Table 1 **Pressure pain thresholds (mean±SD, arbitrary units) in and around the palpable bands**

Location	centre	distal	proximal	medial	lateral
Second day	296±32**	534±61	561±70	839±148	766±112
Seventh day	944±128	982±118	973±136	1057±175	1345±118

** Significantly different from all four other values (Tukey, P<0.01)

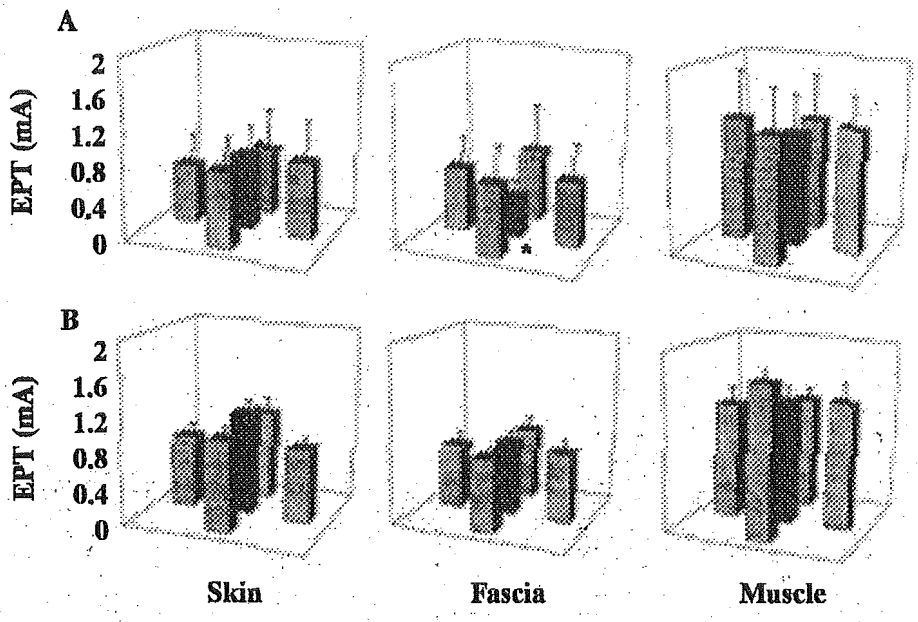

Figure 3 This figure shows the distribution of electrical pain thresholds (EPTs) of the different tissues. (A) shows the readings taken on the second day, and (B) shows the readings taken on the seventh day. The location of measuring sites were the same as in Fig 2. The asterisk shows significant difference from other four sites (nonparametric Tukey's multiple test, P<0.05).

Table 2 **Electrical pain thresholds (mean±SD mA) in sites at and 10mm away from the palpable band on the second day after exercise**

Location	palpable band	distal	proximal	medial	lateral
Skin	0.84±0.11	0.65±0.11	0.77±0.19	0.85±0.18	0.84±0.15
Fascia	0.45±0.08**	0.71±0.11	0.82±0.21	0.73±0.15	0.79±0.15
Muscle	1.27±0.14	1.39±0.21	1.29±0.21	1.29±0.21	1.41±0.18

** Significantly lower than values for other sites in fascia (Tukey's multiple test, P=0.01)

points was detected on the second day (Tukey, P<0.01). There were no differences on the seventh day. The distribution of PPT before exercise was not examined because the precise location of the palpable band could not be predicted.

Changes in Electrical Pain Thresholds

Figure 3 shows the EPT of the skin, fascia and muscle, on the second day (A), and on the seventh day (B), at the tender locus on the palpable band, and four surrounding sites. The values are given in Table 2, which indicates that there was a significantly lower EPT for the fascia only, at the tender locus on the second day (Tukey's multiple test, P=0.01). There was no difference on the seventh day. There was no significant difference among the five EPTs of the skin and muscle.

The sensations elicited by the current pulse stimulation apparently varied with the tissues stimulated. In the skin, volunteers reported

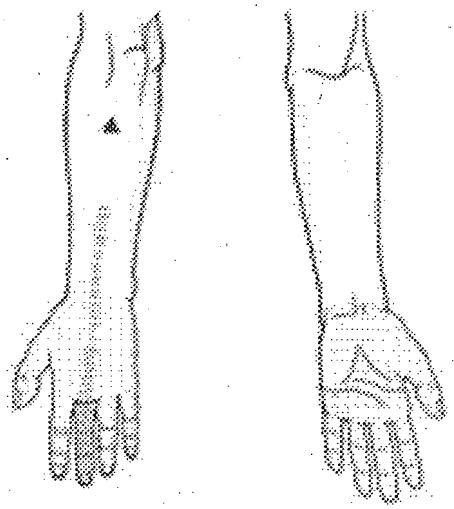

Figure 4 This is a schematic illustration of the pattern of referred pain from the palpable band. Referred pain was elicited by mechanical stimulation of the most localised tender region (▲). More common areas of referred pain are represented by denser shading. Note that the dorsal part of the middle finger is the most frequent area of the pain referral.

localised sharp pain and sometimes burning. In the fascia, dull and uncomfortable pain tended to be reported, and in the muscle, pulling pain.

Development of palpable band

Immediately after the eccentric exercise, the working muscle tended to be swollen and became hard to touch. On the second day after exercise, a clear ropy palpable band could be detected at the musculotendinous attachment area of the working muscle. The band was detected in all 15 exercised subjects by a well-trained observer. In several cases, the observer was asked to detect which of the two arms contained the palpable band in a blinded manner, and in all cases he detected the band correctly. The mean width and length of the palpable band on the second day were 4.4±0.6mm and 33.0±1.9mm respectively. The existence of a taut band was also confirmed during the manual needle insertion for measuring the electrical pain threshold. Resistance was increased when the needle penetrated the palpable band. On the seventh day after exercise, the bands had softened and become diffuse, and could hardly be identified.

Induction of referred pain

Application of an intense pressure to the tender locus usually induced various pain sensations projected over the distal area accompanied by dull and uncomfortable sensations. These referred pain sensations were felt most intensely in the hand, and formed a line that extended along the dorsum of the forearm, wrist, hand, and the middle finger, as shown schematically in Figure 4. The tender locus of the palpable band had the lowest threshold for inducing referred pain.

During the measurement of EPT, referred pain was also frequently reported when the stimulating needle electrode was inserted into the palpable band. The fascia at the tender focus most readily induced referred pain, and stimulation of the skin and muscle hardly provoked the referred pain.

Needle EMG activity at the tender region

In general, no spontaneous EMG activity was recorded from the relaxed muscle of the subjects. In the control session, we could not detect any sustained EMG activity from the fascia or muscle, only a transient injury burst response. On the other hand, sustained unitary EMG activity was frequently recorded when the recording needle was situated close to the fascia at the tender locus on the palpable band. Usually the discharges continued for 30 seconds with regular frequency (1-60Hz), but in one case they continued for more than 10 minutes. Table 3 shows that sustained EMG activity was only recorded from the tender region on the palpable band.

Table 3 **Incidence of the presence EMG (+), or absence EMG (-), of sustained EMG activity recorded at different electrode positions**

Position of electrode	EMG(+)	EMG(-)	total
Tender region on the palpable band	7	1	8
Non-tender region on the palpable band	0	8	8
Non-tender region beside the palpable band	0	8	8

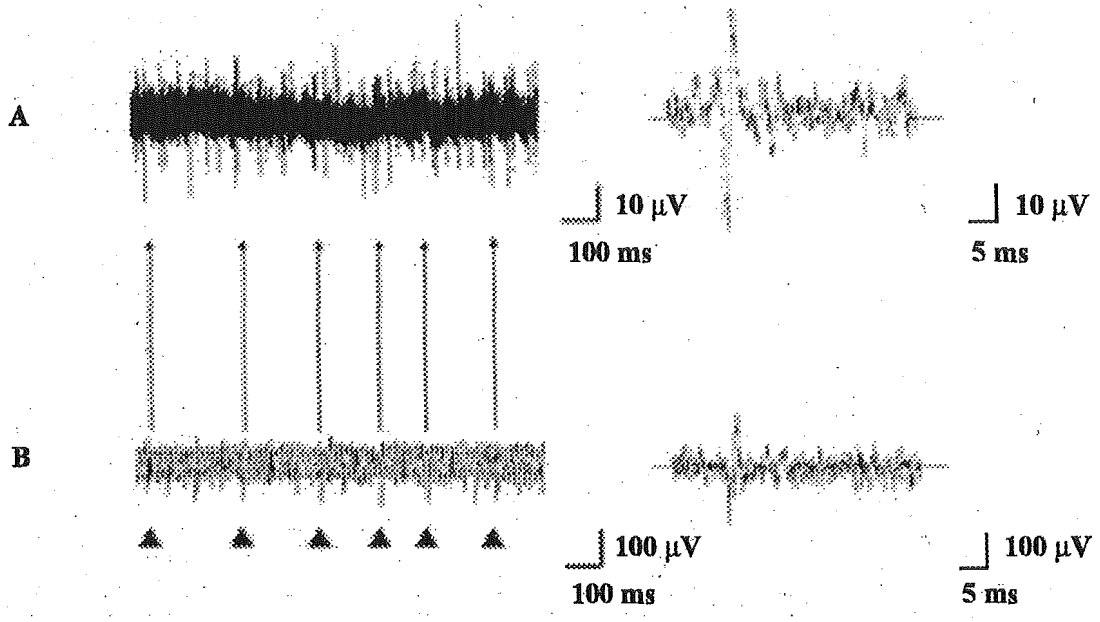

Figure 5 This figure shows simultaneous recordings of sustained EMG activity with the needle and surface electrodes. The needle electrode was placed at the palpable band and surface electrode 50mm away from the needling site. A shows the needle EMGs and B shows the surface EMG recordings (time scales are different in the right and left figures). Solid triangles mark spike activity in the surface EMG, and arrows indicate the spike discharges of needle EMGs. The synchronous appearance of the spike activities should be noted.

Figure 5 demonstrates an example of EMGs recorded from an insulated needle electrode at the palpable band (A) and from a surface electrode on the skin 50mm distal to the needle electrode over the same muscle (B). Synchronized unitary discharges were observed at the two recording sites simultaneously, and also disappeared at the same time.

When EMG activity was recorded, the subjects frequently reported strong dull pain sensation which subjectively appeared to be associated with the frequency of the EMG activities.

Discussion

Repetitive eccentric exercise produced a localised tender region with a palpable ropy band on the second day, with decreased pain threshold of the fascia at the locus. Intense pressure applied to the tender locus on the palpable band also elicited referred pain. Sustained electrical activity was frequently recorded from the palpable band, and was accompanied by a deep, dull pain sensation.

The experimental model of localised tender region

Eccentric contraction is known to produce tissue injury more easily than concentric contraction,[16] because muscle can bear a greater load under eccentric contraction, and therefore the evoked tension of a single muscle fibre is greater.[17]

The majority of previous studies on experimental DOMS have reported only muscle soreness,[18,19] or have given a rough illustration of the distribution of tenderness on the muscle.[20] No previous reports have described a palpable band or a localised tender region, as we describe. Our findings may be due to the size of the muscle studied, and the methods of examination and pain measurement. Previously, relatively large muscles such as the quadriceps femoris or biceps brachialis have been used, whereas we exercised a small forearm muscle and then carefully palpated in order to find the most tender region along the length of a taut band. We identified a reduction in the PPT which was greatest at the tender region in the palpable band, and the EPT of the fascia at that site was selectively decreased. The distribution of

the EPTs in the fascia was similar to that of the PPTs, so the tender structure detected by the pressure algometer was presumably the fascia. This is the first report of a localised tender region on a rope-like taut band appearing after eccentric exercise and of a selective decrease in the pain threshold at the fascia of the extensor digitorum muscle.

The mechanisms of localised tenderness

The facts that the tender region was not spontaneously painful and that pain was evoked only when light pressure was applied suggest that the sensitisation of nociceptors in the damaged tissue might be a possible cause of the restricted tenderness. The time-course of development of DOMS has been shown to be similar to that of inflammatory processes,[21-23] and the production of various inflammatory mediators such as prostaglandin E_2 (PGE_2), bradykinin and histamine in the damaged tissues has been reported.[21] Recently, we found that the application of indomethacin, a prostaglandin synthesis inhibitor, suppressed the development of DOMS in rabbits.[24] An increase of prostaglandin E_2 in the muscle of the DOMS subject has also been demonstrated by microdialysis.[25] These mediators sensitise the nociceptors and may activate silent nociceptors in the muscle.[26-28]

The polymodal-type receptor is proposed as a possible candidate for the formation of the tender point.[29] It is responsive to mechanical (acupuncture), thermal (moxibustion), and chemical stimulation (various chemical substances are produced by acupuncture and moxibustion). Excitation of the polymodal-type receptors produces flare and oedema (neurogenic inflammation), and such phenomena are frequently observed after acupuncture and moxibustion. The sensation elicited by selective activation of the polymodal-type receptors is similar to the *de qi* sensation, and sensitisation of the polymodal-type receptors may be a possible cause of tender points and myofascial trigger points.[29]

Central sensitisation should be considered as another possible explanation for the tenderness. A decrease in the threshold of nociceptive dorsal horn neurons in acute inflammation is well known.[30] Expansion of the receptive field and appearance of a new receptive field were induced by intramuscular injection of chemical algesics.[31] These phenomena, however, do not seem to be the major cause of the formation of localised tender regions. In the present study we found that the tender region on the palpable band was highly restricted to a spot-like area less than 2mm in diameter and located on the fascia. These very localised changes in the electrical pain threshold of a particular tissue are difficult to attribute to the effects of central sensitising mechanisms as they are currently understood.

Formation of the palpable band

In the present study, a ropy palpable band was detected on the second day after the exercise, and disappeared, or was hardly detectable, by the seventh day. Clinically, the development of the palpable band is considered to be a characteristic feature of a myofascial trigger point and is readily detected by well-trained examiners.[32] Fricton et al found that experienced raters were more reliable in detecting the trigger point than inexperienced raters.[5] It should be noted that the training in palpation is important.

The palpable band was initially regarded as a localised muscle contracture induced by the increase of intracellular calcium ion within the muscle cell,[33] then the idea developed into the more sophisticated energy crisis theory.[1;34] This theory postulates an initial release of intracellular calcium from the sarcoplasmic reticulum or inflow from the extracellular fluid through the injured membrane. The increase of intracellular calcium ions causes sustained contraction of the muscle, which may inhibit the local circulation and thus induce a shortage of oxygen and ATP in the tissues. The lack of energy may inhibit calcium re-uptake into the sarcoplasmic reticulum by the calcium pump, which would perpetuate the vicious cycle. While the increased excitability of the motor neuron is undoubtedly a possible mechanism for the increase in muscle tension and rigidity, the lack of EMG activity from the palpable band has been well established,[33] and it seems that muscle contraction is unlikely to be the cause of the palpable band. Localised oedema developing in deep tissues was proposed as another possibility.[29] In the present model,

eccentric exercise undoubtedly produced muscular tissue injury. When the localised muscle fibres are damaged, causing an accumulation of extracellular water and an increase in intra-tissue pressure, this may be detected as a taut band.

Referred pain phenomena, electrical activity and local twitch response

It has been well established that intramuscular injection of hypertonic saline induces particular referred pain patterns depending on the sites of injection.[35] This phenomenon has been well established in recent studies with human subjects.[36;37] Our data clearly demonstrate that the localised tender region is the most sensitive to elicit at the localised tender region and that needle insertion at the fascia also frequently provokes referred pain.

Recently, the participation of spinal neurons in referred pain was demonstrated in anaesthetised rats,[30;31] as an intramuscular injection of bradykinin induced a new receptive field of nociceptive dorsal horn neurons. This suggests possible mechanisms of referred pain phenomena from the muscle.

Lack of electrical activity in the palpable band has long been commonly accepted, and various mechanisms were proposed based on that observation. Recently, however, spontaneous electrical activity (SEA) has been recorded from myofascial trigger points in human subjects.[9;38] The cause of the SEA has not been clarified yet. This SEA was not considered to be the result of spastic hyperexcitability of motor neurons because it was recorded from very restricted sites in the muscle. Simons pointed out the similarity of the SEA to the end-plate potentials in clinical electromyogram studies.[6;39] On the other hand, Hubbard argued that the SEA was activity of the intrafusal muscle of the muscle spindle elicited by the sympathetic nerve.[9]

In the present study we could record the sustained EMG activity at the restricted tender region on the palpable band, and the EMG activity was accompanied by strong deep pain sensation. In other words, the electrical activity seemed to be the result of the insertion of a recording electrode into the tender region which produced deep pain sensation. The fact that the synchronised unitary EMG discharges were recorded simultaneously from the surface electrode and needle electrode (Fig 4) strongly suggests that the recorded sustained EMG activity was the reflex activity evoked by the nociceptive inputs from the fascia. Our recent study demonstrated that sustained EMG activity recorded from the eccentrically exercised rabbit palpable band was clearly suppressed by acupuncture needling to the muscle 50mm away from the band.[40] Although we could not exclude the possibility of the endplate potential, the majority of the sustained EMG activity in the present study might be the reflex activities elicited by nociceptive inputs produced by the needle insertion to the tender region of palpable band.

The local twitch response (LTR) elicited by palpation of the taut band is a useful indicator of a myofascial trigger point, and the LTR was considered to be a spinal reflex elicited by the noxious inputs from the trigger point.[38;41] In our study, similar LTR was frequently observed during the insertion of the needle electrode. Thus, the features of the restricted tender region in our study and those of the trigger point are quite similar. Therefore, some trigger points may be explained as the result of DOMS-like phenomena.

The relationships between myofascial trigger point and acupuncture point

The present experimental model was based on the similarity of myofascial trigger points and acupuncture points. The characteristics of the trigger point such as localised tender points on the palpable band and a typical referred pain and local twitch response are also clinically useful indicators of the acupuncture points. Melzack et al found that the location of acupuncture points are coincident with trigger points in 100% of cases, allowing for a difference of 3cm. Typical referred pain patterns of the trigger point also resemble the meridian patterns. Moreover, sustained electrical activity was frequently recorded from trigger points and acupuncture points.[10;42]

The present model may be useful for further investigation of myofascial trigger points and certain acupuncture points.

Acknowledgement

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Editorial comment

Adrian White
editor in chief

Mike Cummings
editor

editor@medical-acupuncture.org.uk

This paper's passage from submission to publication has presented the editors with a dilemma: whether to continue remorselessly in a reiterative process of review, dispute and revision over several months, or whether to publish the paper and let it stimulate. The findings themselves have been clearly described in the paper, and we are left with differences of opinion that cannot easily be resolved without doing more studies. Therefore, out of respect to the authors' standing in the world of acupuncture physiology, and to the potential importance of this claimed model of the myofascial trigger point, we decided to publish the paper after two sets of revisions; and, out of respect and gratitude to our reviewers (we used four altogether, three of whom specialise in this area of research) we want to summarise the limitations of the paper that they have pointed out, and other possible reasons for caution in accepting this model in its present form and at the present time.

One set of limitations concerns the likelihood that the changes seen in delayed onset muscle soreness (DOMS) are really similar enough to trigger points to provide a meaningful model. DOMS usually affects whole muscles and a number of tender areas can be found. The whole muscle may develop the characteristics which could appear to be the 'taut band' of a myofascial trigger point, but some reviewers questioned whether a taut band in the middle finger portion of the extensor digitorum could be differentiated by palpation from the whole muscle. They commented that the damage in DOMS affects the entirety of the muscle, and therefore is significantly different from the focal symptoms associated with trigger points. They suggested that it was plausible that the 'palpable band' that the authors report in their paper might be a swollen entire muscle in the forearm, which becomes hard because the fascia which envelops it does not allow the oedema to dissipate. The tender sites in DOMS have not been shown previously to refer pain [which does not mean they cannot, of course]. The clinical picture of DOMS is different from that of trigger points: DOMS settles in a week or so, and actually confers some protection against further episodes of DOMS. Trigger points, in contrast, tend to persist at least in latent form and are more likely to be made worse by further insults such as over-exercise. We are in the land of uncertainty since, nobody knows the pathophysiological mechanisms of DOMS or trigger points. An associated problem is an ethical one about using this model in volunteers: the pain of DOMS is difficult to relieve, which may be an acceptable price to pay for a volunteer running a marathon, but hardly fair on those who help trigger point research!

Another area of doubt is how to interpret the various features of the electrical activity reported here. Firstly, the authors report that they recorded the maximal electrical activity from the fascia. It has to be said that the basis for claiming the needle was exactly at the fascia can be challenged – the ultrasound examination was not simultaneous, so the judgement probably relied largely on the 'feel' of the needle-tip, which is subjective. Secondly, the claim that sustained needle EMG activity arose in the fascia needs further explanation and exploration. Fascia seems unlikely to be the source of action potentials, nor is it likely to be a source of other spontaneous electrical activity, though it might *conduct* action potentials from remote muscles (which could be identified by further studies recording simultaneously from intramuscular needles). One reviewer doubted whether the

recordings made simultaneously from the skin could be interpreted correctly without fully analysing their characteristics, particularly the time-course (which would differ from direct recordings, since it is conducted through tissues).

We know that many significant scientific discoveries are greeted by disbelief. Progress relies on the exchange of ideas that provoke others to comment and criticise, and to conduct more experimental studies. We therefore publish this article in the hope of stimulating just such a response.

筋・筋膜性疼痛

川喜田健司
明治鍼灸大学生理学教室

要　旨
筋・筋膜性疼痛の特徴が，トリガーポイントに起因する関連痛であることの再確認とともに，その診断に用いられる索状硬結，圧痛点，関連痛，局所単収縮反応，筋の短縮，筋力低下，自律神経反応などの指標とトリガーポイント発現の機序との関連について概説した．またトリガーポイント不活性化のための多様な治療法を紹介する中で，質の高い臨床試験が求められている現状を指摘し，トリガーポイント注射に関して薬物注射群と生理食塩水群や鍼治療群との間に効果の差がないこと，およびトリガーポイントとツボとの密接な関連性にも言及した．さらに線維性筋痛に関する研究の現状を概説し，筋・筋膜性疼痛との密接な関連性が指摘されている点を併せて紹介した．

(ペインクリニック 25：1024-1031，2004)
キーワード：トリガーポイント，関連痛，索状硬結

はじめに

筋・筋膜性疼痛（myofascial pain syndrome：MPS）は，その名称から痛みの原因が筋や筋膜に存在していると考えがちである．しかし，臨床的に慢性的な筋痛を訴えるMPS患者の症状は，患者が痛みを訴える部位とは離れた所に生じたトリガーポイントの活性化によって引き起こされた関連痛であることを十分に理解する必要がある．またMPSの治療には，その症状をもたらしているすべてのトリガーポイントを正確に検出し，そのすべてを不活性化することが必要である．その不活性化法として，各種の薬物を用いたトリガーポイント注射や鍼灸鍼を用いた機械的刺激，冷却スプレーとストレッチの組み合わせや虚血性圧迫法などが知られている．

このような多様な方法がトリガーポイントを不活性化させる機序については，まだ数多くの仮説が提唱されている段階である．そこで本稿では，トリガーポイントの検出法とその不活性化法，およびその成因に関する諸説について概説する．また，近年注目を浴びるようになった線維性筋痛（fibromyalgia：FM）についても，その病態とMPSとの関連について紹介する．

1．筋・筋膜性疼痛の診断基準

MPS患者では血液所見や痛みを訴える部位の触診やX線像にも異常が認められない．そのような筋痛患者において，トリガーポイントを正確に検出することが重要である．表1にその診断に関連した指標をまとめた[1]．以下にその具体的な方法や問題点について列挙する．

〈Special Article〉 Musculoskeletal pain
Myofascial pain : diagnosis, treatment and pathophysiological mechanisms
Kenji Kawakita
Department of Physiology, Meiji University of Oriental Medicine

表1　筋・筋膜性疼痛の診断に用いるトリガーポイント検出の指標とその診断上の価値と難易度

検査項目	診断上の価値	困難さ
圧痛	＊＊	＊
ジャンプ・サイン	＊	＊＊
痛みの再現	＊＊	＊＊
索状硬結	＊＊＊	＊＊＊
関連痛	＊	＊＊＊
単収縮反応（LTR）	＊＊＊＊	＊＊＊＊

トリガーポイント診断に関連する指標．＊の数が多いほど診断価値が高く，技術的に困難としている．この表ではSimonsの統合仮説に基づきLTRの診断上の意義を強調しているが，多くの臨床家は患者の痛みや症状がトリガーポイントの圧迫で出現することをより重視している（文献1より引用）

1）索状硬結と局在する圧痛点

トリガーポイントを見つけるためには，注意深い触診によって筋にある索状硬結を探し当て，次にその索状硬結の中に特に敏感な圧痛部位を見出すことである．これがいわゆるトリガーポイントであるが，その数は必ずしも特定の筋に1つではなく，複数筋にそれぞれ多数のトリガーポイントが存在することもある．

この索状硬結の成因として，Travellらは筋の部分損傷による筋小胞体からのカルシウムイオンによる筋拘縮説を提唱した[2]．しかし，拘縮した筋に電気活動はみられないにもかかわらず，Hubbardらはトリガーポイントから電気活動を記録し，それが交感神経の支配する筋紡錘の錘内筋の活動電位とした[3]．そこでSimonsらはこれまでの筋拘縮説に代えて，図1に示す運動終板機能の異常亢進と局所拘縮説を加味した統合説を提唱した[4]．その論拠には，トリガーポイントから記録される電気活動が終板電位に類似していること[4]，アセチルコリンの過剰分泌状態を実験的に作ると運動終板の近傍にcontraction knot様の部位が出現すること[5]，また，運動終板部位には無髄神経の分布が密であり痛覚閾値も周囲の筋組織に比べて低いこと[6]などを挙げた．

しかし，この運動終板説は，トリガーポイントが筋や筋膜以外の組織，皮膚，腱，靭帯，骨膜等にも出現するためにMPS様の症状をもたらすという事実を説明できない．

われわれはポリモーダル受容器の感作をトリガーポイントの成因とし，筋拘縮とともに深部組織の浮腫が索状硬結である可能性を挙げている．その詳細やそれ以外の諸説の問題点については別稿を参照されたい[7]．

2）患者の疼痛再現と特徴的な関連痛パターン

MPSの特徴は，患者の訴える疼痛症状がトリガーポイントを原因とする関連痛という点にある．そこで症状の原因となっているトリガーポイントを見つけて圧迫すると，その患者が訴えてきたものと全く同じ症状が再現されることがある．この患者の症状の再現や特定パターンの関連痛の発現は，トリガーポイントの診断上の重要な指標の一つである．特定筋から生じる固有の関連痛パターンは既にTravellやBaldryら[2,8]によって詳しく記載されているので，そのパターンを参考にして患者の訴えに関連するトリガーポイントの存在する筋を予測することが可能である．

この関連痛現象を実験的に明らかにしたのはKellgren[9]である．ヒトの筋に高張食塩水を微量注入すると，注入局所以外の遠隔部位にその筋特有の関連痛パターンが現れる．このような特定パターンを示す関連痛は，筋に限らず，腱，靭帯，骨膜および皮膚の刺激によっても生じる[10]．このような関連痛のメカニズムとして広く知られているのは，脊髄レベルでのニューロンの収束・投射説や収束・促通説であるが，実際にヒトで起こっている感覚現象を十分に説明できるものではない．

3）局所単収縮反応

索状硬結を指で弾くことで局所単収縮反応（local twitch response：LTR）と呼ばれる反応

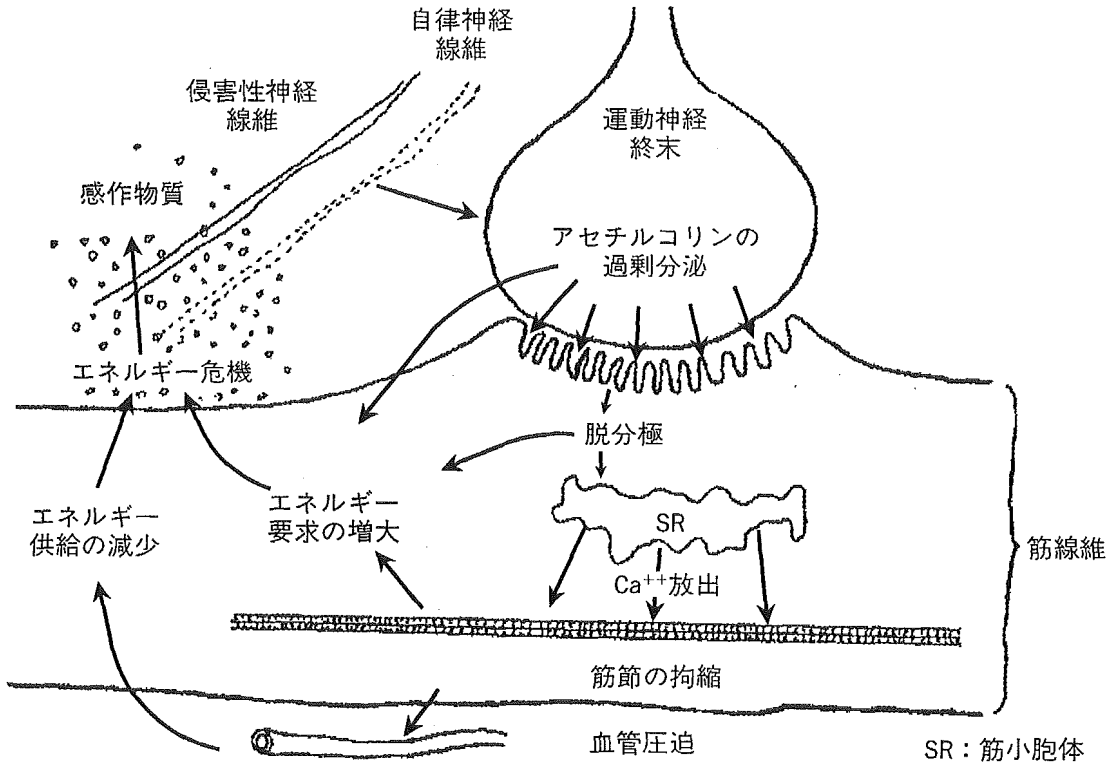

図1　トリガーポイントに関する統合仮説
トリガーポイントは筋の運動終板からアセチルコリンが過剰分泌され，限局した筋線維が過度に短縮した部位の集合体であり，その部位は持続的な筋の拘縮によるエネルギー危機によって組織損傷が起こり侵害受容器の感作をもたらすとしている（文献4より改図）

が生じる．この反応は脊髄反射とされており[11]，トリガーポイントの診断的な意義が極めて高いとされている[1,8]．

しかし，侵害性入力による脊髄反射性の筋収縮反応は，過剰興奮している運動終板近傍の刺激のみで起こる特殊な反応ではなく，筋膜や骨膜に対する侵害刺激で起こりうる現象である．また鍼治療においても鍼の刺入時にLTR様の現象が起こることは稀なことではないため，そのような反応をトリガーポイントの診断の最も重要な指標とするのは適切ではない．

4）筋の短縮時痛と筋力低下および可動域制限

トリガーポイントが出現すると，その筋では筋の短縮や筋力低下が起こり，関節可動域の制限が認められる．そこで，徒手検査によってその筋の筋力や可動域の制限の有無を調べることで，患者の症状の原因となっているトリガーポイントの存在する筋を推測することができる．また，トリガーポイントの存在する筋を短縮させるとその痛みが増悪するので，それを避けるために無意識下にその筋を伸長させた姿勢を取っている患者が多い．そこで，患者の姿勢を注意深く観察することもトリガーポイントの検出には重要である．

5）自律神経反応亢進部位

トリガーポイントのある部位では，交感神経活動の亢進を示す，立毛筋の収縮（鳥肌），発汗，さらには血管収縮なども観察されることがある．トリガーポイントと自律神経活動の亢進との関連についてはまだ不明な点が多いが，交感神経内には神経伝達物質としてノルアドレナ

リンのほか，ATPやneuropeptide Y (NPY)が共存しており，ATPやその分解産物であるアデノシンには発痛作用があり，NPYには血管や筋の収縮作用がある．そこで，トリガーポイントの成因として挙げられている持続的筋収縮に伴う血流阻害とエネルギー危機による発痛物質の産生に，交感神経が関与していることは十分に考えられる．交感神経と筋緊張の関連については拙稿を参照願いたい[12]．

2．トリガーポイントの不活性化法について

1) トリガーポイント注射

トリガーポイントを不活性化する方法としてTravellらが推奨してきた方法の一つが局所麻酔薬の注射であった[2]．しかし，複数のトリガーポイントが同一筋に存在する場合に大量の薬液を使用することが問題とされてきた．わが国ではトリガーポイント注射が保険の適応になっており，各種ビタミンやステロイドなどの薬物が用いられている．トリガーポイントの成因を運動終板でのアセチルコリンの過剰分泌という仮説に基づき，ボツリヌス毒素の使用も試みられている[13]が，その臨床効果はまだ十分に検証されていない．

MPS患者に対するトリガーポイント注射の臨床効果をevidence-based medicine (EBM)の観点から調べた結果から，質の高い臨床試験を抜粋して表2にまとめた．その結果は局所麻酔薬，ステロイド，ボツリヌス毒素Aのいずれを用いた群もプラセボ群以上の効果はなく，また薬液注射群と鍼刺激群との差も認められていない[14]．そこで，現時点では薬物を用いたトリガーポイント注射を選ぶ理由は見当たらず，少なからず有害事象が生じる可能性のある薬物を用いるよりも，生理食塩水や単純に鍼刺激を行う方がより妥当な選択と思われる．

2) ストレッチングと虚血性局所圧迫法

ストレッチングがトリガーポイントの不活性化に有効とされている．トリガーポイントのある筋が短縮していることから，その持続的筋収縮が筋血流を阻害し，筋の弛緩に必要なエネルギー供給を妨げているとすれば，ストレッチを繰り返すことで筋への血流を他動的に促進し，エネルギー供給を助けるほか，痛みの原因となりうる代謝産物を洗い流す効果もあると考えられる．また，虚血性圧迫と呼ばれる，トリガーポイントに15～30秒ほどずつ徐々に圧力を加える方法がある[15]．この場合には，ストレッチ的な要素が，圧迫された筋の周辺で生じているほか，反応性充血と呼ばれる現象が筋組織の血管で起こっている可能性もある．

3) 鍼刺激

トリガーポイントと鍼灸で用いられるツボ(経穴)とは部位的に密接な関連があり[16]，また，鍼灸師が治療部位として選ぶ部位の特徴はトリガーポイントに類似している[17]．表2にあるように，鍼刺激はトリガーポイント不活性化の一つの選択肢である．鍼手技にはいろいろあるが，Baldry[8]はトリガーポイントの皮下に鍼を置鍼する極めて繊細な方法を推奨しているが，それにストレッチを加える方法[18]も考案されている．一方，GunnはMPSを神経根症状の一つとみなし，その治療に筋への鍼通電刺激を勧めている[19]．

図2はポリモーダル受容器を中心にした鍼灸の作業仮説であるが，そこにはトリガーポイントとツボとの密接な関連と関連痛としてのMPSが描かれている．鍼の刺入はポリモーダル受容器を興奮させ，受容器末端から軸索反射としてcalcitonin gene-related peptide (CGRP)などの血管拡張性の神経ペプチドを放出することで局所の血流改善が生じる．それがエネルギー危機の状態を改善したり，感作物質を洗い流してトリガーポイント不活性化させる

表2　筋・筋膜性疼痛に対するトリガーポイント注射等の臨床試験の成績

著者名	発表年	スコア	デザイン	診断	n	介入	アウトカム	追跡期間	結果	備考
Fine ら	1988	5	クロスオーバー	MPS	16	A：bupivacaine の TPI B：同上＋ナロキソン	主観的評価	48 時間	A＞B	$p<0.02$
Frost ら	1980	5	RCT	MPS	60	A：mepivacaine の TPI B：生理食塩水の TPI	ペインスケール	4 日	B＞A	$p<0.05$
Garvey ら	1989	5	RCT	LBP	63	A：lidocaine の TPI B：lidocaine＋ステロイドの TPI C：注射針のみの TPI	ペインスケール	2 週	A＝B＜C	NS
Tschopp ら	1996	5	RCT	MPS	107	A：bupivacaine の TPI（IC 加える） B：lidocaine＋ステロイドの TPI C：生理食塩水の TPI	主観的評価	1 週	A＝B＝C	78％が疼痛緩解
Wheeler ら	1998	4	RCT	MPS	33	A：botulinum toxin A の TPI B：botulinum toxin A 倍量の TPI C：生理食塩水の TPI	VAS, PPT	4 カ月	A＝B＝C	有意に効果あり
Hesse ら	1994	4	RCT	片頭痛	85	A：鍼の TPI＋プラセボ丸薬 B：シャム鍼の TPI＋metoprolol 服用	頭痛日記	17 週	A＝B	有意に効果あり
Hong ら	1994	4	RCT	MPS	58	A：lidocaine の TPI B：注射針のみの TPI	ペインスコア	2 週	A＝B	直後効果のみ有意

システマティック・レビューで検討された文献のリストの中から臨床試験として質の高いものを抜粋してその概要をまとめたもの．スコアは5点を最高点として質の評価を行っている．結果の項では効果の大きい方を＞で示した．薬物注射以外にも生理食塩水や鍼刺激で同等の効果がみられる．詳細は文献14を参照されたい（TPI：トリガーポイント注射，RCT：ランダム化比較試験，LBP：腰痛，IC：皮内注射，PPT：圧痛閾値）

ほか，広汎性侵害抑制調節（diffuse noxious inhibitory controls：DNIC）[20]などの内因性鎮痛系を賦活することが考えられる．

3．線維性筋痛とは何か

1）線維性筋痛の定義

最近注目を浴びている筋痛症の一つに，線維性筋痛（fibromyalgia：FM）がある．FM の名称は，従来慢性筋痛症に対して使われてきた結合組織炎（fibrositis）という用語を見直した結果であるが，その‘線維性’の意味するところは明らかではない．

1990 年の ACR（American College of Rheumatologists：米国リウマチ学会）による定義[21]では，3カ月以上の全身性（wide spread）の痛みの病歴があること，図3に示す18カ所を4 kg の指頭による圧迫を加えた時，全身性に11カ所以上に痛覚を訴えることとしている．その部位がいずれも経穴部位に極めて近い点が注目される．

この定義では FM の主症状を筋痛としているが，実際の患者の訴えは痛み以外にも広範囲にわたっている．そこで 1992 年のコペンハーゲン会議における合意文書[22]では，従来の圧痛点の部位と数だけではなく，持続性の疲労，全身

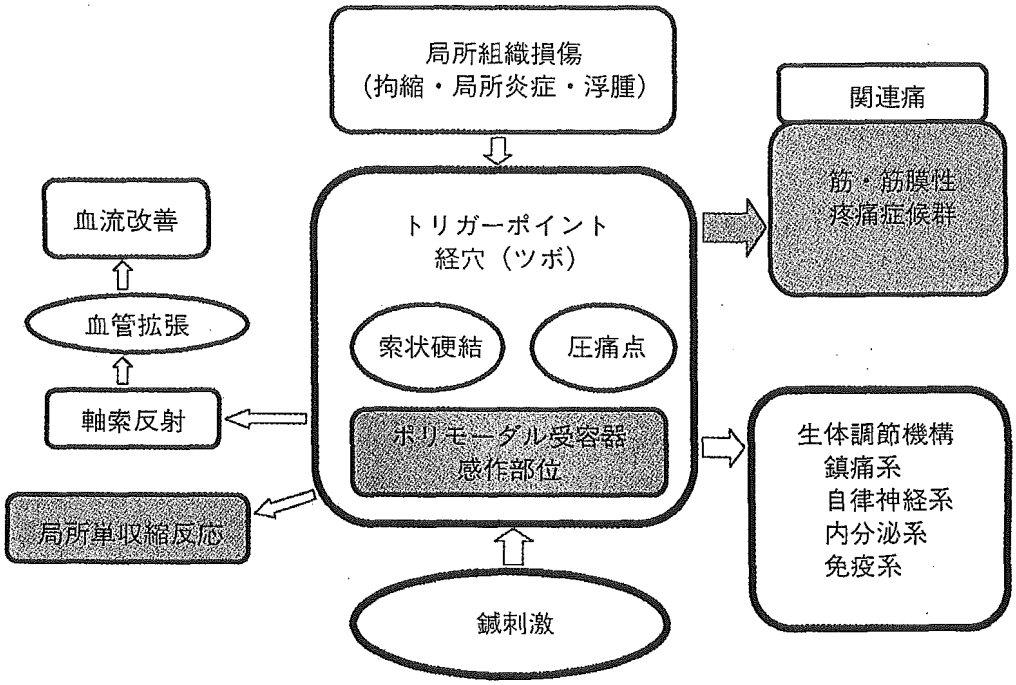

図2　ポリモーダル受容器とトリガーポイント発現の関連を示す模式図
鍼刺激がポリモーダル受容器を興奮させて鎮痛効果をはじめ，軸索反射性に血流の改善をもたらすことのほか，トリガーポイントの特徴として挙げられる索状硬結，圧痛，局所の単収縮反応，関連痛（患者の痛み再現）がツボへの刺激でも生じることを模式的に示している

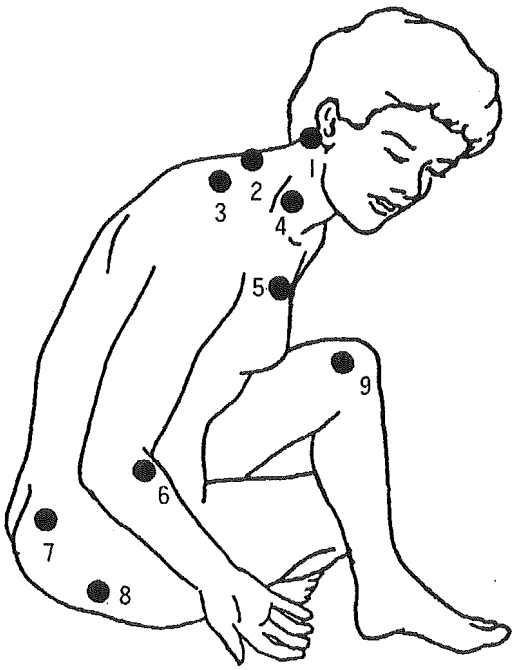

図3　線維性筋痛の診断に用いられる圧痛測定部位
全身の測定部位18カ所（図には片側の9カ所を示す）のうち，11カ所に圧痛が認められたものを線維性筋痛と診断する（文献21より改図）

性の朝のこわばり，不十分な眠り，併発する症状として，頭痛，過敏性膀胱，月経不順，寒冷に対する過敏，下肢の不随意な動き，奇妙なパターンのしびれ感と刺痛，運動の不耐性などを挙げた．その結果，わが国ではこれまでほとんど報告のなかったFM患者の存在がにわかに顕在化することになった．

2）線維性筋痛の成因とその治療法について

FMの原因として様々な可能性が挙げられている．成長ホルモンの分泌異常による睡眠障害がセロトニン産生を阻害してそれが内因性痛覚抑制系の働きを低下させて全身性の痛みを生じる，交感神経系の緊張の亢進による筋虚血によって全身性に痛覚閾値が低下する，筋の微小損傷がカルシウムイオンのリークを生じて筋収縮を強めて酸素供給を阻害する，大脳辺縁系の機能不全が感覚情報を誤認して痛みと感じる，などである．そのほか，アレルギー，感染，毒物，栄養不全などの関与や，多くのFM患者にストレス性の過換気症候群の兆候が認められることが報告されている．上述の多様な症状に関して，様々な薬物治療や運動療法などが試みられているが，いずれも十分な結論には至っていないのが現状である．紙数の関係で詳細については成書[23]を参照されたい．

3）FMとMPSの関連性について

FMとMPSはいずれも慢性的な筋痛が主訴であり現代医学的な検査所見では異常が認められない点に共通性があり，同一患者が両者に診断されることも稀ではない[24]．そのため，FMをMPS患者でトリガーポイントが全身に生じたもの[25]とする説もある．

しかし，FMは特に女性に多いこと，全身性の痛覚過敏や睡眠障害や易疲労感などMPSとは異なる点もみられるが，MPSがFMの病状へ変化することも稀ではないという指摘もある[26]．そこで，FMの治療としてもトリガーポイントの不活性化が有効とされている．一方，FMと慢性疲労症候群（chronic fatigue syndrome）の類似性も指摘されているが，その詳細は省略する．

おわりに

MPSを中心にその診断法と治療法について現状を紹介した．しかし，その成因として提唱されている統合仮説に関しても，まだ問題点は残されており，MPSの治療法を確立するためには，その成因を解明するとともに，いずれの治療方法に関してもきちんとした実験デザインの下で臨床試験を実施し，強いエビデンスを作ることが必要であろう．

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Trigger point acupuncture treatment of chronic low back pain in elderly patients – a blinded RCT

Kazunori Itoh, Yasukazu Katsumi, Hiroshi Kitakoji

Kazunori Itoh
research assistant
licensed acupuncturist
Department of Clinical Acupuncture and Moxibustion

Yasukazu Katsumi
professor
Department of Orthopaedic Surgery

Hiroshi Kitakoji
professor
licensed acupuncturist

Meiji University of Oriental Medicine
Kyoto, Japan

Correspondence:
Kazunori Itoh

k_itoh@
muom.meiji-u.ac.jp

Abstract

Objective There is some evidence for the efficacy of acupuncture in chronic low back pain, but it remains unclear which acupuncture modes are most effective. Our objective was to evaluate the effects of two different modes of trigger point acupuncture on pain and quality of life in chronic low back pain patients compared to standard acupuncture treatment.

Methods Thirty five consecutive out-patients (25 women, 10 men; age range: 65–81 years) from the Department of Orthopaedic Surgery, Meiji University of Oriental Medicine, with non-radiating low back pain for at least six months and normal neurological examination, were randomised to one of three groups over 12 weeks. Each group received two phases of acupuncture treatment with an interval between them. Nine patients dropped out during the course of the study. The standard acupuncture group (n=9) received treatment at traditional acupuncture points for low back pain, while the other acupuncture groups received superficial (n=9) or deep (n=9) treatments on trigger points. Outcome measures were VAS pain intensity and Roland Morris Questionnaire.

Results After treatment, the group that received deep needling to trigger points reported less pain intensity and improved quality of life compared to the standard acupuncture group or the group that received superficial needling to trigger points, but the differences were not statistically significant. There was a significant reduction in pain intensity between the treatment and interval in the group that received deep needling to trigger points ($P<0.01$), but not in the standard acupuncture group or the group that received superficial needling to trigger points.

Conclusion These results suggest that deep needling to trigger points may be more effective in the treatment of low back pain in elderly patients than either standard acupuncture therapy, or superficial needling to trigger points.

Keywords

Trigger point, low back pain, elderly, randomised controlled trial.

Introduction

Chronic low back pain (LBP) is a major health problem in modern society. Seventy to eighty five percent of the population will experience back pain at some time in their lives.[1] Each year, 5–10% of the workforce is off work because of their LBP, but most of them are off for less than seven days. Almost 90% of all patients with acute LBP recover quite rapidly, regardless of therapy.[1] The remaining 10% are at risk of developing chronic pain and disability and account for more than 90% of the social costs for back incapacity.[2] The proportion of elderly patients who have LBP is greater than that of young adults.

In younger adults most back pain arises from the physical stresses on normal spinal structures,[3] whereas in the elderly, osteoarthrosis of the intervertebral joints and/or osteoporosis with collapse of the vertebral bodies is almost invariably present. Analgesia in the elderly is frequently unsatisfactory, and the incidence of adverse drug reactions, particularly to non-steroidal anti-inflammatory drugs (NSAIDs), is well known.[4] For this reason, many patients request physical therapies such as acupuncture.

Acupuncture has been frequently applied to chronic LBP. A number of randomised controlled trials on acupuncture for chronic LBP have already been published.[5,6] In 1997, during the NIH Consensus Conference, and more recently in

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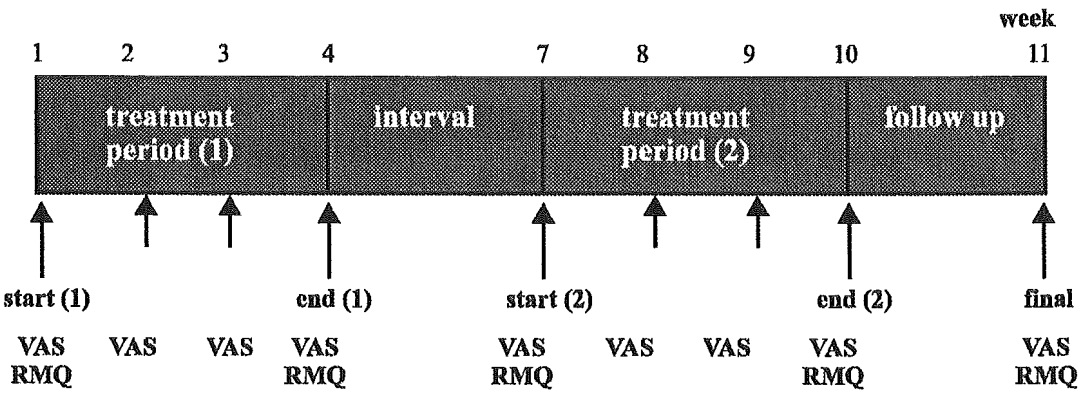

Figure 1 Time line of the trial. Evaluation was performed immediately before each treatment. VAS: visual analogue scale, RMQ: Roland Morris Questionnaire.

systematic reviews indicating equivocal results,[7] the question was asked: can acupuncture contribute to the conservative treatment of chronic LBP? The common conclusion was that all the studies conducted so far lacked adequate design and methodology, including adequate control of the quality of the administered acupuncture. Furthermore, the method of point selection in published trials seems to be more simple and formulary than that used in the standard acupuncture practice at our clinic. We believe that the response to acupuncture and therefore the success of a trial depends to an important degree on the choice and the number of points needled.

Our main aim in this study was to determine whether acupuncture at trigger points is an effective treatment for chronic LBP in the elderly, when compared to standard acupuncture at traditional points.

Methods

Patients

Patients aged 65 years or over with a history of LBP were recruited from the Meiji University of Oriental Medicine Hospital specifically for the study. Inclusion criteria were (1) lumbar or lumbosacral LBP for a duration of six months or longer; (2) no radiation of LBP; (3) normal neurological examination findings of lumbosacral nerve function, including deep tendon reflexes, plantar response, voluntary muscle action, straight leg raising, and sensory function; and (4) no previous treatment with acupuncture for LBP. Exclusion criteria were (1) major trauma or systemic disease; and (2) other conflicting or ongoing treatments. However, patients were included with medical conditions if there had been no change in drugs or dosage taken for one month or longer.

Patients who gave written informed consent were enrolled and randomly allocated, using a computerised randomisation programme, to one of three groups: the standard acupuncture (SA) group, who received acupuncture at traditional points for LBP, or the group that received superficial needling to trigger points (S-TrP) or the group that received deep needling to trigger points (D-TrP). Ethical approval for this study was given by the ethics committee of Meiji University of Oriental Medicine.

Design

The study was a subject and assessor blinded, randomised, controlled clinical trial. The three groups received two phases of acupuncture treatment with an interval between the two phases (the original design was a crossover study). Each phase lasted three weeks and the total experiment period was 12 weeks (Figure 1). Each patient received a total of six 30 minute treatments, one per week.

Blinding

Patients were blinded to their treatment. They were told before randomisation that they would be allocated to one of three treatments. The measurements were performed by an independent investigator who was not informed about the treatment allocation.

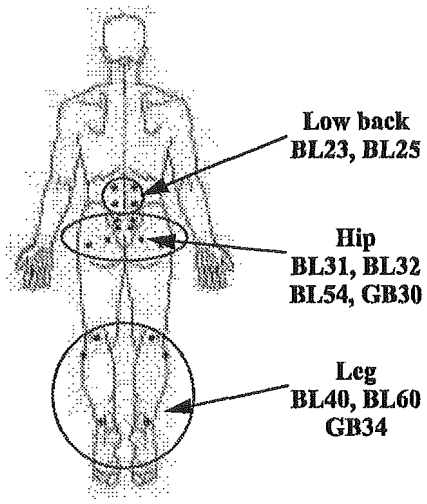

Figure 2 Acupuncture points used for treatment of the standard acupuncture group.

Treatment

The SA group received treatment at traditional points for LBP. After a literature review on acupuncture for LBP, only widely accepted acupoints were selected.[8-11] The standard points in the lumbar region (local points) were BL23, 25, and GB30; standard points on the lower extremity (distal points) were BL40, 60 and GB34. Additionally, up to four *ah shi* points of greatest tenderness were chosen, which were often close to, but not necessarily identical to, BL54, 31 and 32 (Figure 2). In the SA group, disposable stainless needles (0.2mm x 40mm, Seirin Co Ltd) were inserted into the muscle (to a depth of 20mm) and the 'sparrow pecking' technique (alternate pushing and pulling of the needle) was applied. When the subject felt dull pain or the acupuncture sensation *(de qi)*, the manipulation was stopped and the needle retained for ten more minutes.

The S-TrP and D-TrP groups received treatment at trigger points. The correct application of the technique requires experience in palpation and localisation of tender points in taut bands of skeletal muscle (myofascial trigger points). Precise needling of active myofascial trigger points provokes a brief contraction of muscle fibres. This local twitch response should be elicited for successful therapy, but it may be painful and post-treatment soreness is frequent.[12;13]

Table 1 **Muscles treated in the trigger point acupuncture group**

Muscle	S-TrP Group	D-TrP Group
Quadratus Lumborum	6	5
Iliopsoas	4	4
Piriformis	2	4
Gluteus Maximus	5	3
Iliocostalis Lumborum	1	3
Gluteus Minimus	0	1
Hamstring	1	0
Other	1	2

In this study, the most important muscles of the lumbar and lower extremity were examined for myofascial trigger points (Table 1). In the trigger point acupuncture treatment groups, disposable stainless needles (0.2mm x 50mm, Seirin) were inserted into the skin over the trigger point. In the S-TrP group, insertion was to a depth of about 3mm; in the D-TrP group the needle was advanced a further 20mm into the muscle. The 'sparrow pecking' technique was then applied. In S-TrP group, when the subject felt a kind of dull pain or acupuncture sensation *(de qi)*, the manipulation was stopped and the needle retained for ten more minutes. In contrast, in D-TrP group the manipulation was stopped when the local twitch response was elicited, and the needle retained for a further ten minutes. The mean number of insertions was 2.3.

The acupuncture was performed by an acupuncturist who had four years of acupuncture training and seven years of clinical experience.

Evaluation

Primary outcome measures were pain intensity, quantified using a 10cm visual analogue scale (VAS), and pain disability,[14] measured using the Roland Morris Questionnaire (RMQ).[15] The RMQ consists of 24 questions answered yes or no (range 0–24 points, the worst condition being 24).

The VAS measures were assessed immediately before the first treatment (pre) and one, two, three, six, seven, eight, nine, and twelve weeks after the first treatment. The RMQ measures were assessed before the first treatment and three, six, nine, and twelve weeks after the first treatment. The VAS and RMQ measures were completed by participants immediately before each treatment (Figure 1).

Table 2 Characteristics of patients included in RCT of acupuncture

Treatment Group	Trigger point acupuncture		Standard acupuntore
	Superficial needling	Deep needling	
Sample size	9	9	9
Age (y;mean±SD)	70.1±8.9	71.9±3.7	73.8±7.0
Diagnosis, number	Spondylosis 8 Osteoporosis 2 Compression fracture 1 Entrapment neuropathy 1	Spondylosis 8 Osteoporosis 3 Compression fracture 2 Entrapment neuropathy 2	Spondylosis 8 Osteoporosis 2 Compression fracture 1 Entrapment neuropathy 2
Pain duration (y;mean±SD)	5.2±2.6	7.4±4.5	5.4±3.7
VAS (mm;mean±SD)	65.6±17.1	65.6±17.3	64.0±20.2
Drugs treatment used for back pain	Poultice 7 Analgesic 3 Vitamin D 1 Bone resorption inhibitor 2	Poultice 6 Analgesic 3 Vitamin D 3 Bone resorption inhibitor 3	Poultice 5 Analgesic 2 Vitamin D 2 Bone resorption inhibitor 2

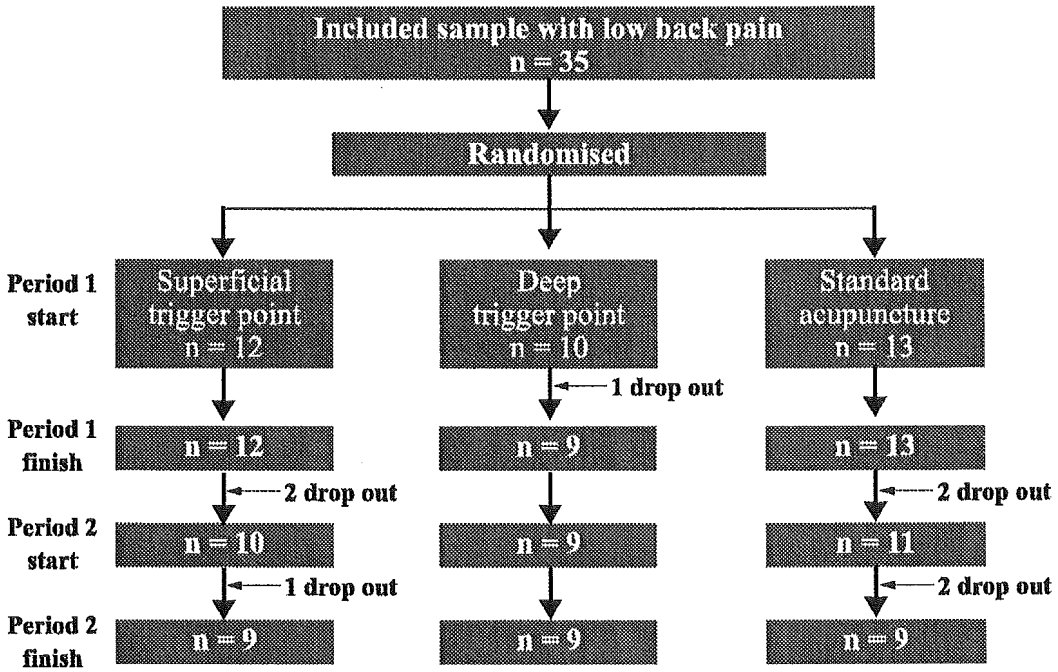

Figure 3 Participation flow in the study. Eight patients were excluded after dropping out.

Statistical analysis

The data are reported as means ± standard deviation (mean±SD). The nonparametric Tukey, Dunnett's multiple test, Fisher's exact test and one-way ANOVA (StatView v5; SAS Institute Inc, NC) were used for the statistical analysis. The level for statistical significance was set at $P<0.05$. Comparisons were made before and after each treatment, and between three kinds of intervention.

Results

Patient characteristics

Thirty-five patients (25 women, 10 men; age range: 65–81 years) were randomised and started treatment. No differences were found between the three groups regarding the variables measured at baseline including age, disease, pain duration, VAS and drug use (Table 2).

Patient progress through the trial is shown in Figure 3. Four patients in the SA group and three