Superficial Dry Needling and Active Stretching in the Treatment of Myofascial Pain – A Randomised Controlled Trial

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Summary

A pragmatic, single blind, randomised, controlled trial was conducted to test the hypothesis that superficial dry needling (SDN) together with active stretching is more effective than stretching alone, or no treatment, in deactivating trigger points (TrPs) and reducing myofascial pain.

Forty patients with musculoskeletal pain, referred by GPs for physiotherapy, fulfilled inclusion / exclusion criteria for active TrPs. Subjects were randomised into three groups: group 1 (n=14) received superficial dry needling (SDN) and active stretching exercises (G1); group 2 (n=13) received stretching exercises alone (G2); and group 3 (n=13) were no treatment controls (G3). During the three-week intervention period for G1 and G2, the number of treatments varied according to the severity of the condition and subject/clinician availability. Assessment was carried out pre-intervention (M1), post-intervention (M2), and at a three-week follow up (M3). Outcome measures were the Short Form McGill Pain Questionnaire (SFMPQ) and Pressure Pain Threshold (PPT) of the primary TrP, using a Fischer algometer. Ninety-one per cent of assessments were blind to grouping.

At M2 there were no significant inter-group differences, but at M3, G1 demonstrated significantly improved SFMPQ versus G3 (p=0.043) and significantly improved PPT versus G2 (p=0.011). There were no differences between G2 and G3. The mean PPT and SFMPQ scores correlated significantly in G1 only, though no significant inter-group differences were demonstrated. Numbers of patients requiring further treatment following the trial were: 6 (G1); 12 (G2); 9 (G3). Conclusion: SDN followed by active stretching is more effective than stretching alone in deactivating TrPs (reducing their sensitivity to pressure), and more effective than no treatment in reducing subjective pain. Stretching without prior deactivation may increase TrP sensitivity.

Keywords

Myofascial trigger point; myofascial pain; acupuncture; superficial dry needling, active stretching exercise; randomised controlled trial.

Introduction

Myofascial Trigger Points (TrPs) are a common source of musculoskeletal pain presenting in primary care.[1] General practitioners frequently refer these patients to physiotherapy departments for treatment. Identifying TrPs requires training and clinical expertise.[2;3] Whilst British undergraduate medical and physiotherapy training does not include TrP identification, substantial literature[4;5] and postgraduate training are available. Despite this there is evidence that TrPs causing musculoskeletal pain often go undiagnosed by both doctors and physiotherapists, leading to chronic conditions. [6-10]

TrPs commonly arise from muscle overload, either as a result of acute strain/trauma, or of a more prolonged nature due to habitual postures or repetitive activities placing abnormal stresses on specific muscle groups.[4] It is thought that if normal healing does not occur, sensitisation of peripheral nociceptors by endogenous substances becomes prolonged, leading to increased local tenderness and referred pain. Sensitisation also takes place at spinal level, where receptive fields in the dorsal horn extend and become sensitive to lesser stimuli.[11] One hypothesis suggests that TrPs develop at motor end plates, where sensitisation of sensory and autonomic nerve endings leads to

excessive release of acetylcholine, preventing normal functioning of the calcium pump mechanism, resulting in sustained contraction of sarcomeres.[4] The contracted muscle fibres compress blood vessels, causing local hypoxia. An energy crisis ensues as the increased energy demand from sustained contraction cannot be met because of local hypoxia.[4]

Whilst universally accepted classification criteria have yet to be established, active TrPs are typified by tender spots in taut muscular bands, pressure on which reproduces the patient's pain in typical patterns for each TrP.[12] Local twitch responses (LTRs) may be elicited by snapping palpation. Spontaneous electrical activity (SEA) has been identified at minute loci of TrPs.[13-15] Macdonald found that both active and passive stretching of muscles containing TrPs, increased pain, whereas movements in which muscles worked as the agonist (isotonically) did not.[16] If resistance was applied to the muscle (isometric contraction) pain was again increased.

TrP deactivation is a term introduced by Baldry to describe the process whereby an active TrP becomes inactive i.e. local tenderness on palpation is resolved and the taut muscular band released, with ensuing symptomatic pain relief.[17] Various methods are used to deactivate TrPs, including ultrasound,[18-20] pressure release,[21;22] cold spray and stretch and injection of local anaesthetic.[4] Dry needling (no substance injected) may be carried out either superficially (SDN)[23] or with deeper insertion (DDN).[24;25]

Much discussion has ensued on the merits of DDN and SDN, though research evidence on the latter is limited.[26] An earlier study using SDN (to a depth of 4mm) on TrPs causing chronic low back pain, showed significant benefits for this method.[27] However study numbers were relatively small (8 SDN, 9 placebo TENS) and since electroacupuncture was introduced if no improvement was gained from SDN, it is impossible to assess the precise effects of needling. A recent RCT comparing the two methods demonstrated DDN to be significantly superior to SDN in reducing shoulder myofascial pain.[28] This study combined traditional acupuncture points with TrPs, using 13 needles at each of eight sessions, therefore its conclusions may not equate with individual TrP needling. A review of 23 studies of needling therapies for myofascial pain (mostly DDN) concluded that since no method demonstrated superiority, patient comfort should be a main consideration.[29]

Baldry claims minimal patient discomfort during 20 years' successful practice using SDN at 5-10mm depth.[23] In common with Baldry, one author (JE) experiences notable success in clinical practice in deactivating TrPs by SDN, needling to a depth of 4mm. It has been suggested that as the needle pierces the skin, A-delta nerve fibres are activated, resulting in inhibition of muscular C-fibres conveying pain from the TrP.[23] Subsequent relaxation of the TrP's taut muscular band enables the energy crisis at the motor end plate to resolve. Restoring the affected muscle to its full range of movement following TrP deactivation is an essential part of recovery, with three slow active stretches being recommended.[4;30]

With preceding factors in mind a pragmatic, randomised, single blind controlled trial was carried out to test the hypothesis that SDN together with active stretching is more effective than stretching alone, or no treatment, in deactivating TrPs and reducing myofascial pain.

Methods

The study took place during the five month period from July to Dec 2001. Following Local Research Ethics Committee approval, subjects were recruited from patients with musculoskeletal pain, referred for physiotherapy by five GPs at an inner city Lancaster practice. A small pilot study of one subject per group preceded the trial. This was useful for the clinicians to standardise algometry techniques and test worthiness of forms for recording measurements, to which minor adjustments were made. Inclusion criteria were: aged 18 and over; presence of active TrP identifiable by a) spot tenderness in a taut muscular band, b) subject recognition of pain on palpation, c) painful limitation of affected muscle's full range of movement, d) LTR, e) pain in expected distribution (a, b, c essential to inclusion; d and e not essential but used to confirm diagnosis); patient agrees not to receive additional treatment for their painful condition during the trial (apart from NSAIDs and pain

killers); patient is capable of complying with the trial. Exclusion criteria were: acute condition requiring treatment before six weeks; skin lesion, infection or inflammatory oedema at TrP site; needle phobia; previous adverse reaction to acupuncture or anaesthetic; serious neurological or systemic disorder. Subjects were randomised into three groups by selection of numbers 1 to 3 in sealed brown envelopes, when a witness was always present. Fourteen subjects were randomised into group 1 (G1) to receive superficial needling and active stretching exercise, 13 subjects to group 2 (G2) for stretching exercise, and 13 into group 3 (G3) who were no treatment controls.

Clinical Assessment

Subjects were given a full musculoskeletal physiotherapy assessment by JE, an experienced senior physiotherapist with training in TrP identification and a further two years' clinical experience. TrPs relevant to the patient's pain complaint were identified by palpation and marked on a body chart, recording precise measurements from bony landmarks if difficulties in ongoing identification were anticipated. A maximum of six TrPs relevant to the condition were included. In JE's experience this number is treatable in one session and allows for inclusion of satellite TrPs. The TrP most closely reproducing the patient's pain on palpation was measured for study purposes.

Interventions

Interventions were carried out by JE in the physiotherapy department attached to the practice.

Group 1

Patients received a course of SDN to affected TrPs, followed by appropriate stretching exercises to be continued at home. Exercises used were those recommended by Simons et al.[4;5] TrPs implicated in the condition were palpated and marked with a small dot on the skin at each treatment session, then needled in turn, usually working from proximal to distal. Sterile stainless steel acupuncture needles (25 x 0.30mm) with coiled copper handles and plastic guide tubes were used (Helio Medical Supplies, Inc.). The needle was inserted to the depth allowed by the guide tube (4mm). If not secured into the skin, further gentle pressure was applied, fractionally increasing penetration. The needle was not manipulated or stimulated and was left in situ until any sensations experienced by the patient, following needle insertion, had subsided. The duration of needle retention was recorded. The number of attendances over the three week treatment depended on the severity of condition and patient / therapist convenience, as is normal physiotherapy practice.

Group 2

Patients received instruction in appropriate stretching exercises, as recommended by Simons et al, for involved muscle(s) containing TrPs.[4;5] As with G1 patients they were asked to carry out home exercises, repeating three stretches three times daily. The importance of relaxing muscles between stretches was stressed. Follow up appointments were made to check / alter exercises according to the condition.

Groups 1 and 2

Both groups were advised on correction of daytime or sleeping postures if contributing to TrP activation. Following the three weeks' intervention, both groups had no treatment for three weeks, but continued with home regimes.

Group 3

Patients randomised to G3 received no treatment over the six week study period.

Outcomes

Outcome measures were the Short Form McGill Pain Questionnaire (SFMPQ) and pressure pain threshold (PPT). The SFMPQ included a visual analogue scale (VAS),[31] 15 pain descriptors, and a 1 to 5 present pain intensity scale, together giving a total pain score. PPT of the primary TrP was measured in kg/cm^2, using a Fischer algometer (Pain Diagnostics and Thermography, New York). The TrP was identified by JE and marked on the skin with a small pen mark. With the patient comfortably supported and relaxed the algometer was applied directly over the TrP, perpendicular to the body surface. Pressure was applied at the rate of 1kg/s and the patient was instructed to say '*now*' when the feeling of pressure turned to pain. Three readings were taken, allowing 30 to 60 seconds between each,

the average being the final score. Measurement took place at M1, pre-intervention; M2, after three weeks of the interventions in G1 and G2, or three weeks with no treatment in G3; and M3 after a follow-up period of a further three weeks. Measurements were carried out blind by two trained observers, the surgery's health care assistant and a research assistant employed by Morecambe Bay PCT. Both received prior training in use of the algometer. On some occasions neither assistant was available. This necessitated one of the authors (JE) taking 24% of measurements. As the pre-treatment M1 measures were still blind to grouping, 9% of measures were not blinded. At the end of the trial patients were asked if they required further treatment or physiotherapy for their condition.

Data Analysis

Data was analysed using SPSS Windows 10/11 programmes. Baseline characteristics of age, gender, pain duration, number and site of TrPs, were analysed using chi square or ANOVA tests to detect any differences between groups. SFMPQ and PPT data were examined visually and found to be reasonably normally distributed. Analysis was therefore carried out using parametric ANOVA and where this indicated significant overall difference ($p \geq 0.05$) two sample t-tests were performed. Ninety five percent confidence intervals were compared for mean outcome per group and used to perform significance tests at the 5% level of significance. Pearson's correlation coefficients between change in PPT and SFMPQ between M1 and M3 were calculated.

Table 1 **Group baseline characteristics, mean and (standard deviation).**

	Needling and Stretch	Stretch	Untreated Controls
	G1	G2	G3
N	14	13	13
Female %	71%	61%	76%
Age	57 (12)	55 (17)	57 (19)
Pain duration in months	16 (23)	10 (12)	16 (19)
No of TrPs involved	4.1 (1.2)	4.6 (1.1)	3.4 (1.6)
Upper body TrPs %	50%	84%	53%

Table 2 **Mean SFMPQ and PPT scores at M1, M2 and M3.**

	Needling and Stretch			Stretch			Untreated Controls		
	G1			G2			G3		
	M1	M2	M3	M1	M2	M3	M1	M2	M3
SFMPQ Mean (SD)	24.3 (6.3)	13.0 (10.2)	9.1 (11.6)	23.1 (7.0)	17.1 (9.4)	15.2 (8.8)	20.2 (8.0)	16.5 (10.2)	14.9 (11.0)
PPT Mean (SD)	1.4 (0.9)	1.8 (1.0)	2.7 (1.4)	1.7 (1.0)	1.8 (1.1)	1.8 (0.9)	1.4 (1.0)	2.0 (1.4)	2.0 (1.6)

Table 3 **Mean SFMPQ and PPT change scores at M2 and M3 with p values for inter group comparisons.**

	Needling and Stretch		Stretch		Untreated Controls	
	G1		G2		G3	
	M2-M1	M3-M1	M2-M1	M3-M1	M2-M1	M3-M1
SFMPQ Mean (SD)	-11.2 (11.3)	-15.2 (13.3)	-6.0 (7.0)	-7.8 (7.3)	-3.7 (7.6)	-5.3 (8.7)
	p<0.10 vs. G2 p<0.05 vs. G3					
PPT Mean (SD)	+0.5 (0.9)	+1.3 (1.0)	+0.1 (0.5)	+0.1 (0.6)	+0.6 (1.0)	+0.5 (1.3)
	p<0.05 vs. G2 p<0.10 vs. G3					

Two sample t-tests were performed where ANOVA indicated a significant overall difference, p values are p>0.05 unless otherwise indicated.

Table 4 **Treatment sessions during trial and further treatment.**

	G1 (n=14)	G2 (n=13)	G3 (n=13)
Mean number of treatment sessions during trial	4.6	2.9	0
Number of patients requiring treatment following trial	6	12	9

Results

Sixty-six patients were assessed for TrPs. Of these 40 fulfilled the inclusion criteria and agreed to take part in the study. All patients completed the trial. A total of 235 needle insertions were carried out on G1 patients. The average number per session was 3.7 (max 5.2, min 2.3). The needles were retained for an average of 3.4 minutes (max 4.8, min 2.5), this time corresponded to the duration of sensations experienced by the patient. Sixteen needles (7%) failed to produce any sensation.

Baseline characteristics (Table 1) showed no significant differences between groups. SFMPQ and PPT means at M1, M2 and M3 are shown in Table 2. Mean change scores at M2 and M3, with inter-group comparisons (Table 3), indicate no significant differences at M2. However, at M3, G1 was significantly different compared to G3 in SFMPQ scores (p=0.043), and compared to G2 in PPT scores (p=0.011). PPT and SFMPQ scores correlated significantly (in the expected direction) only in G1, though no significant difference in correlation coefficients was found between the groups.

The mean number of treatment sessions was lower for G2 (2.9) than for G1 (4.6) (Table 4). The number of patients requiring further treatment following the trial was considerably lower in G1 (Table 4). No side effects following TrP needling were reported by patients, or observed by the therapist. There were no dropouts from the study.

Discussion

Studies assessing methods of TrP deactivation are often single interventions on one TrP, considering only immediate effects.[18;19;24;32;33] It is accepted that several TrPs are commonly affected and may require a course of treatment.[4] Although only the primary TrP was measured by algometry, the present study delivered a course of treatment to up to six affected TrPs, over a three week period, allowing a further three weeks for follow up assessment. All patients completed the trial, which may be due to its pragmatic approach and assurance of physiotherapy on completion if required.

Sixty per cent of patients examined for the trial presented with myofascial pain caused by active TrPs. Prior GP screening for non-musculoskeletal pain in the present study, could account for lower figures (30%) in Skootsky et al's study, which examined 54 patients presenting with pain of 172 consecutive patients at a primary care centre.[1] Other baseline data compare favourably with Skootsky et al, indicating a representative sample.

The study's findings demonstrate no significant differences between groups after three weeks of treatment (M2), but after a further three weeks follow-up (M3) G1 showed significantly improved SFMPQ scores compared to G3 and significantly improved PPT scores compared to G2. This would seem to indicate that in the three week period following treatment, G1 patients continued to improve compared to patients in G2 and G3. Mean scores for SFMPQ and PPT (table 2) highlight this finding, particularly PPT scores, showing no improvement between M2 and M3 for G2 and G3. It can be seen that G2 patients showed less improvement overall in PPT, than the controls (table 2), and considerably more patients in G2 required treatment at the end of the trial (table 4). This may suggest that stretching alone has adverse effects on TrP sensitivity, supporting Macdonald's findings that stretching muscles containing TrPs is more likely to produce pain than when muscles contract isotonically as prime movers.[16] PPT was not measured in Macdonald's study.

Correlations between SFMPQ and PPT changes (M1 to M3) may also indicate that stretching without prior SDN increases TrP sensitivity. Only in G1 did the changes correlate significantly in the expected direction (i.e. a decrease in SFMPQ correlated with an increase in PPT). G2 scores showed least correlation (table 5). However, no significant difference in correlation coefficients was demonstrated between groups. Interestingly, Jaeger and Reeves, treating upper trapezius and levator scapulae TrPs with spray and stretch, found

Table 5 **Analysis of SFMPQ (M3-M1) vs PPT (M3-M1).**

	G1	G2	G3
Correlation coefficient	-0.67	-0.12	-0.37
95% confidence interval	-0.89 to 0.22	-0.63 to +0.46	-0.77 to +0.23
p value	0.009	0.71	0.22

No significant difference in slopes (p=0.12).

no significant correlation between pain and PPT scores, suggesting this was due to non treatment of other affected TrPs.[34]

Baseline characteristics affecting the TrP sensitivity in G2 may be a higher incidence of upper body TrPs (table 1), which have lower PPT scores than lower body TrPs, but this is not reflected in G2 scores at M1 (table 2).[35] Average pain duration was lower for G2 than G1 or G3 (table 1), which might be expected to correlate with a better outcome.[7;36] Although the treatment sessions in G2 were significantly fewer than in G1 (table 4), which might be considered to result in reduced non-specific effects (therapeutic interaction and expectation), Hopwood and Abrams, analysing 193 TrP injections, found that a successful outcome did not correlate with the number of treatments.[36]

PPT threshold measurements in this study indicate that SDN is an effective method of deactivating TrPs. The study would appear to show that active TrPs require deactivation prior to stretching, with probable release of taut muscular bands and resolution of the energy crisis at motor end plates.[4] Stretching muscles without prior deactivation of the TrP, may reduce subjective pain, but seems to have minimal effect on TrP sensitivity or overall resolution of the condition, as demonstrated in G2. Although PPT scores improved more in G1 than G3, this was not significant ($p<0.10$), therefore no treatment was shown to be as effective as SDN and stretch in reducing TrP sensitivity. However, since SDN with stretch was significantly more effective in reducing pain scores than no treatment, overall the study demonstrated greater benefits from SDN with stretch than either stretching alone or no treatment.

This study indicates that significant numbers of patients with musculoskeletal pain referred by general practitioners to physiotherapy departments may suffer from myofascial TrP pain. As TrPs are frequently located some distance from the pain, skill and training are required in order to detect them. SDN is a relatively quick and pain free method of TrP deactivation. It is thought to work by needle prick stimulation of Aδ fibres inhibiting of C fibre pain through descending mechanisms and dorsal horn interneurones.[23] SDN inevitably causes less discomfort to recipients than DDN methods.[24;25] There is also likely to be lower risk of traumatic side effects, particularly in anticoagulated patients. Since patient safety and comfort should be prime considerations in choice of needling therapy for myofascial pain,[29] SDN followed by active stretching is an appropriate treatment choice. Physiotherapists and general practitioners practising acupuncture are well placed to deliver this treatment, which if provided in a primary care setting, may well help prevent the development of chronic pain and dysfunction.

Study Limitations

Greater numbers and longer term follow up would have added weight to the study's findings. As nine percent of measurements were not blind to grouping, it is possible that bias was introduced. However, every effort was made to maintain impartiality, and previous scores were concealed during measurements. In addition, if measures fell between two scale indices (SFMPQ and PPT) the score in the opposite direction to expected bias was recorded.

Implications for Research

Since this study used SDN together with stretch, more research is needed to evaluate the effectiveness of SDN alone, and SDN compared with SDN and stretch. Also, the duration of TrP needling warrants further research.

Conclusion

This study supports the hypothesis that SDN followed by active stretching is more effective than stretching alone, or no treatment, in the management of myofascial pain caused by active TrPs. Stretching alone, in some cases, may

increase TrP sensitivity, leading to delayed resolution.

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Reference list

1. Skootsky SA, Jaeger B, Oye RK. Prevalence of myofascial pain in general internal medicine practice. *West J Med* 1989;151(2):157-60.
2. Gerwin RD, Shannon S, Hong CZ, Hubbard D, Gevirtz R. Interrater reliability in myofascial trigger point examination. *Pain* 1997;69(1-2):65-73.
3. Hsieh CY, Hong CZ, Adams AH, Platt KJ, Danielsen CD, Hoehler FK, Tobis JS. Interexaminer reliability of the palpation of trigger points in the trunk and lower limb muscles. *Arch Phys Med Rehabil* 2000;81(3):258-64.
4. Simons DG, Travell JG, Simons PT. *Travell & Simons' Myofascial Pain & Dysfunction. The Trigger Point Manual. Volume 1. Upper Half of Body.* 2nd ed. Baltimore: Williams & Wilkins; 1999.
5. Travell JG, Simons DG. *Myofascial Pain & Dysfunction. The Trigger Point Manual. Volume 2. The Lower Extremities.* Baltimore: Williams & Wilkins; 1992.
6. Ingber RS. Iliopsoas myofascial dysfunction: a treatable cause of 'failed' low back syndrome. *Arch Phys Med Rehabil* 1989;70(5):382-6.
7. Hong C-Z, Simons DG. Response to treatment for pectoralis minor myofascial pain syndrome after whiplash. *J Musculoskele Pain* 1993;1(1):89-131.
8. Ingram-Rice B. Carpal tunnel syndrome: more than a wrist problem. *J Bodywork Mov Ther* 1992;1(3):155-62.
9. Feinberg BI, Feinberg RA. Persistent pain after total knee arthroplasty: treatment with manual therapy and trigger point injections. *J Muscoloskele Pain* 1998;6(4):85-95.
10. Tay A, Chua K, Chan K-F. Upper quarter myofascial pain syndrome in Singapore: Characteristics and treatment. *J Musculoskele Pain* 2000;8(4):49-56.
11. Mense S. Nociception from skeletal muscle in relation to clinical muscle pain. *Pain* 1993;54(3):241-89.
12. Russell J. Reliability of clinical assessment measures for classification of myofascial pain syndrome. *J Musculoskele Pain* 1999;7(1-2):309-24.
13. Hubbard DR, Berkoff GM. Myofascial trigger points show spontaneous needle EMG activity. *Spine* 1993;18(13):1803-7.
14. Simons D, Hong C-Z, Simons LS. Prevalence of spontaneous electrical activity at trigger spots and at control sites in rabbit skeletal muscle. *J Musculoskele Pain* 1995;3(1):35-48.
15. Ward AA. Spontaneous electrical activity at combined acupuncture and myofascial trigger point sites. *Acupunct Med* 1996;14(2):75-9.
16. Macdonald AJ. Abnormally tender muscle regions and associated painful movements. *Pain* 1980;8(2):197-205.
17. Baldry PE. *Acupuncture, trigger points & musculoskeletal pain.* 2nd ed. Edinburgh: Churchill Livingstone; 1993.
18. Hong C-Z, Chen Y-C, Pon C,Yu J. Immediate effects of various physical modalities on pain threshold of active myofascial trigger points. *J Musculoskele Pain* 1993;1(2):37-53.
19. Lee J, Lin D, Hong C-Z .The effectiveness of simultaneous thermotherapy with ultrasound and electrotherapy with combined AC and DC current on the immediate pain relief of myofascial trigger points. *J Musculoskele Pain* 1997;5(1):81-91.
20. Gam AN, Warming S, Larsen LH, Jensen B, Hoydalsmo O, Allon I, et al. Treatment of myofascial trigger points with ultrasound combined with massage and exercise- a randomised controlled trial. *Pain 1998*;77(1):73-9.
21. Hanten WP, Olson SL, Butts NL, Nowicki AL. Effectiveness of a home program of ischemic pressure followed by sustained stretch for treatment of myofascial trigger points. *Phys Ther 2000*;80(10):997-1003.
22. Simons DG. Understanding effective treatments of myofascial trigger points. *J Bodywork Mov Ther* 2002;6(2):81-8.
23. Baldry PE. Superficial dry needling at myofascial trigger point sites. *J Musculoskele Pain* 1995;3(3):117-26.
24. Hong CZ. Lidocaine injection versus dry needling to myofascial trigger point. The importance of the local response. *Am J Phys Med Rehabil* 1994;73(4):256-63.
25. Gunn CC. *The Gunn Approach to the Treatment of Chronic Pain*. New York: Churchill Livingstone 1996.
26. Baldry PE. Superficial versus deep dry needling. *Acupunct Med* 2002;20(2-3):78-81.
27. 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.
28. Ceccheerelli F, Bordin M, Gagliardi G, Caravello M. Comparison between superficial and deep acupuncture in the treatment of the shoulder's myofascial pain: a randomised and controlled study. *Acupunct Electrother Res* 2001;26(4):229-38.
29. Cummings TM, White AR. Needling therapies in the management of myofascial trigger point pain: a systematic review. *Arch Phys Med Rehabil* 2001;82(7):986-92.
30. Hubbard DR. Persistent muscular pain: approaches to relieving trigger points. *J Musculoskele Med* 1998;15(5):16-26.
31. Melzack R. The short form McGill Pain Questionnaire. *Pain* 1987;30(2):191-7.
32. Hsueh TC, Cheng PT, Kuan TS, Hong CZ. The immediate effectiveness of electrical nerve stimulation and electrical muscle stimulation on myofascial trigger points. *Am J Phys Med Rehabil* 1997;76(6):471-6.
33. Hanten W, Barret M, Gillespie-Plesko M, Jump K, Olson S. Effects of active head retraction with retraction/ extension and occipital release on the pressure pain threshold of cervical and scapular trigger points. *Physiother Theory Pract* 1997;13:285-91.
34. Jaeger B, Reeves JL. Quantification of changes in myofascial trigger point sensitivity with the pressure algometer following passive stretch. *Pain* 1986;27(2):203-10.
35. Fischer AA. Pressure algometry over normal muscles. Standard values, validity and reproducibility of pressure threshold. *Pain* 1987;30(1):115-26.
36. Hopwood MB, Abram SE. Factors associated with failure of trigger point injections. *Clin J Pain* 1994;10(3):227-34.