that SEA is not significantly influenced by the spinal cord or higher centers[16]. They have concluded that SEA appears to be a local motor endplate phenomenon[16]. However, based on their study, it could not be completely excluded that SEA is a local phenomenon *not related to the ACh release in the end plates*. Our study revealed that SEA was not permanently persistent after the transection of its innervating nerve. SEA remained present for only one day after the transection. It appears that nerve impulse from its innervating nerve (neural mechanism) does not plays a significant role in the occurrence of SEA since SEA does not disappear immediately after transection of the innervating nerve. On the other hand, neural transmitters delivered from the nerve terminals (humoral mechanism) is more likely to be the most important factor related to the occurrence of SEA since ACh *should* still present in the nerve terminals one day after nerve transection (in rabbit).

The similarity of SEA morphology and prevalence in the control data during the whole study course supported that the needling during SEA-searching has little influences on the prevalence and amplitude of SEA.

CONCLUSION

SEA is one of the most important characteristics of myofascial trigger point. Unlike LTR, SEA is not controlled or influenced by the neural circuit in the central nervous system. This study further supported that SEA is a local phenomenon of dysfunctional motor endplate, which is controlled or influenced by the humoral mechanism (Acetylcholine release) rather than the neural mechanism (direct nerve impulses from the central nervous system).

ACKNOWLEDGEMENT

This study was supported by National Science Council (Taiwan) Grant NSC89-2320-B-006-030 and Ministry of Education, Academic Excellence Grant 89-B-FA08-1-4. The authors would like to thank to Lin-Yi Chou, Shu-Hwei Chen, and Chi-Chen Chuan for their valuable contributions in laboratory assistance.

REFERENCES

1. Travell JG, Simons DG. Myofascial pain and dysfunction: the trigger point manual, Volume 1, Baltimore: Williams & Wilkins; 1999. p.31-45.
2. Travell JG, Simons DG. Myofascial pain and dysfunction: the trigger point manual, Volume 2, Baltimore: Williams & Wilkins; 1992. p.1-6.
3. Hong CZ. Pathophysiology of myofascial trigger point. J Formos Med Assoc 1996;95:93-104.
4. Simons DG. Clinical and etiological update of myofascial pain from trigger points. J Musculoske Pain 1996; 4:93-121.
5. Hong CZ, Torigoe Y. Electrophysiologic characteristics of localized twitch responses in responsive bands of rabbit skeletal muscle fibers. J Musculoske Pain 1994;2(2):17-43.
6. Hong CZ. Consideration and recommendation of myofascial trigger point injection. J Musculoske Pain 1994;2(1):29-59.
7. Hubbard DR, Berkoff GM. Myofascial trigger points show spontaneous needle EMG activity. Spine 1993; 18:1803-7.
8. Simons DG, Hong CZ, Simons LS. Nature of myofascial trigger points: active loci. J Musculoske Pain 1995;3(Suppl 1):62.
9. Simons DG, Hong CZ, Simons LS. Spontaneous electrical activity of trigger points [abstract]. J Musculoske Pain 1995;3 (Suppl 1):124.
10. Simons DG, Hong CZ, Simons LS. Spike activity in trigger points [abstract]. J Musculoske Pain 1995; 3(Suppl 1):125.
11. Hubbard DR. Chronic and recurrent muscle pain: pathophysiology and treatment, and review of pharmacologic studies. J Musculoske Pain 1996;4(1/2):123-43.
12. Simons DG, Hong CZ, Simons LS. Prevalence of spontaneous electrical activity at trigger spots and at control sites in rabbit skeletal muscle. J Musculoske Pain 1995;3(1):35-48.
13. Hong CZ, Simons DG. Pathophysiologic and electrophysiologic mechanisms of myofascial trigger points. Arch Phys Med Rehabil 1998;79:863-72.
14. Kuan TS, Chen SM, Chen JT, et al. The basic unit of a myofascial trigger point. J Rehab Med Assoc ROC 1998;26:161-8.
15. Hong CZ, Torigoe Y, Yu J. The localized twitch responses in responsive bands of rabbit skeletal muscle fibers are related to the reflexes at spinal cord level. J Musculoske Pain 1995;3(1):15-33.
16. Hong CZ, Yu J. Spontaneous electrical activity of rabbit trigger spot after transection of spinal cord and peripheral nerve. J Musculoske Pain 1998;6(4):45-58.
17. Moulder JB. Anesthesia in the rabbit using a combination of Ketamine and Promazine. Lab Animal Sci 1978;28:321-2.
18. Gerwin RD, Shannon S, Hong CZ, et al. Identification of myofascial trigger points: interrater agreement and effect of training. Pain 1997;69:65-73.
19. Hong CZ, Chen JT, Chen SM, et al. Histological findings of responsive loci in a myofascial trigger spot of rabbit skeletal muscle from where localized twitch responses could be elicited. Arch Phys Med Rehabil 1996;77:962.

20. Hong CZ, Chen YN, Twehous D, et al. Pressure threshold for referred pain by compression on the trigger point and adjacent areas. J Musculoske Pain 1996; 4:61-79.
21. Chang YC, Kao SF, Kuan TS, et al. Distribution of sensitive loci where localized twitch responses can be elicited in rat skeletal muscle. J Rehab Med Assoc ROC 1998; 26:1-8.
22. Hong CZ. Myofascial trigger point injection. Cri Rev Phys Rehabil Med 1993;5:203-17.
23. Hong CZ, Simons DG. Response to standard treatment for pectoralis minor myofascial pain syndrome after whiplash. J Musculoske Pain 1993;1:89-131.
24. Liley AW. An investigation of spontaneous activity at the neuromuscular junction of the rat. J Physiol 1956; 132:650-66.
25. Wiederholt WC. 'End-plate noise' in electromyography. Neurology 1970; 20: 214-24.
26. Ito Y, Miledi R, Vincent A. Transmitter release induced by a 'factor' in rabbit serum. Pro R Soc Lond B 1974;187:235-41.
27. Kimura J: Electrodiagnosis in diseases of nerve and muscle. Philadelphia: F.A. Davis; 1989. p.232-3.
28. Hong CZ, Chen JT, Chen SM, et al. Sensitive loci in a myofascial trigger point region are related to sensory nerve fibers [abstract]. Am J Phys Med Rehabil 1997; 76:172.

切除支配神經對兔子骨骼肌上激痛點內之自發性電位活動的影響

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肌筋膜疼痛症候群(MPS)以激痛點(MTrP)的存在爲其特徵，局部抽搐反應(LTR)和自發性電位活動(SEA)是激痛點之兩個很重要的客觀性指標。激痛點的基本單元，就是肌筋膜激痛點小點(MTrP locus)，它包含了*感覺性成分*(感覺小點；或 LTR 小點)與*運動性成分*(活動小點；或 SEA 小點)。在動物實驗中發現，在高於支配該肌肉之下運動神經元處截斷其脊髓後，兔子的 LTR 在脊髓休克期時會被暫時性地壓抑，可見 LTR 顯然經由脊髓來做調節。在激痛點裡的 SEA，是因爲乙醯膽素(Acetylcholine)過量釋放所引起的不正常之終板電位(endplate potentials)。不過，由於 SEA 的出現率在脊髓截斷後並沒有很明顯的改變，SEA 應該不是經由脊髓來做調節。本研究收集了十四隻紐西蘭成兔，來評估所支配之神經被切除後，其對激痛點的 SEA 所造成的影響。我們在兔子坐骨神經切除前、切除之後、切除後第一天、切除後第二天、切除後第一週、切除後第二週、切除後第三週與切除後第一個月，分別追蹤兔子股二頭肌上 SEA 的發生率。發現 SEA 在神經切除後還持續存在約一天，但從切除後第二天開始，SEA 就消失不見了，直到本研究追蹤結束時。

由本研究的結果可知，SEA 的發生，神經脈衝傳導的神經性因素是比較不重要，而神經介質傳導的內分泌性因素則相當重要。此研究更進一步證實肌筋膜激痛點裡的 SEA，是運動終板機能障礙所導致的一種局部性的現象。（中華復健醫誌 2001; 29(2): 65 - 75）

關鍵詞：肌筋膜疼痛症候群(myofascial pain syndrome)，(人類)激痛點 (myofascial trigger point)，(動物)激痛點 (myofascial trigger spot)，自發性電位活動(spontaneous electrical activity)，局部抽搐反應 (local twitch response)

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