Management of Myofascial Trigger Point Pain

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Summary
Successful management of myofascial trigger point (MTrP) pain depends on the practitioner finding all of the MTrPs from which the pain is emanating, and then deactivating them by one of several currently used methods. These include deeply applied procedures, such as an injection of a local anaesthetic into MTrPs and deep dry needling (DDN), and superficially applied ones, including an injection of saline into the skin and superficial dry needling (SDN) at MTrP sites. Reasons are given for believing that DDN should be employed in cases where there is severe muscle spasm due to an underlying radiculopathy. For all other patients SDN is the treatment of choice. Following MTrP deactivation, correction of any postural disorder likely to cause MTrP reactivation is essential, as is the need to teach the patient how to carry out appropriate muscle stretching exercises. It is also important that the practitioner excludes certain biochemical disorders.

Keywords
Myofascial trigger points, deep dry needling, superficial dry needling.

Introduction
For the successful management of myofascial trigger point (MTrP) pain it is essential to first identify all of the MTrPs from which the pain is emanating, and to deactivate them by one or other of several methods currently employed. Following this, measures should be adopted as necessary to prevent reactivation of the MTrPs. In addition, treatment should be started as early as possible, before pain-perpetuating changes take place, in particular spinal cord neuroplasticity (central sensitisation).

Systematic Search for Pain-Producing MTrPs
The identification of all of the active MTrPs is mandatory, because if only one of them is overlooked the persistence of a certain amount of pain is inevitable.

It is therefore necessary to locate MTrPs not only in the primarily affected muscles, but also in their synergists and antagonists (secondary MTrPs). In addition, it is necessary to search for any satellite MTrPs that may be present in the primary and secondary MTrPs' zones of pain referral.

Guidance as to where to look for these MTrPs may be obtained from carefully noting the distribution of pain and by observing which movements are restricted as a result of it.

The search should be carried out by means of the palpating finger being drawn across each part of a muscle in a manner similar to that employed when kneading dough.

Some authorities advocate the use of flat palpation for any muscle where only one of its surfaces is accessible for palpation, and pincer palpation where both sides of the muscle are accessible, such that it is possible to grasp it between the fingers. The difficulty with employing the latter technique, however, as Sir Thomas Lewis pointed out over 50 years ago,[1] is that normal healthy muscle is extremely tender when firmly squeezed. Because of this, it is my personal preference to use flat palpation for all muscles. When doing this the pressure applied with the examining finger must be very firm (approximately 4kg), or the characteristic 'jump' (involuntary flexion withdrawal) and 'shout' (the utterance of an expletive) reactions at an active or latent MTrP site will not be elicited. It cannot be emphasised too strongly that one of the

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commonest reasons for MTrPs being overlooked is that palpation has been carried out too gently.

When palpating a superficially placed muscle in the manner just described it is often possible to feel a MTrP-related taut band. Should this be 'snapped', by drawing the examining finger across it at a TrP (trigger point) site in a manner similar to that employed when plucking a violin string, a transient contraction of the muscle fibres may be evoked. This local twitch response may be either visible, or felt under the examining finger, or both.

When pressure is applied for about 10-15 seconds to a pain-producing MTrP it is possible to reproduce the patient's spontaneously experienced pain. It might be thought that it is worthwhile doing this routinely in order to confirm that the TrP is in an active phase. However, carrying this out at a number of MTrP sites is liable to cause the patient considerable discomfort. As Hong et al have shown,[2] this pressure-induced pain referral is not confined to active MTrPs, for it may at times be observed with latent trigger points.

My pragmatic approach therefore,[3] is to avoid this test, for when, in the absence of any other obvious pain-producing disorder, MTrPs with their characteristic 'jump' and 'shout' reactions are found, in a region of the body affected by a persistent dull aching type of pain, and the latter is relieved by one or other of the MTrP deactivating procedures to be discussed, it is reasonable to assume that the pain must have been emanating from them.

There is no general consensus as to the essential criteria for the diagnosis of the MTrP pain syndrome. In view of this unfortunate state of affairs, and in an attempt to rectify it, the International Myopain Society has been recently engaged in conducting a large-scale multi-centre study.[4] Its findings are awaited with great interest.

Treatment of Pain-Producing Myofascial Trigger Points - Historical Review

Historically, the method which must take pride of place as having been the first to be employed, in the 7th century A.D. by the Chinese physician Sun Ssu-Mo, is dry needling, of what he called Ah-Shih points.[5] Clearly, from his description of them, they are what are currently referred to as MTrPs.

News that insertion of needles into the body for therapeutic purposes had been a long established practice, first in China, and then in Japan, reached Europe in the 17th century, principally as a result of the Dutch physician Willem ten Rhijne.[6] Whilst working as a medical officer on the staff of the Dutch East Indies Company in Java, he wrote a book describing what he had observed.

His contemporaries in the Western World, however, viewed this type of treatment with considerable incredulity, particularly as, by that time, renaissance anatomists such as Vesalius, had, during the course of dissecting the human body, failed to find evidence of channels corresponding to those containing Qi (vital energy) that had been described by the Chinese.

Consequently, Europeans took no further interest in acupuncture until the early 19th century, when, somewhat surprisingly, books recommending its use appeared in France, Italy, and England.

In England, the London medical practitioner JM Churchill drew attention to the merits of dry needling by writing about it in two books, the first published in 1821[7] and the second in 1828.[8]

It is obvious, from reading Churchill's books, that he restricted himself to treating the disorder that he called rheumatalgia, which today is called the MTrP pain syndrome. It is equally clear that he employed a strong acupuncture stimulus, for, from looking at the length of the flanged needles he used (see figure 1), he clearly inserted them deeply into muscle at points of maximum tenderness, and then left them in situ for five to six minutes.

Although Churchill reported good results with this treatment, its use for the rest of the 19th century was restricted to a few centres, seemingly because no one could offer a plausible explanation as to how it might work. One of its most distinguished exponents, however, was Sir William Osler,[9] who, in the 8th edition of his student text book, published in 1912, at a time when he was Regius Professor of Medicine at Oxford University, wrote,

"...For lumbago, acupuncture is in acute cases the most efficient treatment. Needles of from three to four inches in length (ordinary bonnet needles, sterilised will do) are thrust into the lumbar muscles at the seat of the pain and withdrawn after five to ten minutes..."

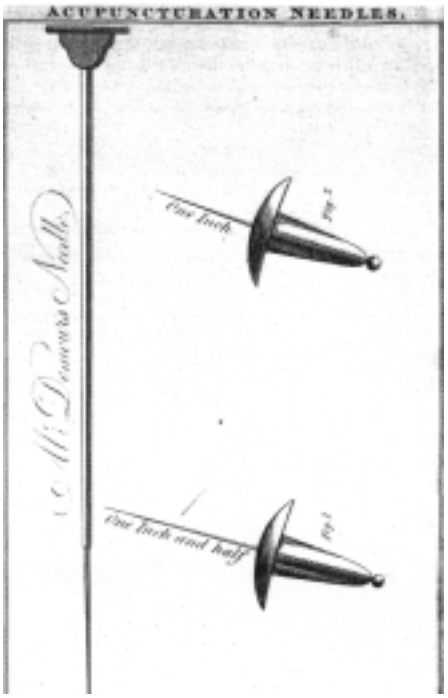

Figure 1 Needles employed by JM Churchill, from his book 'A treatise on acupuncturation…' published in 1821.

From his description, he, like Churchill, was clearly employing strongly applied deep needling, at what today would be called MTrPs.

Despite the high esteem in which Osler was held, his teaching concerning acupuncture for the treatment of pain from the disorder which, during the 19th century, was called muscular rheumatism, and which from the beginning of the 20th century was for some time called fibrositis,[10] was largely ignored. There were two reasons for this; a lack of understanding as to how acupuncture might work, and ignorance concerning the pathophysiology of this muscle pain disorder.

Insight into the latter was not gained until the 1930s. In London, Sir Thomas Lewis and his young assistant Jon Kellgren investigated the referral of pain from a noxious stimulus (hypertonic saline) applied to various muscles in healthy volunteers,[11] and then, Kellgren studied similar pain in patients suffering from what he called myalgia.[12]

Kellgren observed that the spontaneously occurring pain in this disorder may be reproduced, by applying sustained pressure to points of maximum tenderness in muscle, and may be alleviated, by injecting 1% procaine (Novocain) into them.

These extremely important observations were largely ignored in Britain, and might have been lost sight of completely had they not, together with those made by others,[13;14] come to the attention of the American physician Janet Travell. Travell, from the 1940s onwards, made a life time study of the subject of myofascial pain, introducing this term and the term MTrP, and showing that each muscle in the body has its own specific pattern of MTrP pain referral.

With respect to treatment, Travell was quick to realise that the analgesia produced by injecting procaine into a MTrP could not, as Kellgren had assumed, be due to its nerve blocking effect, as it lasted too long. In addition, she found that pain relief of a similar duration could be obtained by simply inserting a needle into the MTrP.[15] She also found, however, that the latter is an extremely painful procedure, and in order to suppress this ephemeral treatment-evoked pain, decided to continue to employ Kellgren's method of injecting a local anaesthetic through the needle.

A disadvantage of using a local anaesthetic for this or any other purpose is that it very occasionally leads to the development of an allergic, or even life-threatening, anaphylactic reaction.

For this reason, during the 1950s, the American physician Anders Sola and his co-workers decided to see whether it was possible to deactivate MTrPs by simply injecting saline into them. Sola and Kuitert carried out this procedure on 100 consecutive patients and concluded that, '*the use of normal saline has none of the disadvantages often associated with the use of a local anaesthetic but appears to have the same therapeutic effect*'.[16] Sola and Williams then carried out the same procedure on 1000 consecutive patients and confirmed its efficacy.[17]

Despite these encouraging results, which, in retrospect, were likely due to the effect of the needle rather than the saline injected through it, no further interest seems to have been taken in the technique until 1980, when Frost compared the effect of injecting saline with that of the long-acting local anaesthetic mepivacaine into MTrPs.[18] Frost had decided to use saline in one of the two groups on the assumption that it would have no

more than a placebo effect. He was therefore surprised to find that 76% of patients in the saline group had pain relief, in contrast with 57% in the local anaesthetic group. This led him to comment, 'The study raises questions about the mechanism by which local injections into muscles relieves pain, since there is a possibility that a similar effect might also be achieved by merely inserting a needle into the trigger point.'

A conclusion that, seemingly unbeknown to him, had been reached many years previously.

Currently Employed Methods - Deeply Applied Techniques

These include injection into MTrPs of a corticosteroid, a non-steroidal anti-inflammatory drug, or botulinum A toxin; injection into MTrPs of a local anaesthetic; and deep dry needling (DDN) of MTrPs.

Injection of a Corticosteroid

Bourne compared the effect of injecting into MTrPs a corticosteroids/local anaesthetic mixture with local anaesthetic alone and found that the mixture gave better results.[19;20] However, a corticosteroid repeatedly injected into tissues is liable to damage them. Therefore its use for the deactivation of MTrPs cannot be recommended.

Injection of a Non-Steroidal Anti-Inflammatory Drug

Drewes et al carried out a double-blind study comparing the relative pain-relieving effectiveness of injecting prednisolone or diclofenac into MTrPs.[21] Thirty-eight patients completed the study, and it was shown that both drugs are equally effective, with 84 % of patients being significantly improved. It has to be pointed out, however, that injection of a steroid into a muscle is liable to damage its fibres, and when given into superficial tissue is liable to cause the skin to become depigmented. Also, injection of diclofenac into superficial tissue may produce skin necrosis. The routine use of either of these two treatments cannot therefore be recommended.

Injection of Botulinum A Toxin

Cheshire explored the possibility of employing injection of botulinum A toxin in the treatment of MTrP pain,[22] in view of its ability to relax muscle, and its usefulness because of this in treating dystonia. In his small study, four out of six patients with MTrP pain had a pain reduction of at least 30% with this form of therapy.

Yue then carried out a retrospective study of 112 patients who had had their myofascial pain treated by this means.[23] He found that 86% had reported fair to excellent pain reduction but 17% had reported moderate to severe side effects, which included impaired motor function and the eventual development of muscle atrophy. Clearly, there is no place for this procedure in the routine treatment of this type of pain.

Injection of a Local Anaesthetic

The manner in which this technique has been employed over the years has recently been considerably modified. It has long been known that when a needle is rapidly inserted into a MTrP a local twitch response (LTR) can be evoked. Hong recently observed that when he either injected a local anaesthetic, or inserted a needle, into a MTrP, he could produce a succession of LTRs.[24] From this he concluded that a MTrP is made up of a number of individual loci, and that for its successful deactivation each of these loci has to be penetrated, with the consequent production of numerous LTRs. Carrying out of this technique requires considerable manual dexterity. It is a very painful procedure, and gives rise to appreciable post-treatment soreness that is generally considered to be due to needle-induced bleeding into the tissues.

Deep Dry Needling (DDN)

DDN has been used intermittently over the centuries, but the first person in recent times to become a strong advocate of its use was the Czech physician Karel Lewit. In his classic paper on the subject, published in 1979,[25] he described the results of treating myofascial pain in 241 patients, by inserting a needle into what he variously called sites of maximal tenderness, trigger zones, and pain spots, or what, from his description, would be currently called MTrPs. He admitted that deep needling of this type gives rise to a considerable amount of pain, but, undeterred, stated that its effectiveness is related to the intensity with which the pain is felt at the trigger zone, and that this in

turn is dependant on the precision with which the site of maximum tenderness is located by the needle.

Since that time Chan Gunn in Vancouver has written extensively and lectured widely about the myofascial pain-relieving effect of this type of treatment.[26] He calls his particular technique intramuscular stimulation.

DDN's Proposed Neurophysiological Mechanism

As stated previously one of the effects of rapidly inserting a needle into the substance of an active MTrP is to produce a local twitch response, with consequent alterations taking place in the length and tension of muscle fibres. This in turn leads to the arousal of mechanoreceptive activity and the development of a large diameter sensory afferent input to the dorsal horn. Chu has postulated that this sensory input has the 'gate'-like effect of blocking the intra-dorsal horn passage of noxious information generated in MTrP nociceptors, with consequent alleviation of the myofascial pain.[27]

Both Chu[28;29] and Hong[30] believe that evoking multiple twitch responses increases the effectiveness of DDN. For this reason Chu now refers to it as twitch-obtaining intramuscular stimulation (TOIMS).

DDN is not only a very painful procedure but is liable to damage neighbouring structures, including nerves and blood vessels. As stated earlier, it is because of the latter that there is a high incidence of post-treatment soreness.

In my opinion, because superficially applied techniques have none of the disadvantages of deep stimulation, and seem largely to be equally effective, I recommend the use of the former, in particular superficial dry needling (SDN), for the majority of cases. DDN should be reserved for those cases where a particularly strong stimulus is required, such as when a paravertebral muscle is in severe spasm as a result of an underlying radiculopathy.

Currently Employed Methods - Superficially Applied Techniques

Stretch and Spray

Kraus first introduced this technique in 1941,[31] but its main protagonist for the deactivation of MTrPs was Janet Travell. Initially ethyl chloride was sprayed on to the skin, but because this is highly inflammable Travell introduced the safer alternative flouri-methane.[32] This is not universally available, and therefore it is not widely used. Those who continue to employ it do so mostly in combination with exercises designed to stretch muscles that remain shortened despite carrying out some other MTrP deactivating procedure.

Intradermal and Subcutaneous Injections

During the early 1990s Byrn and his co-workers found that injecting sterile water into the skin overlying MTrPs in the neck and shoulder girdle muscles of patients suffering from whiplash injuries, relieved the pain emanating from these points for significant periods of time.[33;34]

Unfortunately, one important disadvantage of injecting water into the skin is that it gives rise to an intense and very distressing burning sensation. Byrn et al therefore carried out a trial comparing the relative effectiveness of injecting sterile water, or normal saline, into the subcutaneous tissues at MTrP sites in patients with this type of injury. A subcutaneous injection of water proved to be the most effective treatment, but again, as when inserted into the skin, it gives rise to a transitory but intense burning sensation similar to that produced by a wasp sting. They concluded, however, that despite this, *'most patients tolerate it because the treatment works'*. They have since used this method widely in the treatment of the MTrP syndrome.

The myofascial pain relieving effect of this technique must be due to water having a stimulating effect on A-delta nociceptors in the skin and subcutaneous tissues, in a manner similar to that brought about by SDN, but with the important difference that with the latter there is no discomfort other than the production of a transitory pricking sensation.

Superficial Dry Needling (SDN)

When first starting to treat MTrP pain in the late 1970s it was my practice to employ the deep needling technique advocated by Lewit.[25]

In the early 1980s, however, a patient was referred to me with pain down the arm from a MTrP in the scalenus anterior muscle. In view of the proximity of the apex of the lung, rather than push the needle deeply into the muscle, I considered it more prudent to insert it into the subcutaneous tissues immediately overlying the

MTrP. This proved to be sufficient, for after leaving the needle in situ for a short time and then withdrawing it, the exquisite tenderness at the MTrP site disappeared and the spontaneously occurring pain in the arm was alleviated.

This SDN was then used to deactivate MTrPs in other parts of the body, where it was found to be equally effective, even when the muscle containing the MTrPs was deep lying. Furthermore, any palpable bands found to be present before the treatment disappeared after it.

At about the same time Macdonald et al confirmed the efficacy of SDN in a well conducted trial on patients with MTrP pain in the lumbar region.[35]

Mechanisms Responsible for SDN's Pain-Suppressing Effect

Bowsher has explained that the MTrP pain relieving effect of inserting a needle into the skin and subcutaneous tissues at a MTrP site is because it stimulates A-delta nerve fibres, with the consequent release of opioid peptides from enkephalinergic inhibitory interneurons in the dorsal horn.[36] These peptides then inhibit the intra-dorsal horn transmission of nociceptive information conveyed to the cord via group IV sensory afferents from the MTrP (see figure 2).

Confirmation that needle-induced analgesia is opioid peptide mediated, comes from it having been shown that it is abolished by the administration of the endorphin antagonist naloxone.[37]

A needle inserted into the skin and subcutaneous tissues stimulates A-delta fibres not only mechanically, but also by setting up a low-intensity galvanic current of injury, brought about as a result of the difference in electrical potential that exists between the needle and the skin.

This current is generated not only whilst the

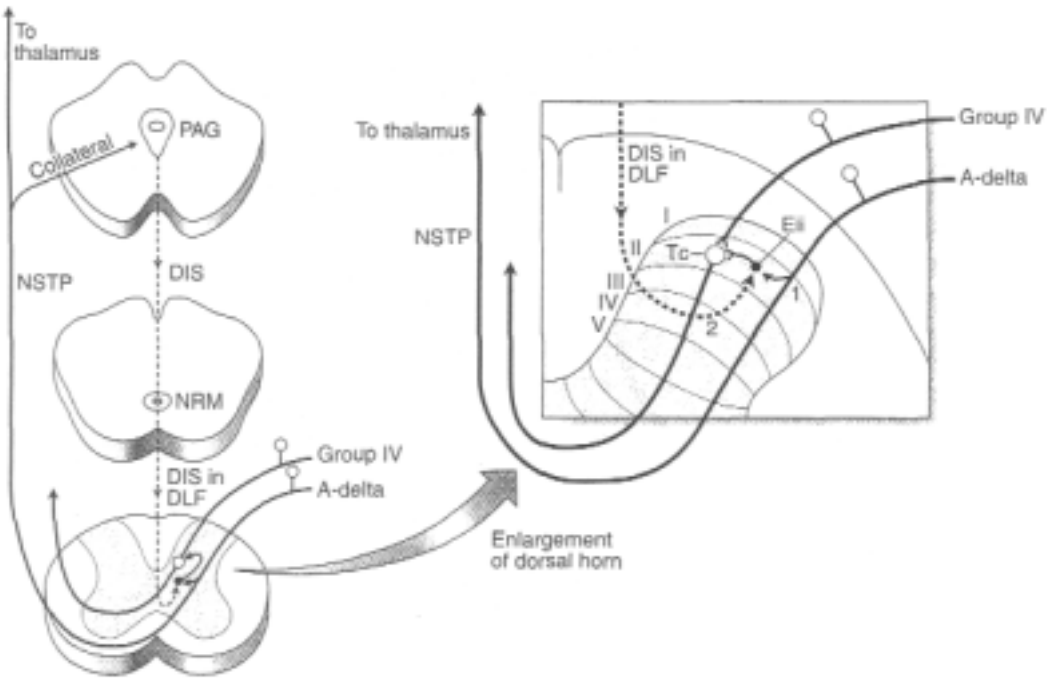

Figure 2 Diagram to show mechanisms considered to be responsible for the blocking of intra-dorsal horn transmission of MTrP group IV nociceptive information as a result of segmental superficial dry needling of A-delta nerve fibres.
Enkephalinergic inhibitory interneurons (Eii) in the dorsal horn become activated as a result of A-delta nerves having a direct link with them (1), and an indirect link with them (2). The latter being a result of the neospinothalamic pathway (NSTP) up which A-delta sensory information is transmitted having a collateral which projects to the periaqueductal grey area (PAG) in the midbrain at the upper end of the serotonergic descending inhibitory systems (DIS) which, from the nucleus raphe magnus (NRM) in the medulla, descends in the dorsolateral funiculus (DLF), and which, on reaching dorsal horns, projects to Eiis. Opioid peptides produced by these Eiis then inhibit activity in the transmission cells (Tc) that are projected onto by group IV sensory afferents.

needle is in situ, but also for an appreciable time after it has been taken out. This sustained effect, as Karavis has pointed out,[38] is because '*after withdrawing the needle, the unequal distribution of electrical potential as a result of the high concentration of potassium ions round the edges of the injury creates an electrical flux potential field which acts as a stimulator of the free nerve endings in the skin for 72 hours'*.

It follows, therefore, that when a needle is inserted into the skin and subcutaneous tissues overlying a MTrP, for the purpose of deactivating the latter, A-delta nerve fibres are stimulated briefly mechanically, and more long-lastingly by the development of an electric current.

Strong, Average and Weak Responders

As Mann has pointed out,[39] patients are either strong, average or weak responders to acupuncture. A person who is a strong reactor to dry needle stimulation is liable to have a temporary exacerbation of MTrP pain should the needling be carried out too vigorously. Conversely, a weak reactor obtains pain relief only if the stimulus applied is a strong one. There is no way of telling into which category a patient belongs, other than by practising a graduated approach to dry needle stimulation the first time it is carried out.

Determination of Optimum Superficial Dry Needling Stimulus

When treating a patient for the first time it is my practice to insert an acupuncture needle (0.3x30mm) into the tissues overlying a MTrP to a depth of about 5-10mm, and to leave it in situ, without any form of manipulation, for about 30 seconds. This is to produce the minimum neural stimulation required to abolish the exquisite tenderness which, before needling, had given rise to a pressure-induced wince (the jump sign), and in some cases the utterance of an expletive (the shout sign). On withdrawing the needle, pressure equal to that applied before needling is applied to the MTrP site, to see whether this has been achieved. If so, then the patient is a strong responder. If not, the needle has to be re-inserted and left in situ for 2-3 minutes. Occasionally, even this is not sufficient, due to the patient being a very weak responder. In such a case the needle has to be once again re-introduced and left in place for an even longer period, whilst at the same time being vigorously twirled.

For those who are very strong responders, even a 30 second period of stimulation may prove too much, and in such cases all that may be required is to insert the needle and then to immediately withdraw it. Every patient who undergoes SDN for the first time should therefore be informed that the initial treatment may temporarily exacerbate the pain, although admittedly any such flare-up usually lasts for only 12-24 hours.

Providing that there has not been a flare-up of pain following the first treatment, which should be the case for the most part, where the graduated approach just described has been followed, the time for which needles should be kept in situ on subsequent occasions should be either the same, or increased if the pain relief has not been as good as might have been expected.

Indications for the Use of SDN.

The author contends, based on 20 years of experience, that SDN, because it is safe and readily carried out, should be used in the majority of cases for the deactivation of MTrPs. DDN should be reserved for that relatively small number of cases where a particularly strong stimulus is required, either because the patient is an exceptionally weak responder, or because there is particularly severe muscle spasm, such as not infrequently occurs in the paravertebral region due to an underlying radiculopathy.

Before leaving this subject it should be pointed out that the deactivation of MTrPs should be carried out as early as possible, before various pain-perpetuating mechanisms, including in particular central sensitisation resulting from neuroplasticity in the dorsal horn, (see figure 3) have had time to develop.[3]

Post-MTrP Deactivation Procedures

It is essential that measures should be taken to correct such MTrP reactivating factors as postural disorder, relative shortness of an upper limb and leg length inequality. It is also necessary to stress the importance of teaching post-deactivation muscle stretching exercises. Biochemical factors may also have to be corrected. Gerwin has drawn

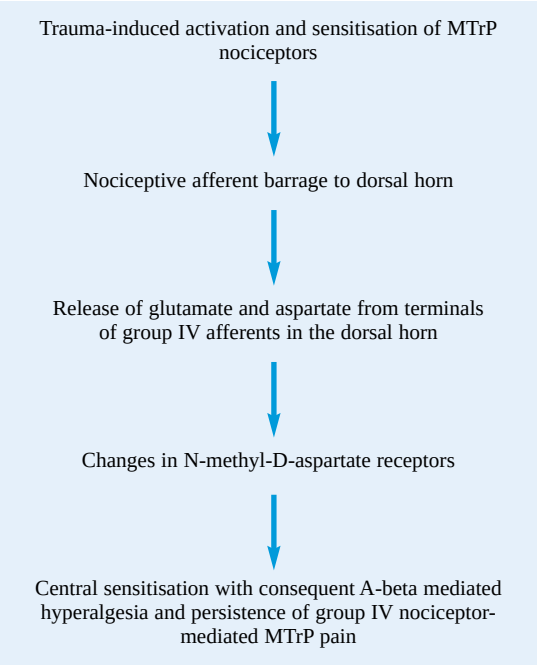

Figure 3 Dorsal horn neuronal plasticity (central sensitisation)

attention to the importance of recognising the presence of subclinical hypothyroidism, folic acid or iron deficiency in patients with myofascial pain syndrome, as, in his experience, a failure to correct any of these may cause MTrP activity to persist.[40]

Conclusion

In conclusion, whilst a variety of techniques appear to be efficacious in the treatment of MTrP pain, the author prefers to use SDN. Using this technique it is important to search for, and deactivate, all the relevant MTrPs. If successful, this approach minimizes the discomfort related to needling, and any post-needling soreness. If the response to SDN is inadequate, the practitioner may then use a more invasive approach, however, in the experience of the author, this is rarely necessary. If pain recurs frequently, or treatment effects are not sustained, the presence of MTrP reactivating factors should be considered.

Reference list

1. Lewis T (Sir). *Pain*. New York: Macmillan; 1942. p. 41.
2. Hong C-Z, Chen Y-N, Twehouse D, Hong DH. Pressure threshold for referred pain by compression on the trigger point and adjacent areas. *J Musculoskele Pain* 1996;4(3):61-79.
3. Baldry PE. *Myofascial Pain and Fibromyalgia Syndromes*. Edinburgh: Churchill Livingstone; 2001.
4. Russell IJ. Reliability of clinical assessment measures for the classification of myofascial pain syndrome. *J Musculoskele Pain* 1999;7(1/2):309-24.
5. Lu G-D, Needham J. *Celestial Lancets. A history and rationale of acupuncture and moxa*. Cambridge: Cambridge University Press; 1980. p. 127.
6. Carrubba RW, Bowers JZ. The Western World's first detailed treatise on acupuncture: Willem Ten Rhijne's De acupunctura. *J Hist Med Allied Sci* 1974;29(4):371-98.
7. Churchill JM. *A treatise on acupuncturation being a description of a surgical operation peculiar to the Japanese and Chinese and by them denominated zin-king, now introduced into European practice, with directions for it's performance and cases illustrating its success.* London: Simpkins and Marshall; 1821.
8. Churchill JM. *Cases illustrative of the immediate effects of acupuncturation in rheumatism, lumbago, sciatica, anomalous muscular diseases and in dropsy of the cellular tissue, selected from various sources and intended as an appendix to the author's treatise on the subject.* London: Callow and Wilson; 1828.
9. Osler W (Sir). *The principles and practice of medicine.* 8th ed. New York: Appleton; 1912. p. 1131.
10. Gowers WR. Lumbago: it's lessons and analogues. *BMJ* 1904;1:117-21.
11. Kellgren JH. Observations on referred pain arising from muscle. *Clin Sci* 1938;3:175-90.
12. Kellgren JH. A preliminary account of referred pains arising from muscle. *BMJ* 1938;1:325-7.
13. Edeiken J, Wolferth CC. Persistent pain in the shoulder region following myocardial infarct. *Am J Med Sci* 1936;191:201-10.
14. Steindler A. The interpretation of sciatic radiation and the syndrome of low back pain. *J Bone Joint Surg* 1940;22:28-34.
15. Travell J, Rinzler SH. The myofascial genesis of pain. *Postgrad Med* 1952;11:425-34.
16. Sola A.E, Kuitert JH. Myofascial trigger point pain in the neck and shoulder girdle. *N West Med* 1955;54:980-4.
17. Sola AE, Williams RL. Myofascial pain syndromes. *Neurology* 1956;6:91-5.
18. Frost P.A, Jessen B, Siggaard-Andersen J.A . Controlled double-blind comparison of mepivacaine injections versus saline injections for myofascial pain. *Lancet* 1980;1:499-501.
19. Bourne IHJ. Treatment of backache with local injections. *Practitioner* 1979;222:708-11.
20. Bourne IHJ. Treatment of chronic back pain comparing corticosteroid-lignocaine injections with lignocaine alone. *Practitioner* 1984;228:333-8.
21. Drewes AM, Andreasen A, Poulsen LH. Injection therapy for treatment of chronic myofascial pain: a double-blind study comparing corticosteroid versus diclofenac injections. *J Musculoskele Pain* 1993;1(3/4):289-94.
22. Cheshire WP, Abashian SW, Mann J.D. Botulinum toxin in the treatment of myofascial pain syndrome. *Pain* 1994;59(1):65-9.
23. Yue SK. Initial experience in the use of botulinum toxin A for the treatment of myofascial related muscle dysfunctions. *J Musculoskele Pain* 1995;3(suppl 1):22.
24. Hong C-Z. Considerations and recommendations regarding myofascial trigger point injections. *J Musculoskele Pain* 1994;2(1):29-59.
25. Lewit K. The needle effect in the relief of myofascial pain. *Pain* 1979;6(1):83-90.

26. Gunn CC. The Gunn *Approach to the treatment of chronic pain.* Edinburgh: Churchill Livingstone; 1998.
27. Chu J. Dry needling (intramuscular stimulation) in myofascial pain related to lumbosacral radiculopathy. *Eur J Phys Med Rehabil* 1995:5(4):106-21.
28. Chu J. Twitch-obtaining intramuscular stimulation (TOIMS): Effectiveness for long-term treatment of myofascial pain related cervical radiculopathy. *Arch Phys Med Rehabil* 1997;78(9):1042.
29. Chu J. Twitch-obtaining intramuscular stimulation. Observations in the management of radiculopathic chronic low-back pain. *J Musculoskele Pain* 1999;7(4):131-46.
30. 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(4):256-63.
31. Kraus H. The use of surface anaesthesia in the treatment of painful motion. *JAMA* 1941;116:2582-3.
32. Travell J. *Office Hours: day and night.* New York: The World Publishing Company: 1968.
33. Byrn C, Borenstein P, Linder LE. Treatment of neck and shoulder pain in whip-lash syndrome patients with intracutaneous sterile water injections. *Acta Anaesthesiol Scand* 1991;35(1):52-3.
34. Byrn C, Olsson I, Falkheden L, Lindh M, Hosterey U, Fogelberg M *et al*. Subcutaneous sterile water injections for chronic neck and shoulder pain following whiplash injuries. *Lancet* 1993;341(8843):449-52.
35. Macdonald AJ, Macrae KD, Master BR, Rubin AP. Superficial acupuncture in the relief of chronic low back pain. *Ann R Coll Surg Engl* 1983;65(1):44-6.
36. Bowsher D. Mechanisms of acupuncture. In: Filshie J, White A, editors. *Medical Acupuncture, A Western scientific approach.* Edinburgh: Churchill Livingstone; 1998. p. 69-82.
37. Pomeranz B. Acupuncture analgesia - basic research. In: Stux G, Hammerschlag R, editors. *Clinical Acupuncture. Scientific basis.* Berlin: Springer-Verlag; 2001. p. 7-8.
38. Karavis M. The neurophysiology of acupuncture: A viewpoint. *Acupunct Med* 1997:15(1);33-42.
39. Mann F. *Reinventing acupuncture.* Oxford: Butterworth-Heinemann; 1996. p. 32-41.
40. Gerwin R. The clinical assessment of myofascial pain. In: Turk DC, Melzack R, editors. *Handbook of pain assessment.* New York: Guildford Press; 1992. ch. 5.