Superficial Versus Deep Dry Needling
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Summary
Ninety percent of my patients with myofascial trigger point (MTrP) pain have this alone and are treated with superficial dry needling. Approximately 10% have concomitant MTrP pain and nerve root compression pain. These are treated with deep dry needling.

Superficial Dry Needling (SDN)
The activated and sensitised nociceptors of a MTrP cause it to be so exquisitely tender that firm pressure applied to it gives rise to a flexion withdrawal reflex (jump sign) and in some cases the utterance of an expletive (shout sign). The optimum strength of SDN at a MTrP site is the minimum necessary to abolish these two reactions. With respect to this patients are divided into strong, average and weak responders. The responsiveness of each individual is determined by trial and error. It is my practice to insert a needle (0.3mm x 30mm) into the tissues immediatelyoverlaying the MTrP to a depth of 5-10mm and to leave it in situ long enough for the two reactions to be abolished. For an average reactor this is about 30secs. For a weak reactor it is several minutes. And for a strong reactor the insertion of the needle and its immediate withdrawal is all that is required. Following treatment muscle stretching exercises should be carried out, and any steps taken to eliminate factors that might lead to the reactivation of the MTrPs.

Deep Dry Needling (DDN)
This in my practice is only used either when primary MTrP activity causes shortening of muscle sufficient enough to bring about compression of nerve roots. Or when there is nerve compression pain usually from spondylosis or disc prolapse and the secondary development of MTrP activity. Unlike SDN, DDN is a painful procedure and one which gives rise to much post-treatment soreness.

Keywords
Superficial dry needling, deep dry needling, myofascial trigger point pain.

Introduction
At the outset it has to be said that in the majority of my patients with myofascial trigger point (MTrP) pain superficial dry needling (SDN) is used, but in a small number of them deep dry needling (DDN) is employed.1-5 As it is the underlying pathophysiology that influences my choice in this matter, it is first necessary to consider what type of pain a patient with uncomplicated myofascial pain syndrome (MPS) suffers from. Is it of a nociceptive, neuropathic or nerve root compression type? In considering this question it is important to use these terms in their widely accepted sense; otherwise we are in danger of creating the type of confusion that prevailed at Alice in Wonderland’s Mad Hatter’s Tea Party, when those present idiosyncratically altered the meanings of words.

It is my submission that approximately 90% of my patients with MPS have primary somatogenic tissue damage-evoked MTrP nociceptive pain. Such pain has a persistent dull aching quality that is quite unlike the burning or electric shock-like sensation experienced by those with either neuropathic pain (i.e. pain due to dysfunction of the peripheral or central nervous systems) or nerve root compression pain (nociceptive nerve pain, nerve trunk pain, radiculopathic pain).

Although neuropathic pain must in part be due to the activation of nociceptors its distinctive quality is probably due, as Fields has pointed out, to disruption of the sensory apparatus, so that a normal pattern of neural activity is no longer transmitted to the perceptual centres.6 Similarly, although nerve root compression pain must be due to the activation of nociceptors in a nerve root’s coverings its radiation is readily distinguishable from that of MTrP nociceptive pain referral, because, unlike the latter, it is invariably along the length of the affected nerve.7 The remaining 10% of my patients with MPS have concomitant nociceptive nerve root compression pain and MTrP nociceptive pain.

Each of these two very different groups of patients will now be considered in turn.

Primary MTrP Nociceptive Pain
It is generally accepted that the commonest reason for MTrP activity arising, with consequent development of nociceptive pain, is trauma to muscle, brought about either because of direct injury to it, or as a result of it becoming overloaded. This MTrP activity develops either centrally, in the belly of a muscle, or at its peripheral attachments.

There is now compelling evidence to show that a MTrP is to be found at a site where a branch of a muscle’s motor nerve enters the muscle and terminates in a number of motor endplates.8 At this site there is also a neurovascular bundle, containing large and small sensory nerves, the latter having terminal nociceptors, and blood vessels with closely associated autonomic nerve fibres.

The tissue damage caused by the trauma leads to the release of a variety of different chemical substances. These include an excessive amount of acetylcholine from each motor endplate, with a resultant motor hyperactivity that leads to the development of a contraction knot. It is because of these knots that a palpable band is so often to be found at a MTrP site. Other chemical substances liberated include bradykinin, serotonin, histamine and potassium ions. These bring about the activation of nociceptors at Group IV sensory afferent terminals, with consequent development of MTrP nociceptive pain. Following this there is a release of prostaglandins, and these, together with bradykinin sensitize these nociceptors with a resultant lowering of their pain threshold.

The pain is therefore primarily nociceptive. However, the sensory afferent barrage set up by the activated MTrPs and sensitised Group IV nociceptors invariably leads, eventually, to neuroplastic changes in N-methyl D-aspartate (NMDA) receptors in dorsal horn neurones, with consequent development of central sensitisation. It is this secondary neuropathic change that is responsible for the ultimate chronicity of MTrP pain, and clearly the next great advance in its treatment will be the discovery of a substance like ketamine, without its undesirable side effects, that will act as an NMDA receptor antagonist.
It may therefore be seen that MTrP pain is primarily nociceptive, but, if present for more than a short time, invariably develops a neuropathic component.

**Superficial Dry Needling**

Because in 1979 the Czech physician Karel Lewit advocated the use of DDN for the deactivation of MTrPs I initially employed this technique routinely.9 However, when in the early 1980s a patient presented to me with pain down the arm as a result of MTrP activity in the scalenus anterior muscle, fearful of producing a needle-induced pneumothorax, I simply slid the needle under the skin at the MTrP site and left it in situ for a short time. On withdrawing the needle I found that both the exquisite tenderness at the MTrP site and the arm pain had disappeared. In view of this I tried out the SDN procedure in patients with MTrP activity in other muscles, including deeply situated ones, and found it to be equally successful. At about the same time Macdonald et al confirmed the efficacy of this technique in a controlled trial carried out on patients with lumbar MTrP pain.10 I have therefore, for the past 20 years, continued to use SDN for the treatment of primary MTrP nociceptive pain, and have taught the technique to a very large number of doctors and physiotherapists in courses held in various parts of the world.

It is interesting to note that SDN is not some new 20th century discovery. It was first described 2000 years ago in the book Huang Ti Nei Ching where, during the course of discussing the use of moxibustion, it says that if acupuncture is used the depth of the needling should be shallow, with the points employed being called fan ying tien (stimulus and response points), or Ya thung tien (pain-pressure points).11 Such points were clearly comparable to those which Sun Seu Mo in his book, also published in the 1st century, called ah shih points11 and which I now call 'jump and shout' MTrP points.4

**Determination of the Optimum Strength of the Superficial Dry Needling Stimulus**

As Felix Mann has pointed out, the responsiveness of patients to acupuncture is widely variable. The majority of people are average reactors.12 A minority however are either strong or weak reactors. For the acupuncture treatment of MTrP pain to be successful it is essential to establish the optimum strength of needle-induced MTrP nociceptor stimulation required for each individual patient. This is because a stimulus that is not sufficiently powerful to alleviate the MTrP pain of a weak reactor may be enough to cause a strong reactor to experience a distressing, albeit temporary, exacerbation of the pain.

A MTrP is of such exquisite tenderness that the application of firm pressure to it gives rise to a reflex flexion withdrawal (the ‘Jump’ sign), and often in addition the utterance of an expletive (the ‘Shout’ sign). Experience has shown that the optimum strength of needle stimulation required to alleviate an individual’s MTrP pain is the minimum amount necessary to abolish these two reactions.

In view of this it is my practice on first treating a patient to insert an acupuncture needle (0.3mm x 30mm) into the tissues overlying the MTrP to a depth of about 5-10mm and to leave it in situ for 30 seconds. The needle is then withdrawn, and pressure, equal to that exerted before treatment, is reapplied to the MTrP site to see whether the ‘Jump’ and ‘Shout’ reactions have been abolished. If not, the needle has to be reinserted and left in situ for several minutes. In some cases it prove necessary to increase the strength of stimulation still more by means of intermittently twirling the needle.

There is a small group of particularly strong reactors in which a stimulus applied for as short a time as 30 seconds proves to be more than is necessary. In this group all that is required is for the needle to be inserted into the superficial tissues overlying the MTrP and for it then to be immediately withdrawn.

Two groups of patients are, in my experience, invariably strong responders. These are migraineurs and sufferers from fibromyalgia.

**Modus Operandi**

Bowsher has explained that the effect of inserting a needle into the skin is to stimulate Aδ nerve fibres and by so doing to create activity in enkephalinergic inhibitory interneurons in the dorsal horn.13 It follows that when this latter activity is brought about, by needle-induced stimulation of these nerve fibres in the tissues overlying a MTrP, the effect is to block the centripetal transmission of nocuous information generated in the MTrP’s Group IV sensory afferent nociceptors. The needle-induced stimulation of the Aδ nerves is both an immediate one, and one lasting several hours, perhaps due to the setting up of a tissue injury-induced galvanic current.14

**Post - treatment procedures**

Firstly, it is necessary at the end of treatment to see whether or not it is possible to put the affected part through its full range of movements, in order to ascertain whether there is still any restriction, and also to determine whether there is any residual pain. If so it is essential to carry out a search for overlooked MTrPs. Secondly, it is important to teach the patient how to carry out muscle stretching exercises on a regular basis. Thirdly, it is essential, by means of history taking and examination, to identify any factors that might be liable to cause MTrP re-activation, and where possible to carry out measures to avoid this.

**Advantages of SDN**

My reasons for advocating the use of SDN for those with primary nociceptive MTrP pain are as follows:

From its successful use, in a very large number of patients with such pain, over a considerable number of years, there can be little or no doubt as to its effectiveness.

The procedure is very easily carried out.

In contrast to DDN it is a painless procedure other than for an initial short sharp prick.7 There is minimal risk of damage to nerves, blood vessels and other structures.1 Because of minimal bleeding there is a low incidence of post-treatment soreness.2

**Concomitant MTrP Nociceptive Pain and Nerve Root Compression Pain**

In my view DDN is mainly indicated for treatment of the minority of patients where there is concomitant MTrP nociceptive pain and nerve root compression pain.

There are two groups of such patients.
Patients in Group 1 have primary MTrP nociceptive pain and secondary radiculopathic pain that develops when a muscle, shortened as a result of MTrP activity, compresses nerve roots. Examples of this include MTrP-induced shortening of the pectoralis minor muscle giving rise to compression of roots of the brachial plexus, and a piriformis muscle, shortened for the same reason, exerting pressure on the sciatic nerve.

Patients in Group 2 have primary nerve root compression pain, usually brought about as a result of spinal spondylosis or disc prolapse, and the secondary development of MTrP nociceptive pain.

Deep Dry Needling (DDN)

Chu has made a special study of a group of patients with clinical and electromyographic evidence of multi-level spondylotic radiculopathy, complicated by the secondary development of MTrP pain.15-17 For this group she has developed a technique in which she obtains needle-evoked multiple local twitch responses at the MTrP sites, a procedure she has called twitch-obtaining intramuscular stimulation (TOIMS). This method of carrying out DDN in this particular group of patients has proved very effective.

It is my view that this and any of the other methods of carrying out DDN should be reserved for the relatively small number of patients in whom there is both MTrP and nerve root compression pain, and not used routinely for the treatment of uncomplicated MTrP nociceptive pain. My reason for saying this is because DDN gives rise to considerable treatment-evoked pain; it is liable to cause damage to neighbouring structures, particularly blood vessels, and the bleeding associated with the latter is responsible for the development of much troublesome posttreatment soreness.

Conclusion

For the reasons stated above I submit that SDN is the treatment of choice for the vast majority of patients who suffer from uncomplicated MTrP nociceptive pain, and that DDN should be reserved for the minority of those in whom there is concomitant MTrP and nerve root compression pain.

Reference list
