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Review Article

Myofascial Low Back Pain

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Objective: This review article describes the etiology, pathogenesis, clinical characters and management of low back pain (LBP) caused by myofascial trigger points (MTrPs), i.e. myofascial low back pain.

Findings: Based on the currently available knowledge and our clinical experience, we analyzed the basic and clinical aspects of myofascial LBP. Most cases of myofascial LBP are related to injuries, either current or previous. Active MTrPs that cause LBP are usually activated as a consequence of other etiological lesions. Therefore, it is important to determine and treat the underlying pathological lesions in order to avoid recurrence of myofascial LBP. If the underlying pathological lesion is unable to be identified and the pain in MTrPs is very severe, we may still have to suppress the active MTrP for pain control. To inactivate MTrPs, effective approaches include manual therapy, physical therapy modalities, and needling including acupuncture and MTrP injection.

Conclusion: It is important to find out the underlying etiological lesion, which causes LBP, and to provide appropriate management based on our best knowledge. (Tw J Phys Med Rehabil 2008; 36(1): 1 - 14)

Key Words: myofascial pain, myofascial trigger points, low back pain

INTRODUCTION

Background of Low Back Pain due to Myofascial Trigger Points

Low back pain (LBP) syndrome has been considered as one of the important causes of disability.1-3 Simons and Travell have described the myofascial origins of LBP (myofascial low back pain).4-6 Previously, the diagnosis of myofascial LBP is usually given when there is no organic lesion can be identified. This unscientific approach should be clarified since the pathophysiology of myofascial trigger points (MTrPs) has now been better understood.7,9 In clinical practice, MTrPs can be frequently identified in the trunk muscles and lower limb muscles in patients with LBP due to various causes including lumbar disc lesions and facet joint lesions in addition to myofascial LBP.4-6,10 Therefore, myofascial LBP is not a synonym of “LBP with MTrPs”. In some LBP patients, the pain in the low back is caused by the MTrPs in the lumbar paraspinal muscles.4-6 The thera-
peutic approach to myofascial LBP has usually focused on the elimination of MTrPs including manual therapy, physical therapy modalities, and needling of MTrPs. However, it may only provide temporary pain relief. The recurrent rate seems to be fairly high based on the clinical observation on our patients who had been previously treated simply with MTrP relief. The major reason of the therapeutic failure is due to an inaccurate diagnosis and/or an inappropriate treatment. The understanding of the pathogenesis of MTrPs in LBP is a critical issue to provide an optimal therapeutic approach in the management of myofascial LBP.

Clinical Characteristics of Trigger Points

Myofascial trigger points have been defined as the hyperirritable (hypersensitive) spots in a taut band of skeletal muscle fibers.[9] Some important clinical observations and basic science studies have supported the existence of MTrPs. All MTrPs locate within the endplate zone,[8,9,11-14] and the endplate noise (EPN) can be recorded more frequently in an MTrP region than a region with normal muscle tissue.[9,12-17] Based on the figures showing the location of MTrPs in the Trigger Point Manual,[9,18] a most tender spot, the latent MTrP (tender, but not painful spontaneously), can be identified in almost all normal adult skeletal muscles. Latent MTrPs can be observed in the early life, but not in newborns or babies less than one-year-old.[19,20] A latent MTrP can be activated to become an active MTrP, which is painful and much tendered. In clinical observation, when an active MTrP is suppressed, it is still tender but not painful, since it becomes a latent MTrP. The latent MTrP can be activated to become an active one secondary to a certain pathological lesion. After appropriate treatment of this lesion, the activated MTrP can be suppressed to be inactive. Theoretically, the MTrP does not disappear, but just converted from active to latent.[7,8,19] Myofascial pain syndrome is a pain phenomenon due to activation of latent MTrPs as a consequence of certain pathological conditions including chronic repetitive minor muscle strain, poor posture, systemic disease, or neuromusculoskeletal lesions such as strain, sprain, enthesopathy, arthritis, vertebra disc lesion, etc.[8,19,21,22] Compression of the MTrP can reproduce or aggravate a patient’s usual complaint of pain recognition,[23] and inactivation of the MTrP can relieve the pain and uncomfortable symptom. Stronger compression of MTrP can elicit referred pain.[24,25] For different patients, similar referred pain patterns can be elicited by compression of the same MTrP in each individual muscle.[9,18] Needling to the tiny loci (nociceptors) in the MTrP region can induce pain, referred pain, and local twitch response (LTR, a brisk contraction of muscle fibers in the taut band), which can be recorded electromyographically.[26-28] High-pressure stimulation, such as needling, to the MTrP can elicit LTR and suppress the pain.[16,19,21,29] Immediate relief of MTrP pain can be expected if LTRs are elicited during needling of the MTrP.[21,29,30]

Pathophysiology of Trigger Points

Based on recent human and animal studies, it has been concluded that there are multiple MTrP loci in an MTrP region.[7,9,19,21,22,29] An MTrP locus contains both sensory and motor components. The sensory component of the MTrP locus is the sensitive locus from which pain, referred pain, and LTR can be elicited in response to a high pressure mechanical stimulation. It probably contains one or more nociceptors[24,31] and is also defined as an LTR locus. The motor component is the active locus from where EPN can be recorded using electromyography (EMG);[7,9,12-14] it is also defined as an EPN locus. It is probably a dysfunctional endplate with excessive acetylcholine leakage.[8,13,14,16,17] It may be the precursor to form a taut band based on the evidence of local contracture of sarcomeres near the endplate region. There are morphological evidences of taut bands and contraction knots in the MTrP region (endplate zone) in EMG and ultrasonic studies.[9,32]

In a latent MTrP, there are a few MTrP loci (sensitized nociceptors) that are painful only in response to pressure compression (tenderness).[22] When the compression pressure is increased, referred tenderness (pain in the remote sites in response to pressure compression) may occur. High-pressure stimulation (needling) to the MTrP loci of a latent MTrP can also elicit LTRs. An active MTrP contains more MTrP loci than a latent one. Less pressure is required to elicit referred pain or LTR in an active MTrP than a latent MTrP. A very active MTrP may have spontaneous referred pain (without pressure compression to the MTrP). It has been suggested that a very
active MTrP contains many MTrP loci, but a latent MTrP contains only a few MTrP loci. Therefore, the amount of MTrP loci in one MTrP region is proportionate to the irritability of that MTrP.

Both referred pain and LTR are integrated in the spinal cord. The term “myofascial trigger point circuit (MTrP circuit)” has been used to represent the interneuronal connections in the dorsal horn of spinal cord. Via these connections, persistent pain, referred pain, LTR, and autonomic influence may occur (Figure 1).

PATHOGENESIS AND CHARACTERISTICS OF MYOFASCIAL LOW BACK PAIN

In clinical practice, LBP syndrome is usually divided into two categories based on the presence of abnormal findings in radiological studies including x-ray, magnetic resonance imaging (MRI), computerized tomography scan, etc (Table 1). In fact, most lesions with abnormal radiological findings are originally caused by soft tissue (especially ligament) injury. The abnormal radiological findings including vertebral osteophytes, disc space narrowing, facet joint instability (vertebral retrolisthesis or anterolisthesis, mild degree), are the consequences of ligament injury (Figure 2). In the acute stage, we may just make a diagnosis of “sprain of spine” when we see no neurological or radiological findings. Many years later, in the chronic stage, we call it “degenerative joint disease of spine” or “degenerative disc disease of spine” when osteophytes or disc space narrowing can be observed in the X-ray film. Therefore, for many patients with low back pain, there may be no radiological finding in the acute stage, but abnormal radiological findings can be observed in the chronic stage. Hong has suspected that most of degenerative lesions occurred under age of 60 years are related to previous injuries with either significant tissue damages or repetitive minor trauma (unpublished data). Age is another factor to cause degenerative lesions. Without previous injury, no degenerative lesion may occur as a consequence of aging process until significantly old enough. Hong interviewed more than 20 patients who were older than 80 years and had no obvious degenerative changes in the x-ray of lumbar spine. All of them could recall neither any significant back injury nor history of heavy weight bearing (unpublished data).

Myofascial Low Back Pain without Radiological Findings

The LBP in this category includes those in the acute stage of soft tissue injury and minor chronic trauma without secondary bony changes. It is usually due to ligament lesion, tendon lesion, or muscle strain. It is always associated with ipsilateral paraspinal muscle spasm. The distribution of MTrPs is also in the lesion side. Unfortunately, in most cases, the location of soft tissue lesion cannot be identified accurately. Theoretically, most LBP caused by the soft tissue lesions are mostly due to ligament injury as described below.

In the initial stage of disc lesion, annulus fibrosus ligaments are stretched or torn. Subsequently, the insertion site at the vertebral body would have chronic inflammation with periosteum irritation that eventually would induce the formation of osteophytes. In acute stage of lumbar disc herniation, severe pain and tingling frequently occur in the lower limb, but low back pain may not be a major symptom. In such case, no or little MTrPs can be identified in the back muscles, and this is not a case of myofascial LBP.

Similarly, in the early stage of facet joint lesion, the ligaments around the facet joint are stretched, and later become loosening of facet ligaments that would cause mild spondylolisthesis (anterolisthesis or retrolisthesis) between two vertebrae, and finally, form osteophytes in the intervertebral foramen or hypertrophic facets. In many cases of facet joint lesion, LBP can be caused by the existence of MTrPs in the ipsilateral paraspinal muscles. However, some patients may just have sore pain in the sacral and gluteal regions without MTrPs, and cannot be diagnosed as myofascial LBP.

Young persons involved in heavy sports or heavy lifting may have sprain of iliolumbar ligaments. They may have active MTrPs in the ipsilateral lower lumbar paraspinal muscles and sometimes in the ipsilateral gluteal muscles, but rarely in the lower limb muscles.

Patients with fibromyalgia may have low back pain due to MTrPs. Hong and Simons have suggested that a fibromyalgia patient has a lower pain threshold than normal person and thus many latent MTrPs become active ones. MTrPs in paraspinal muscles of a fibromyalgia
patient is usually symmetrically distributed. However, if a fibromyalgia patient has pre-existing injury in one side, there may be more active MTrPs in the pre-injured side than the other side.

**Figure 1.** The “MTrP Circuit”. MTrP = myofascial trigger point, ReP = referred pain, LTR = local twitch response

**Examples of Bony Lesion from Poor Healing of Acute Injury**

<table>
<thead>
<tr>
<th>Acute Stage</th>
<th>Chronic Stage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disc Herniation [annulus fibrosus]</td>
<td>Reduced disc space; Osteophyte of Vertebral Body</td>
</tr>
<tr>
<td>Ligament Injury</td>
<td>Spondylolisthesis; Osteophyte of Facet Joint</td>
</tr>
<tr>
<td>Facet Ligament Sprain</td>
<td>Osteophyte of Iliac Crest [posterior]</td>
</tr>
<tr>
<td>Iliolumbar Ligament Sprain</td>
<td>Osteophyte of Iliac Crest [anterior]</td>
</tr>
<tr>
<td>Tendon/ Muscle Injury</td>
<td>Quadratus Lumborum Strain</td>
</tr>
</tbody>
</table>

**Figure 2.** Abnormal radiological findings in chronic stage of soft tissue lesion
Table 1. Classification of myofascial low back pain

I. LBP without Radiological Findings:
   A. Lesions in the anterior segment of spine:
      1. Strain of psoas muscles.
      2. Disc lesions –MTrPs in paraspinal muscles.
      4. Others.
   B. Lesions in the posterior segment of spine:
      1. Strain of quadratus lumborum.
      2. Strain of multifidi.
      3. Strain of longissimus.
      4. Strain of iliocostalis.
      5. Sprain of iliolumbar ligament.
      6. Sprain of facet joint –MTrPs of multifidi and longissimus.
      7. Sprain of interspinous ligament –MTrPs in multifidi.
      8. Others.
   C. Others: spinal cord lesions (tumor, transverse myelitis, infection, infarction, etc.), nerve root lesions, visceral organ lesions, peripheral nerve lesions (polyneuritis, herpes zoster, etc.), etc.

II. LBP with Radiological Findings:
   A. Lesions in the anterior segment of spine:
      1. Degenerative disc lesions –MTrPs in paraspinal muscles.
      2. Compression fracture of vertebral body –MTrPs in paraspinal muscles.
      3. Others.
   B. Lesions in the posterior segment of spine:
      1. Facet joint lesions –MTrPs in multifidi and longissimus.
      2. Spondylolysis / Spondylolisthesis –MTrPs in paraspinal muscles.
      3. Others: transverse process fracture, etc.
   C. Others: osteophytes in iliac crest (iliolumbar ligament lesions, iliocostalis lesions, quadratus lumborum lesions), etc.

LBP = low back pain, MTrP = myofascial trigger point

Myofascial Low Back Pain with Radiological Findings

The most common causes of LBP in this category are disc lesions (anterior segment of spine) and facet joint lesions (posterior segment of spine).\(^{[3,36]}\) Either traumatic or degenerative lesions in the disc or facet joint may cause nerve root irritation or compression to elicit radicular pain. The paraspinal muscle spasm is usually in the side opposite to the radicular pain, in order to avoid further compression. In such cases, active MTrPs can be found in the ipsilateral limb muscles in addition to the paraspinal muscles.

A facet lesion can be caused by a direct injury or secondary to a chronic disc lesion. Direct injury can cause loosening of facet ligament or damage to the facet joint. Chronic disc lesions usually produce intra-disc desiccation, and, subsequently, decrease of disc space. When two consecutive vertebral bodies are coming together, the corresponding facet joint may become unstable, and then injured as a consequence of repetitive spinal movement. In our clinical experience, L4-5 facet lesion may activate MTrPs in the middle and lower lumbar paraspinal muscles, gluteus minimus, and gluteus medius muscles, while L5-S1 facet lesion may activate MTrPs in the lower lumbar paraspinal muscles, piriformis, and gluteus maximus.
muscles. Active MTrP in L4-S1 multifidus can cause referred pain to the L4-S1 interspinous ligament and ipsilateral posterior superior iliac spine, and sometimes, sacro-iliac (SI) joint. Frequently, MTrPs in lower lumbar multifidi would be mis-diagnosed as SI dysfunction. In such case, treatment of SI joint, even with local steroid injection, cannot relieve the SI pain. Gluteus minimus and piriformis are the only two gluteal muscles that can elicit pain referred down to the leg and foot, similar to radicular pain, but no associated tingling sensation.

**DIAGNOSIS OF MYOFASCIAL LOW BACK PAIN**

**Identification of Myofascial Trigger Points in the Paraspinal Muscles and Limb Muscles**

1. **Pointing Out by Patient:** This is the easiest way to find an MTrP. When the painful region consists of only one single MTrP or few MTrPs, the patient can use a fingertip to point out the painful spot. When a patient has multiple MTrPs in a local area or several MTrPs distributed in a large portion or several different areas of the body, it may be difficult for the patient to point them out. In such cases, the examiner has to palpate the MTrPs carefully.

2. **Palpation:** Palpation of taut bands and MTrPs is the most important procedure to make an accurate diagnosis of MTrPs. The technique of palpation has been described in detail. However, the regular technique of pincer palpation or snap palpation cannot be applied on the lumbar paraspinal muscle when an MTrP is deeply seated. In such case, deep pressure palpation should be used to locate the MTrP. During the pressure compression, in some cases, referred pain with a typical pattern can also be elicited that can help to confirm the location of a certain MTrP. In an active MTrP, the referred pain pattern can be elicited much more easily than a latent one. The referred pain elicited by the pressure compression is defined as “referred tenderness” (to distinguish from the spontaneous referred pain).

3. **Pain Recognition:** Pain recognition is the most important sign to confirm the accurate MTrP to be treated. The patient should recognize the identified MTrP as the one to cause or to aggravate the pain or discomfort similar to patient’s primary complaint.

**Identification of Etiological Lesions of Low Back Pain**

The first stratagem for the management of myofascial LBP is to identify the underlying etiological lesion that causes LBP. The following steps are required to confirm the etiological lesion of LBP: 1. pain history, 2. functional limitation, and 3. Provoking tests. Sometimes, imaging studies (sonography, MRI, etc) and electrophysiological studies (EMG, nerve conduction, etc.) may be necessary.

A patient with radiculopathy usually complains of tingling in the ipsilateral lower extremity in the corresponding dermatome. In the acute stage of radiculopathy due to disc herniation, the patient may have extreme limb pain in the related dermatome and myotome few hours after the onset of disc herniation as a consequence of severe chemical inflammation. Chronic LBP with a referred pain around the gluteal area is usually related to lumbar facet lesion.

Forward flexion of lumbar spine would cause or aggravate pain if the lesion is in the anterior segment, such as disc herniation. On the other hand, ipsilateral rotation followed by extension of spine (facet sign) would cause or aggravate pain if the lesion is in the posterior segment, such as facet joint sprain or arthritis. Facet sign is a provoking test that can stretch the facet ligament or compress the facet joint in order to elicit the pain similar to patient’s complaint (pain recognition). In the case of spinal stenosis, spondylolisthesis, or compression fracture of vertebral body, either flexion or extension of lumbar spine would cause or aggravate pain (provoking test). In the case of ilio-lumbar ligament sprain, simultaneous flexion and rotation of lumbar spine would cause or aggravate pain.

Plain films of x-ray are usually required to confirm the diagnosis of lesions with radiographic changes. In acute stage of disc herniation or protrusion, the radiological findings are usually unremarkable. However, in the chronic stage when the involved disc is desiccated, a decrease of intervertebral disc space can be noticed in the plain films. If the disc space is reduced, facet instability may occur to cause facet lesion. In the plain film, even a
mild degree of anterolisthesis or retrolisthesis can usually indicate the evidence of facet joint instability. Soft tissue lesions with negative findings in the plain film may be confirmed by MRI studies.

Sonographic techniques can help to identify degenerative and inflammatory processes at certain vertebral levels when only soft tissue structures are involved. Ultrasonography could be very useful for both the diagnosis and the assessment of spondylarthropathy activity. Comparing with MRI investigation, transabdominal ultrasonography of the lumbar herniated disc proved to be distinctly inferior because of methodical limitations and lower diagnostic accuracy.

Electromyography or nerve conduction studies of the proximal segment can provide the assessment of the nerve function and may also help to locate the level of the lesion.

**Guidelines for Treatment of Etiological Lesions**

For a severe acute lesion involving tendon, ligament, joint, or bone, a certain period of immobilization, such as application of corset or brace, may be required. Avoidance of over-movement can provide adequate time for complete healing. Muscle relaxant is not frequently necessary, since the sedative effect, generalized weakness, or other side effects, may be harmful. Muscle relaxation is not equal to immobilization. During immobilization, rhythmic isometric muscle contraction, if it is not very painful, is encouraged since it may improve local circulation, and thus, facilitates the healing process and avoids scar tissue formation, which may impair local vascularity in the later life. In acute stage, systemic administration of non-steroidal anti-inflammatory drug (NSAID) immediately after trauma may prevent the consequence of chronic changes, especially scar tissues. Strong analgesic medicine is given only if the pain is intolerable.

In chronic cases, scar tissues may interfere with local circulation in the chronic inflammatory site. Therefore, systemic NSAIDs may not be very effective on the chronic inflammatory site due to poor absorption in sites with poor circulation. Systemic NSAIDs may have effectiveness for pain relief only, but not for chronic inflammation. In such case, local steroid injections may be effective to eliminate the chronic inflammatory lesion. However, concomitant application of heat over the chronic inflammatory site to improve local circulation may facilitate the absorption of systemic medication. Therefore, when a patient is given oral NSAIDs for a chronic lesion, local application of heat may facilitate the anti-inflammatory action of systemic NSAIDs.

Physical therapy is usually required for the treatment of chronic LBP. Heat (thermotherapy) can cause vasodilatation and improvement of local circulation. It can also provide adequate relaxation, which allows the patient to perform an exercise program, and it should therefore be performed prior to each exercise session. To improve local circulation, superficial heat is usually adequate to cause vasodilatation in both superficial and deep tissues. In both cases, the hemodynamic changes are due to reflex autonomic responses. Direct spread of thermal energy via skin is quite limited. In our clinical
experience, therapeutic ultrasound is very effective in treating lumbar facet lesions. Manual therapy encompasses all forms of massage, mobilization, manipulation, and traction and is frequently used for treating chronic musculoskeletal injuries. Benefits include reductions in edema and spasm and improving flexibility and range of joint motion, as well as psychological effects. Massage, mobilization, or manipulation therapy is frequently used to improve local circulation and provides muscle relaxation. Manipulation may cause immediate pain relief by stretching the tight muscle or by sharp stimulation to the facet joint. Intermittent pelvic traction may reduce the paraspinal muscle spasm quickly. Exercise therapy is frequently prescribed for muscle strengthening, stretching, increasing circulation, and relaxation. Additionally, back schools also reduce pain and improve function for patients with chronic and recurrent LBP. Modified William exercise for posterior segment lesions and McKenzie exercise for anterior segment lesions may be beneficial in relieving LBP, and can be instructed to patients as home programs. For patients with degenerative joint lesions, isometric exercise is recommended to increase muscle strength for joint protection. Dynamic exercise can improve microcirculation if it is carried out carefully. The general principle is to avoid heavy, rapid, or pronged exercise.

Local steroid injection can be used for anti-inflammatory in chronic cases, since oral NSAIDs may not be well absorbed via scar tissues. In the acute stage, oral NSAIDs can be effective for inflammatory control, and local steroid injection is usually unnecessary. Facet injection with steroid may be beneficial in relieving LBP, and can be instructed to patients as home programs. For patients with MTrPs in addition to the thermal effects, electrotherapy in the form of nerve stimulation, such as transcutaneous electrical nerve stimulation, can provide temporary pain relief, while muscle stimulation can be effective for the relief of muscle tightness. The muscle contraction caused by the electrical stimulation is similar to focal massage and can improve local circulation. Therapeutic effectiveness of electrotherapy on MTrPs has been documented. A study suggested that a combination of ultrasound and electrotherapy could provide better results than a single therapy. Laser (Light Amplification by Stimulated Emission of Radiation) therapy is a new modality used in pain control. It seems to be successful in relieving pain and improving function in myofascial pain syndrome. However, its mechanism on MTrP therapy is still not established. Leinfort and Foley considered laser as a needless (painless) acupuncture. The electromagnetic energy from laser may penetrate and irritate the MTrP and provide a hyperstimulation analgesia similar to dry needling, but not via the pain pathway. Snyder-Mackler et al found an increase in skin resistance after laser therapy and suggested its sympathetically mediated effect.

Regarding therapeutic exercise, patients who have

**Myofascial Therapy: Inactivation of Active Myofascial Trigger Points**

The commonly used methods for MTrP therapy include physical therapy, chiropractic manipulation, and needling. The frequently used physical therapy programs to inactivate MTrP are manual therapy, therapeutic modalities, and therapeutic exercise. Procedures of MTrP needling include acupuncture, dry needling, and MTrP injection.
clinical evidence of fibromyalgia syndrome should perform conditioning exercise. There is an evidence that generalized conditioning exercise can activate the endogenous opioid system. Chiropractic manipulation has been one of the most popular techniques for pain control in United States of America. Significant relief of MTrP pain after spinal manipulation therapy has been documented in the literature. However, the mechanism of pain control is still unclear. It is probably that the pain relief effect is a result of re-arrangement of neural connections in an “MTrP circuit” from the mechanical stimulation to the nociceptors in the “facet trigger points” similar to hyper-stimulation analgesia during needling to an MTrP. It is also likely that lumbar manipulation can stretch the tight paraspinal muscles to provide muscle relaxation and to improve local circulation.

Trigger point injection has been considered to be very effective for an immediate inactivation of MTrPs. Before considering MTrP injection, the underlying etiological lesion should be treated and conservative treatment for the inactivation of MTrP should be tried. The frequently injected paraspinal muscles included iliocostalis, longissimus, multifidus, and quadratus lumborum. During injection of a superficial MTrP, the exact location of MTrP should be identified and confirmed by a finger of the non-dominant and the syringe held by the dominant hand. Local twitch responses should be elicited as much as possible to ensure that many sensitive LTR loci in the MTrP region are encountered. Hong has suggested a “fast-in and fast-out” technique to provide high pressure of needle insertion for eliciting LTRs and to avoid side movement of the needle. When a deep muscle is injected, the needle can be perpendicularly inserted into the MTrP region since simultaneous palpation of the MTrP and taut band during injection is unlikely. Travell and Simons has recommended using 0.5 percent procaine or lidocaine for MTrP injection. They did not recommend additional steroid to inject MTrPs in order to avoid possible myotoxicity. Since the MTrP is not an inflammatory lesion, local corticosteroid may not provide any therapeutic effect. However, in our clinical practice, a small amount of corticosteroid added into the local anesthetic agent may prevent post-injection soreness in the back muscles, and never cause muscle damage. Botulinum toxin A can provide a pre-synaptic block of acetylcholine release in the motor endplates and subsequently relieve the taut band in the MTrP region. The efficacy of MTrP injection with botulinum toxin A to control myofascial pain has been documented in the literature. The suppressive effect of botulinum toxin A on EPNs recorded in the MTrP region has been demonstrated in an animal study. It has also been recently found that the prevalence of EPN in a MTrP region is proportionate to the intensity of MTrP pain in a human study. Fischer has recommended a technique of “pre-injection block” by infiltration with local anesthetic to the paraspinous sensory nerves supplying the area to be injected prior to giving a MTrP injection to the paraspinous muscles.

Acupuncture and other dry needling (without injection of any medication) have been applied in the control of MTrP pain. The similarity among dry needling, acupuncture, and MTrP injection has been documented. Melzack considered 80 percent of MTrPs as acupuncture points. Hong suggested the referred pain patterns of some MTrPs are similar to the acupuncture meridian. The importance of eliciting LTRs (similar to “De-Qui” or “The-Chi” effect in acupuncture) during needling has been emphasized for obtaining an immediate and complete pain relief. The mechanism of acupuncture or dry needling for pain control is still unclear. Hong hypothesized that the strong pressure stimulation to the MTrP loci can provide very strong neural impulses to the dorsal horn cells in the spinal cord to break the vicious cycle of the “MTrP circuit”, similar to hyper-stimulation analgesia. The techniques of dry needling include intramuscular stimulation (IMS) to a motor point, twitch-obtaining intramuscular stimulation, electrical twitch-obtaining intramuscular stimulation, and superficial dry needling. The acupuncture needle is too flexible and hard to handle, particularly for the lumbar paraspinal muscles. The use of EMG needle for paraspinal muscle needling would be more appropriate for patients with strong paraspinal muscle spasm. The application of needling at the acupuncture points of limbs is another good solution for such a problem. The commonly treated acupuncture points in the lower limbs for the control of myofascial LBP include MTrPs of piriformis, popliteus, tibialis anterior, peroneus
longus, gastrocnemius, and soleus muscles. More recently Chou et al have developed a new technique of acupuncture therapy. They used acupuncture needle to perform a procedure similar to MTrP dry needling with insertion into multiple sites in the MTrP region. They also applied the "fast-in and fast-out" technique. Since an acupuncture needle usually has a small diameter and is very flexible, they screwed (rotation and penetrating or withdrawing) the needle “fast-in and fast-out” to elicit LTRs and to avoid bending of the small-sized needle. This treatment is particularly beneficial to patients with fibromyalgia since the acupuncture needle (small diameter) can reduce focal tissue damage and decrease post-injection pain or discomfort that may last for many days in fibromyalgia patients.

Combination therapy consisting of various methods has been frequently applied to inactivate MTrPs. A clinician can make choice of any combination based on his best knowledge and clinical experience. The patient's preference should also be considered. However, it should be based on a founded scientific wisdom.

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肌筋膜背痛

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目的：本篇回顧文章描述肌筋膜引起背痛的病因、病理及臨床的特性和其治療。

發現：基於目前可得到的知識及臨床經驗，我們分析肌激痛點引起背痛的基本和臨床的觀點。大多數肌激痛點引起背痛的病例與受傷有關。許多潛在病因可使「隱性肌激痛點」活化成為「活性肌激痛點」，而造成肌筋膜背痛。因此，為了根除肌筋膜背痛且避免其再發，最重要是應找出並治療潛在病因。「活性肌激痛點」本身之有效治療(即去活化)包括徒手治療、物理治療和肌激痛點的針刺治療。

結論：查明引起肌筋膜背痛的病因並且提供適當的處理是重要的。（台灣復健醫誌 2008；36(1): 1 - 14）

關鍵字：肌筋膜疼痛(myofascial pain)，肌激痛點(myofascial trigger points)，背痛(low back pain)

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