Rehabilitation Practice and Science

Volume 36
Issue 1 *Taiwan Journal of Physical Medicine and Rehabilitation (TJPMR)*

Article 1

12-31-2008

Myofascial Low Back Pain

Mu-Jung Kao

Ta-Shen Kuan,

Yueh-Ling Hsieh

Jeng-Feng Yang

Chang-Zern Hong

Review Article

Myofascial Low Back Pain

Mu-Jung Kao,[1,2] Ta-Shen Kuan,[3] Yueh-Ling Hsieh,[4] Jeng-Feng Yang,[5] Chang-Zern Hong[4]

[1]Department of Physical Medicine and Rehabilitation, Taipei City Hospital, Taipei;
[2]Institute of Rehabilitation Science and Technology, Yang Ming University, Taipei;
[3]Departments of Physical Medicine and Rehabilitation, and [5]Physical Therapy, College of Medicine, National Cheng-Kung University, Tainan;
[4]Department of Physical Therapy, Hungkuang University, Taichung.

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 lesions can be identified. This unscientific approach should be clarified since the pathophysiology of myofascial trigger points (MTrP) 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-

Submitted date: 24 September 2007. Revised date: 18 December 2007. Accepted date: 28 December 2007.
Correspondence to: Dr. Chang-Zern Hong, Department of Physical Therapy, Hungkuang University, No. 34, Chung-Chie Road, Shalu, Taichung 433, Taiwan.
Tel：(04) 26321865 ext 3301 e-mail：johnczhong@yahoo.com

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 at a 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.[22] Therefore, the amount of MTrP loci in one MTrP region is proportionate to the irritability of that MTrP.[22,33]

Both referred pain and LTR are integrated in the spinal cord.[8] The term "myofascial trigger point circuit (MTrP circuit)" has been used to represent the interneuronal connections in the dorsal horn of spinal cord.[34,35] 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.[36]

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.[36] 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.[36] 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.[8] 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.

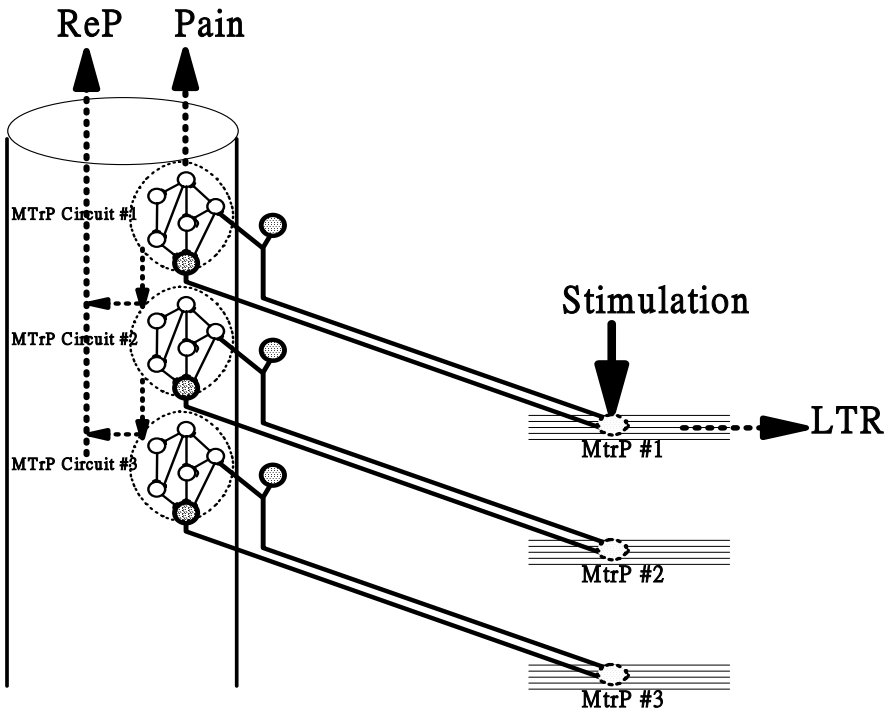

Figure 1. The "MTrP Circuit". MTrP = myofascial trigger point, ReP = referred pain, LTR = local twitch response

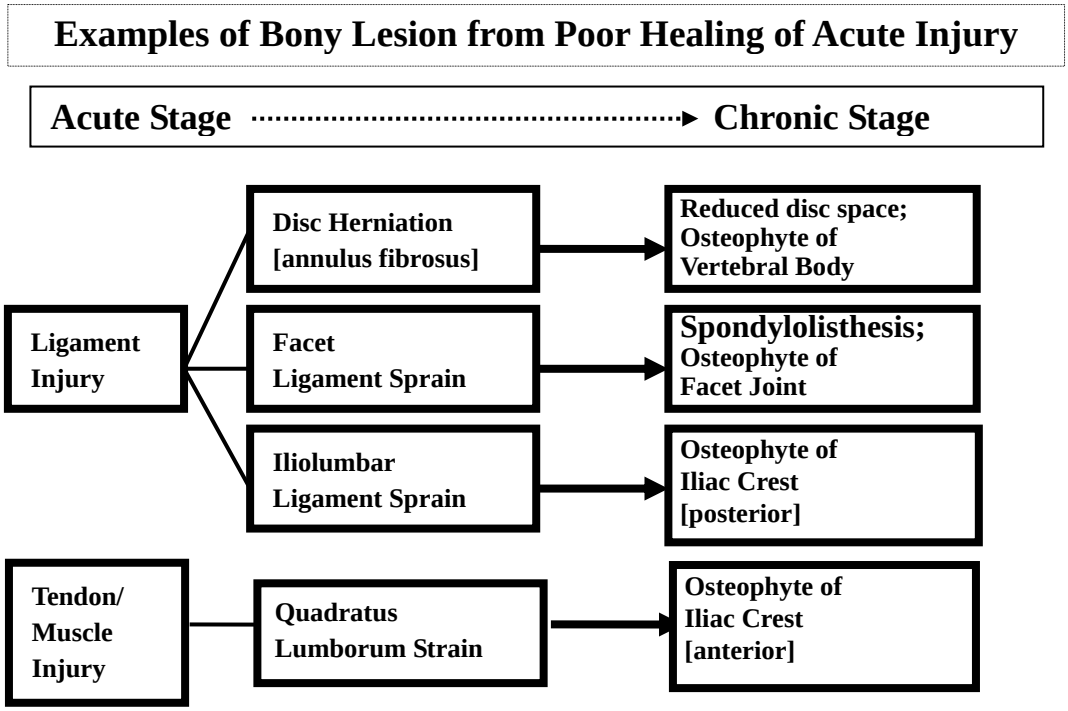

Figure 2. Abnormal radiological findings in chronic stage of soft tissue lesion

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.
3. Sprain of anterior longitudinal ligament –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.[9] 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.[9,23] 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.[21,34,35] 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.[37] Chronic LBP with a referred pain around the gluteal area is usually related to lumbar facet lesion.[3]

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.[38] Ultrasonography could be very useful for both the diagnosis and the assessment of spondylarthropathy activity.[39] 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.

TREATMENT OF MYOFASCIAL LOW BACK PAIN

Treatment of the Underlying Pathological Lesions to Avoid Recurrence of Trigger Point Pain

Myofascial pain can be suppressed easily by appropriate treatment, such as stretch, massage, or MTrP injection. However, it frequently recurs a few days or a few weeks later if the related pathological lesion is not eliminated.[21,34,35] When the underlying etiological lesion is completely eliminated, the active MTrPs can be inactivated permanently (unless re-injured). It was reported that the total number and pain intensity of MTrPs were significantly reduced after physical therapy or surgery for lumbar disc herniation.[40] In most cases, active MTrPs are activated from latent MTrPs secondary to neurological, muscular, or skeletal lesions. It less frequently occurs as a consequence of primary muscle lesion.[8,19,22] Activation of MTrPs can cause pain to avoid any movement that may interfere with the healing process of the primary lesion.[8,19] This is an important defense mechanism. Therefore, it is most important to identify the pathological lesion that causes the activation of MTrPs. The treatment of underlying pathology is the fundamental approach in the management of pain caused by MTrPs.

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.[41] Heat (thermotherapy) can cause vasodilatation and improvement of local circulation.[42] 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.[43,44] 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.[45] 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.[46,47] Manipulation may cause immediate pain relief by stretching the tight muscle or by sharp stimulation to the facet joint.[48] Intermittent pelvic traction may reduce the paraspinal muscle spasm quickly.[49] Exercise therapy is frequently prescribed for muscle strengthening, stretching,[50-52] increasing circulation, and relaxation. Additionally, back schools also reduce pain and improve function for patients with chronic and recurrent LBP.[47,53] 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.[1-3] 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- inflammation 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 to treat the posterior segment lesions, and epidural steroid injection, including caudal epidural injection, for the anterior segment lesions.

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.

Manual therapy for myofascial pain control has been well described by many researchers.[9,18,54,55] It is important to understand the basic pathological lesion of myofascial LBP and apply the manual technique appropriately and carefully, so that complications can be prevented. Frequently, the immediate effectiveness is obvious. However, the long-term effectiveness is still questionable. The most frequently used manual therapies include traditional spray and stretch, manipulation, mobilization, MTrP pressure release contraction-relaxation technique, deep pressure massage, acupressure, etc.[9] Chiropractic manipulation is one of the most popular types of manual therapy, but it will be discussed separately below.

Physical therapy modalities can be used for myofascial pain control. Thermotherapy is frequently used as an adjunct therapy both before and after any manual therapy. Although the heat is not very effective in myofascial pain control, it is the most important modality to treat the soft tissue lesion since it can improve local circulation to facilitate the healing process. It has been suggested to use therapeutic ultrasound for MTrP control since it may also provide mechanical energy directly to the MTrPs in addition to the thermal effects.[9] 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.[56] 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.[56-60] A study suggested that a combination of ultrasound and electrotherapy could provide better results than a single therapy.[59] 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.[61-64] However, its mechanism on MTrP therapy is still not established. Leinfort and Foley[65] 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[64] found an increase in skin resistance after laser therapy and suggested its sympathetically mediated effect.

Regarding therapeutic exercise, patients who have

clinical evidence of fibromyalgia syndrome should perform conditioning exercise.[66] There is an evidence that generalized conditioning exercise can activate the endogenous opioid system.[67]

Chiropractic manipulation has been one of the most popular techniques for pain control in United States of America.[68,69] Significant relief of MTrP pain after spinal manipulation therapy has been documented in the literature.[47,70,71] 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.[9,21,29] 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.[21,29] 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[9] 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.[72-75] The suppressive effect of botulinum toxin A on EPNs recorded in the MTrP region has been demonstrated in an animal study.[15] 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.[76] Fischer[77,78] has recommended a technique of "pre-injection block" by infiltration with local anesthetic to the paraspinal sensory nerves supplying the area to be injected prior to giving a MTrP injection to the paraspinal muscles.

Acupuncture and other dry needling (without injection of any medication) have been applied in the control of MTrP pain.[11,29,30,79,80] The similarity among dry needling, acupuncture, and MTrP injection has been documented.[11,19,81,82] Melzack[83] considered 80 percent of MTrPs as acupuncture points. Hong[19] 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.[11,19,21,29,30,79,81] The mechanism of acupuncture or dry needling for pain control is still unclear. Hong[34] 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.[34] The techniques of dry needling include intramuscular stimulation (IMS) to a motor point,[11] twitch-obtaining intramuscular stimulation,[79] electrical twitch-obtaining intramuscular stimulation,[81] and superficial dry needling.[82,84] 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[79] 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.[85,86] 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.[21,29] 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.

REFERENCES

1. Joines JD. Chronic low back pain: progress in therapy. Curr Pain Headache Rep 2006;10:421-5.
2. Koes BW, van Tulder MW, Thomas S. Diagnosis and treatment of low back pain. BMJ 2006;332:1430-4.
3. Mooney V. Facet syndrome. In: Weistein JN, Wiesel SW, editors. Philadelphia: W.B. Saunders Co; 1990. p.422-41.
4. Simons DG, Travell JG. Myofascial origins of low back pain. 1. Principles of diagnosis and treatment. Postgrad Med 1983;73(2):66, 68-73.
5. Simons DG, Travell JG. Myofascial origins of low back pain. 2. Torso muscles. Postgrad Med 1983;73(2):81-92.
6. Simons DG, Travell JG. Myofascial origins of low back pain, 3. Pelvic and lower extremity muscles. Postgrad Med 1983;73(2):99-108.
7. Hong CZ. Current research on myofascial trigger points: pathophysiological studies. J Musculoske Pain 1999;7(1/2): 121-9.
8. Hong CZ, Simons DG. Pathophysiologic and electrophysiologic mechanism of myofascial trigger points. Arch Phys Med Rehabil 1998;79:863-72.
9. Simons DG, Travell JG, Simons LS. Travell & Simons’s myofascial pain and dysfunction: the trigger point manual. Vol. 1, 2nd ed. Baltimore: Williams & Wilkins; 1999.
10. Lewit K. Management of muscular pain associated with articular dysfunction. In: Fricton JR, Awad EA, editors. Myofascial pain and fibromyalgia (advances in pain research and therapy), Vol 17, Chap. 21. New York: Raven Press; 1990, p.315-23.
11. Gunn CC. Gunn approach to the treatment of chronic pain: intramuscular stimulation for myofascial pain of radiculopathic origin. London: Churchill Livingston; 1996.
12. Kuan TS, Chang YC, Hong CZ. Distribution of active loci in rat skeletal muscle. J Musculoske Pain 1999;7(4): 45-54.
13. 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.
14. Simons DG, Hong CZ, Simons LS. Endplate potentials are common to midfiber myofascial trigger points. Am J Phys Med Rehabil 2002;81:212-22.
15. Kuan TS, Chen JT, Chen SM, et al. Effect of botulinum toxin on endplate noise in myofascial trigger spots of rabbit skeletal muscle. Am J Phys Med Rehabil 2002; 81:512-20.
16. Simons DG. Diagnostic criteria of myofascial pain caused by trigger points. J Musculoske Pain 1999;7(1/2): 111-20.
17. Simons DG. Do endplate noise and spikes arise from normal motor endplates? Am J Phys Med Rehabil 2001; 80:134-40.
18. Travell JG, Simons DG. Myofascial pain and dysfunction: the trigger point manual. Vol. 2. Baltimore: Williams & Wilkins; 1992.
19. Hong CZ. Myofascial trigger points: pathophysiology and correlation with acupuncture points. Acupunct Med 2000;18:41-7.
20. Kao MJ, Han TI, Kuan TS, et al. Myofascial trigger points in the early life. Arch Phys Med Rehabil 2007; 88:251-4.
21. Hong CZ. Consideration and recommendation of myofascial trigger point injection. J Musculoske Pain 1994; 2(1):29-59.
22. Hong CZ. Pathophysiology of myofascial trigger point. J Formos Med Assoc 1996;95:93-104.

23. Simons DG. Clinical and etiological update of myofascial pain from trigger points. J Musculoske Pain 1996; 4(1/2):93-121.
24. 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(3): 61-79.
25. Hong CZ, Kuan TS, Chen JT, et al. Referred pain elicited by palpation and by needling of myofascial trigger points: a comparison. Arch Phys Med Rehabil 1997;78:957-60.
26. Fricton JR, Auvinen MD, Dykstra D, et al. Myofascial pain syndrome: electromyographic changes associated with local twitch response. Arch Phys Med Rehabil 1985; 66:314-7.
27. 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.
28. 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.
29. Hong CZ. Lidocaine injection versus dry needling to myofascial trigger point: the importance of the local twitch response. Am J Phys Med Rehabil 1994;73:256-63.
30. Chu J. Dry needling (intramuscular stimulation) in myofascial pain related to lumbar radiculopathy. Eur J Phys Med Rehabil 1995;5:106-21.
31. Hong CZ, Chen JT, Chen SM, et al. Sensitive loci in a myofascial trigger point region are related to sensory nerve fibers. Am J Phys Med Rehabil 1997;76:172.
32. Gerwin RD, Simons DG, Pongratz D. Neure ergebnisse zur pathogeneses myofaszialer schmerzsyndrome. Nervenheilkunde 2002;21:35-7. (Full text in Ukrainian, abstract in English)
33. Hong CZ. Algometry in evaluation of trigger points and referred pain. J Musculoske Pain 1998;6(1):47-59.
34. Hong CZ. Myofascial pain therapy. J Musculoske Pain 2004;12(3/4):37-43.
35. Hong CZ. Treatment of myofascial pain syndrome. Curr Pain Headache Rep 2006;10:345-9.
36. Cailliet R. Soft tissue pain and disability. Philadelphia: F. A. Davis Company; 1977.
37. Saal JS. The role of inflammation in lumbar pain. Spine 1995;20:1821-7.
38. Gongal'skyi VV, Ambartsumov RM, Kopyl IeG. Accelerated development of spinal degenerative changes during low back pain in young people. Lik Sprava 2001;(5-6): 98-101. (Full text in Ukrainian, abstract in English)
39. D'Agostino MA, Said-Nahal R, Hacquard-Bouder C, et al. Assessment of peripheral enthesitis in the spondylarthropathies by ultrasonography combined with power Doppler: a cross-sectional study. Arthritis Rheum 2003; 48:523-33.
40. Wu CM, Chen HH, Hong CZ. Myofascial trigger points in patients with lumbar radiculopathy due to disc herniation before and after surgery. J Surg Assoc ROC 1997; 30:175-84.
41. Rakel B, Barr JO. Physical modalities in chronic pain management. Nurs Clin North Am 2003;38:477-94.
42. Weber D, Brown A. Physical agent modalities. In: Braddom R, editor. Physical medicine and rehabilitation. 2nd ed. Philadelphia: W B Saunders; 2000. p. 440-58.
43. Kellogg DL Jr. In vivo mechanisms of cutaneous vasodilation and vasoconstriction in humans during thermoregulatory challenges. J Appl Physiol 2006;100:1709-18.
44. Davison JL, Short DS, Wilson TE. Effect of local heating and vasodilation on the cutaneous venoarteriolar response. Clin Auton Res 2004;14:385-90.
45. Geffen SJ. Rehabilitation principles for treating chronic musculoskeletal injuries. Med J Aust 2003;178:238-42.
46. Furlan AD, Brosseau L, Imamura M, et al. Massage for low back pain (Cochrane Review). In: The Cochrane Library, Issue 4, 2002. Oxford: Update Software.
47. Hsieh CY, Adams AH, Tobis J, et al. Effectiveness of four conservative treatments for subacute low back pain: a randomized clinical trial. Spine 2002;1;27:1142-8.
48. Powell FC, Hanigan WC, Olivero WC. A risk/benefit analysis of spinal manipulation therapy for relief of lumbar or cervical pain. Neurosurgery 1993;33:73-8.
49. Sari H, Akarirmak U, Karacan I, et al. Computed tomographic evaluation of lumbar spinal structures during traction. Physiother Theory Pract 2005;21:3-11.
50. Khalil TM, Asfour SS, Martinez LM, et al. Stretching in the rehabilitation of low-back pain patients. Spine 1992; 17:311-7.
51. Nielsen AJ. Spray and stretch for myofascial pain. Phys Ther 1978;58:567-9.
52. Rubin D. Myofascial trigger point syndromes: an approach to management. Arch Phys Med Rehabil 1981;

62:107-10.

53. Heymans MW, van Tulder MW, Esmail R, et al. Back schools for nonspecific low back pain: a systematic review within the framework of the Cochrane Collaboration Back Review Group. Spine 2005;30:2153-63.
54. Kostopoulos D, Rizopoulos K. The manual of trigger point and myofascial therapy. New Jersey: Slack Incorporated; 2001.
55. Kraft GH, Stanton DF, Mein EA. Manual Medicine. Phys Med Rehabil Clin N Am 1996;7: 679-932.
56. Hsueh TC, Cheng PT, Kuan TS, et al. The immediate effectiveness of electrical nerve stimulation and electrical muscle stimulation on myofascial trigger points. Am J Phys Med Rehabil 1997;76:471-6.
57. Graff-Radford SB, Reeves JL, Baker RL, et al. Effects of transcutaneous electrical nerve stimulation on myofascial pain and trigger point sensitivity. Pain 1989;37:1-5.
58. Hou CR, Tsai LC, Cheng KF, et al. Immediate effects of various physical therapeutic modalities on cervical myofascial pain and trigger point sensitivity. Arch Phys Med Rehabil 2002;83:1406-14.
59. Lee JC, Lin DT, Hong CZ. The effectiveness of simultaneous thermotherapy with ultrasound and electrotherapy with combined AC and DC current on the immediate pain relief of myofascial trigger point. J Musculoske Pain 1997;5(1):81-90.
60. Tanrikut A, Özaras N, Kaptan HA, et al. High voltage galvanic stimulation in myofascial pain syndrome. J Musculoske Pain 2003;11(2):11-5.
61. Hakguder A, Birtane M, Gurcan S, et al. Efficacy of low level laser therapy in myofascial pain syndrome: an algometric and thermographic evaluation. Lasers Surg Med 2003;33:339-43.
62. Ilbuldu E, Cakmak A, Disci R, et al. Comparison of laser, dry needling, and placebo laser treatments in myofascial pain syndrome. Photomed Laser Surg 2004;22:306-11.
63. Lin CP, Chen SM, Chen JT, et al. Therapeutic effectiveness of low-level laser on myofascial trigger points. J Phys Ther Assoc ROC 2000;25:15-26.
64. Snyder-Mackler L, Barry AJ, Perkins AI, et al. Effects of helium-neon laser irradiation on skin resistance and pain in patients with trigger points in the neck or back. Phys Ther 1989;69:336-41.
65. Kleinkort JA, Foley RA. Laser Acupuncture: its use in physical therapy. Am J Acupuncture 1984;12:51-6.
66. McCain GA, Bell DA, Mai FM, et al. A controlled study of the effects of a supervised cardiovascular fitness training program on the manifestations of primary fibromyalgia. Arthritis Rheum 1988;31:1135-41.
67. Howlett TA. Hormonal responses to exercise and training: a short review. Clin Endocrinol 1987;26:723-42.
68. Assendelft WJ, Morton SC, Yu EI, et al. Spinal manipulative therapy for low back pain. A meta-analysis of effectiveness relative to other therapies. Ann Intern Med 2003;138:871-81.
69. Meeker WC, Haldeman S. Chiropractic: a profession at the crossroads of mainstream and alternative medicine. Ann Intern Med 2002;136:216-27.
70. Hsieh CY, Hong CZ. Effect of chiropractic manipulation on the pain threshold of myofascial trigger point. Proceedings of the 1990 International Conference of Spinal Manipulation. Los Angeles: Los Angeles College of Chiropractic; 1990. p.359-63.
71. Lewit K. Manipulative therapy in rehabilitation of the locomotor system. 2nd ed. Oxford: Butterworth Heinemann; 1991.
72. Acquadro MA, Borodic GE. Treatment of myofascial pain with botulinum A toxin. Anesthesiology 1994;80: 705-6.
73. Cheshire WP, Abashian SW, Mann JD. Botulinum toxin in the treatment of myofascial pain syndrome. Pain 1994; 59:65-9.
74. Yue SK. Initial experience in the use of botulinum toxin A for the treatment of myofascial related muscle dysfunctions. J Musculoske Pain 1995;3 (Supp 1):22.
75. Wheeler AH, Goolkasian P, Gretz SS. A randomized, double-blind, prospective pilot study of Botulinum toxin injection for refractory, unilateral, cervicothoracic, paraspinal, myofascial pain syndrome. Spine 1998;23:1662-7.
76. Kuan TS, Hsieh YL, Chen SM, et al. The myofascial trigger point region: correlation between the degree of irritability and the prevalence of endplate noise. Am J Phys Med Rehabil 207;86:183-9.
77. Fischer AA. Injection techniques in the management of local pain. J Back Musculoskel Rehabil 1996;7:107-17.
78. Fischer AA. Treatment of myofascial pain. J Musculoske Pain 1999;7(1/2):131-42.
79. Chu J. Twitch-obtaining intramuscular stimulation: observation in the management of radiculopathic chronic low

back pain. J Musculoske Pain 1999;7(4):131-46.

80. Lewit K. The needle effect in relief of myofascial pain. Pain 1979;6:83-90.
81. Chu J, Neuhauser DV, Schwartz I, et al. The efficacy of automated/electrical twitch-obtaining intramuscular stimulation (ATOIMS/ETOIMS) for chronic pain control: evaluation with statistical process control methods. Electromyogr Clin Neurophysiol 2002;42:393-401.
82. Baldry P. Superficial dry needling in the treatment of fibromyalgia and myofascial syndrome. In: Chaitow CL, editor. Fibromyalgia syndrome: a practitioner's guide to treatment. Edinburgh: Churchill Livingston; 2000.
83. Melzack R. Myofascial trigger points: relation to acupuncture and mechanism of pain. Arch Phys Med Rehabil 1981;62:114-7.
84. Goddard G, Karibe H, McNeill C, et al. Acupuncture and sham acupuncture reduce muscle pain in the myofascial pain patients. Orofac Pain 2002;16:71-6.
85. Hong CZ. New advance in management of myofascial pain syndrome. The proceedings of the 4th world congress of Internation Society of Physical and Rehabilitation Medicine; 2007; Korea. p.231-6.
86. Chou LW, Hong J, Hong CZ. A new technique for acupuncture therapy and its effectiveness in treating fibromyalgia syndrome: a case report. J Musculoske Pain (in press).

肌筋膜背痛

高木榮[1,2]　官大紳[3]　謝悅齡[4]　楊政峰[5]　洪章仁[4]

台北市立聯合醫院復健科[1]　陽明大學復健科技輔具研究所[2]
國立成功大學醫學院復健學科[3]　弘光科技大學物理治療學系[4]
國立成功大學醫學院物理治療學系[5]

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和臨床經驗，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」活化成爲「活性肌激痛點」，而造成肌筋膜背痛。因此，爲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)

通訊作者：洪章仁醫師，弘光科技大學物理治療系，台中縣 433 沙鹿鎮中棲路 34 號
電話：(04) 26321865 轉 3301　　e-mail：johnczhong@yahoo.com