



## Original Research—CME

# Dry Needling Alters Trigger Points in the Upper Trapezius Muscle and Reduces Pain in Subjects With Chronic Myofascial Pain

Lynn H. Gerber, MD, Jay Shah, MD, William Rosenberger, PhD,  
Kathryn Armstrong, DPT, Diego Turo, PhD, Paul Otto, BS, Juliana Heimur, BS,  
Nikki Thaker, BS, Siddhartha Sikdar, PhD

---

## Abstract

**Objective:** To determine whether dry needling of an active myofascial trigger point (MTrP) reduces pain and alters the status of the trigger point to either a non—spontaneously tender nodule or its resolution.

**Design:** A prospective, nonrandomized, controlled, interventional clinical study.

**Setting:** University campus.

**Participants:** A total of 56 subjects with neck or shoulder girdle pain of more than 3 months duration and active MTrPs were recruited from a campus-wide volunteer sample. Of these, 52 completed the study (23 male and 33 female). Their mean age was 35.8 years.

**Interventions:** Three weekly dry needling treatments of a single active MTrP.

**Main Outcome Measures:** Primary Outcomes: Baseline and posttreatment evaluations of pain using a verbal analogue scale, the Brief Pain Inventory, and the status of the MTrP as determined by digital palpation. Trigger points were rated as active (spontaneously painful), latent (requiring palpation to reproduce the characteristic pain), or resolved (no palpable nodule).

**Secondary Outcomes:** Profile of Mood States, Oswestry Disability Index, and Short Form 36 scores, and cervical range of motion.

**Results:** Primary outcomes: A total of 41 subjects had a change in trigger point status from active to latent or resolved, and 11 subjects had no change ( $P < .001$ ). Reduction in all pain scores was significant ( $P < .001$ ). Secondary outcomes: Significant improvement in posttreatment cervical rotational asymmetry in subjects as follows: unilateral/bilateral MTrPs ( $P = .001$  and  $P = .21$ , respectively); in pain pressure threshold in subjects with unilateral/bilateral MTrPs, ( $P = .006$  and  $P = .012$ , respectively); improvement in the SF-36 mental health and physical functioning subscale scores ( $P = .019$  and  $P = .03$ ), respectively; and a decrease in the Oswestry Disability Index score ( $P = .003$ ).

**Conclusions:** Dry needling reduces pain and changes MTrP status. Change in trigger point status is associated with a statistically and clinically significant reduction in pain. Reduction of pain is associated with improved mood, function, and level of disability.

---

## Introduction

Myofascial pain syndrome (MPS) is a common and significant clinical problem, accounting for 15% of general medical visits [1]. MPS negatively affects function and participation in life activities [2,3].

MPS has generated controversy in part because there has been disagreement about diagnostic criteria. The syndrome has had many names, including fibrositis, myofasciitis, and myogelosis [4,5], reflecting a lack of agreement about etiology, pathophysiology, and the primary tissue involved. MPS has been confused with other pain syndromes such as

fibromyalgia and neuropathic pain, and although confusion remains, there is general acceptance of the term “myofascial pain syndrome” and its diagnostic components [6-8].

There is active debate about whether the myofascial trigger point (MTrP) is a necessary condition for the diagnosis of MPS, and whether it should be the target for pain relief. This article explored this relationship in part because there seems to be agreement that the MTrP is an objective finding associated with MPS that is reliably identified and useful in assessing pain [9-12].

In this study, we used Travell and Simons’ definition of MPS, namely a regional pain syndrome in which there

is a palpable, discrete nodule within a taut band of skeletal muscle that is spontaneously painful [9,10]. This is referred to as an active trigger point (a-MTrP), defined as a spontaneously painful nodule. A latent myofascial trigger point (l-MTrP) is a trigger point that is not spontaneously painful and that requires palpation or motion/activity to induce pain.

Dry needling is a nonpharmacological treatment for MPS that is commonly used for reducing pain associated with a-MTrPs [13,14]. It is frequently performed by a clinician using a 32-gauge acupuncture needle inserted into the palpably painful nodule using a superficial (10–20 mm) or deep (25–40 mm) needling technique. Elicitation of 1 or more local twitch responses is a goal of dry needling and often benefits individuals with pain secondary to MTrPs [3].

The effectiveness of dry needling has been difficult to demonstrate due to a lack of objective measures of pain. Currently, assessment of patients with MPS relies upon patient self-reports of pain. Patient-reported outcomes (PROs) are reliable measures, but their sensitivity to change, the variety of ways of expressing pain by individual patients, correlations with physical findings, and other objective measures have made validation difficult.

Our research team used the status of the MTrP as the treatment target and an outcome measure to assess the changes that resulted from treatment and to determine whether change in its status correlated with change in posttreatment level of pain.

This article presents the results of a prospective, interventional clinical study designed to assess whether dry needling of an a-MTrP alters patient-reported pain and contemporaneously alters the status of the trigger point. We selected a technique that is widely used in clinical practice and that has been shown to be effective in reducing MPS, but effect of which on the MTrP is not known [3,13,14]. We also measured the impact of dry needling on self-reports of mood and function.

To our knowledge, this is the first study to investigate the association between dry needling and its effect on pain reduction and MTrP status.

## Methods

The study was approved by the Chesapeake Institutional Review Board. Subjects were recruited by posting flyers around a university community. No remuneration was offered to participants. All provided consent.

Study entry required that participants were adult (aged 18–65 years) and had experienced pain without provocation for at least 3 months in the neck/shoulder girdle region and a palpable MTrP in 1 or both of the specific locations of the upper trapezius. The spontaneous pain had to be in the area of the prescribed MTrP locations, and its palpation had to exacerbate pain. Radiation to head, neck, or face on palpation was acceptable but was not required

for inclusion. All evaluations and treatments were performed by 2 experienced clinicians, each with more than 20 years of treatment experience. Patients selected which day of the week was preferable for treatment and follow-up, and were assigned to the physician who treated on a specific day of the week. That is, physician 1 treated on Fridays and physician 2 on Thursdays. Occasionally, patients were seen on the alternative day if scheduling required a change.

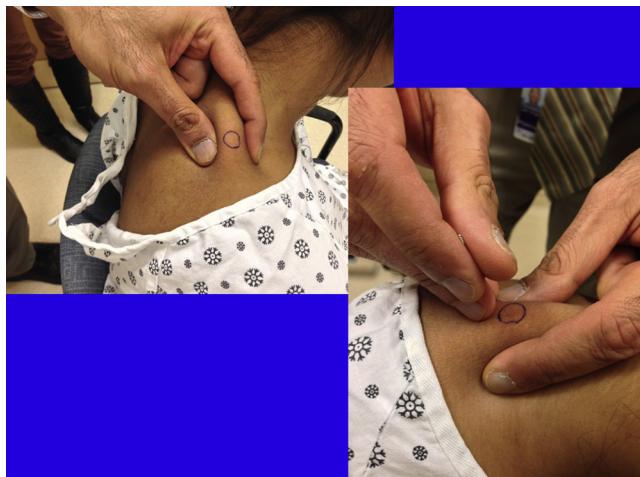
Interobserver reliability for the 2 treating physicians was tested using 14 treatment-naïve volunteers with and without pain. Each provided informed consent for evaluation. Two sites were examined independently by each of the two examiners and scored as active, latent, or nonpainful nodule/normal. Interrater reliability was assessed using a  $\kappa$  statistic. The  $\kappa$  statistic for site 2 is 0.74 ( $P = .003$ ) and for site 3 is 0.87 ( $P < .001$ ).

Entry exclusions included the following: chronic fatigue syndrome, fibromyalgia, chronic Lyme disease, cervical radiculopathy, head/neck/shoulder girdle surgeries, new medication or change within 6 weeks, and current use of acupuncture.

All study subjects received 3 successive dry needling sessions weekly. Posttreatment evaluations were performed at 3 weeks. Treatment technique was standardized as follows: 4 predetermined examination areas were palpated and point(s) were identified [2]. They were 2 cm medial to the acromioclavicular joint on the left and right sides and at 2 additional sites in the upper trapezius as it turns cephalad lateral to the spinous process of C7. Trigger points reported to be spontaneously painful were considered to be a-MTrP; those not spontaneously painful but painful upon palpation were designated as l-MTrPs. Only 1 a-MTrP was selected for treatment. If there was more than 1 a-MTrP, we selected the most symptomatic site for dry needling. Hence, there may have been untreated a-MTrPs.

Some subjects had a-MTrPs on only 1 side, which we defined as "unilateral." Some subjects had at least 1 a-MTrP on each side, which we defined as "bilateral." We defined "responders" as patients whose status changed from a-MTrP to l-MTrP, or a-MTrP to an asymptomatic palpable nodule or no nodule palpable. "Non-responders" were those whose a-MTrP remained active (spontaneously painful). This status was determined by a treating physician (not always the one who performed the dry needling treatment) who palpated and assessed whether the findings were consistent with a-MTrP, l-MTrP, nonpainful nodules, or no palpable nodule.

The selected a-MTrP was prepared by wiping the area with an alcohol pad, and a 32-gauge needle with its plastic guide tube in place was placed over the a-MTrP (Figure 1). A tapping motion was used to advance the needle. Occasionally, needle movement was performed around the nodule following a 4-points-of-compass technique with rotation along its long axis in an effort to elicit a small muscle twitch. This was achieved in



**Figure 1.** Demonstration of needle insertion into myofascial trigger point.

approximately 70% of subjects on the first, 66% on the second, and 50% on the third treatment. Change in verbal analogue scale (VAS) score was not statistically correlated with eliciting the twitch response.

All evaluations were performed at baseline and after the third treatment at 3 weeks. Primary outcomes were measures of pain reduction and change in trigger point status from a-MTrP to either l-MTrP or no palpable nodule. A VAS was used for pain assessment. It was scored from 0 to 10 (0 = no pain, 10 = unbearable pain). The question was asked as follows: "Are you having pain now? Please rate it on a scale of 0-10. Do you have pain on the right side of your neck? Please rate this 0-10. Do you have pain on the left side of your neck? Please rate this 0-10." Palpation was performed on 4 standard sites. Nodules were either active (spontaneously painful), latent (required overpressure to elicit pain), or not palpable (and no pain associated with palpation).

Secondary outcomes included range of motion (ROM) which was determined in 3 planes of movement (flexion/extension, side bending, and rotation) using the Deluxe Cervical Range of Motion Instrument (CROM), model 12-1156 (Fabrication Enterprises, White Plains, NY). A ratio of measures of ROM over the normal range was determined for the left and right sides. The asymmetry was evaluated at baseline and at the end of treatment (3 weeks). Two additional measures of pain included a measure of pain pressure threshold (PPT) and the Brief Pain Inventory (BPI) [15]. PPT was obtained at 4 sites, following a standard procedure for assessing relative comparisons among the anatomical sites using a pressure algometer (Commander Algometer, Tech Medical, Salt Lake City, UT; <http://www.jtechmedical.com/Commander/commander-algometer>) (Figure 2). Subjects were instructed to identify the moment at which symptoms underwent a qualitative shift from pressure to pain during algometer compression. The reading at that time was determined to be the PPT score. A high



**Figure 2.** Algometer used for measuring pain pressure threshold (Tech Medical, Salt Lake City, UT).

score, namely, that which requires more pressure to be applied to produce pain, was associated with improved pain symptoms.

Additional measures included the Oswestry Disability Index, a measure of disability secondary to the spine and adjacent musculoskeletal system. Subjects were instructed to reply with reference to the neck and upper thoracic area in terms of limitations [16]. The MOS 36-Item Short-Form Health Survey (SF-36), a health status questionnaire [17] was used, as well as a short version of the Profile of Mood States (POMS) [18], a symptom checklist of mood that included such items as anxiety and depressive symptoms. Subjects with high scores on the Oswestry Disability Index, POMS, and VAS were considered to be more symptomatic or more disabled. A high score on the SF-36 was considered to indicate better health status.

Sample size was determined to be 90 subjects, with the assumption that 5% of patients would spontaneously improve their MTrP status without dry needling. We wished to detect an increase of 10% for responders post-treatment. We conducted a conditional power analysis after 56 patients were accrued and determined the study to be substantially overpowered, and our hypothesized percentage of responders was underestimated [19].

StatXact [20] was used to conduct an exact binomial test that the percentage of responders exceeded 5% at the 0.05 (2-sided)  $\alpha$  level. Paired *t*-tests compared pain and variables of interest before and after treatment. These variables included both objective and self-reported outcomes. Analysis of covariance was used to detect changes in outcome measures for responders versus nonresponders.

Changes from baseline in VAS, BPI, and PPT scores were analyzed, and were adjusted for baseline value,

age, gender, group (unilateral/bilateral), and exercise status, based on response to treatment, using regression analysis. For all models, studentized residual plots were inspected. For VAS scores and BPI scores, the residuals appeared homoscedastic with no outliers. For PPT scores, 1 subject was considered as an outlier. A Q-Q plot of residuals exhibited no indication of nonnormality.

Each model was adjusted for gender, age, and exercise status, and none of these characteristics was significant in any of the models. Regression diagnostics were graphically depicted, including checks for outliers and heteroscedasticity, and Q-Q plots to verify the normal error assumption. There were no outliers, and no transformations were deemed necessary. All regression analyses were conducted using SAS software, version 9.3 (SAS Institute, Cary, NC).

## Results

In all, 52 subjects were included in the study. A total of 56 were originally eligible and underwent study baseline procedures. Two subjects did not complete 3 weekly dry needling sessions and dropped out for unknown reasons. One subject started new treatments after the first study treatment, and 1 subject did not have complete follow-up data for analysis. **Table 1** presents the distribution of the descriptive variables and a summary of treatments that subjects had selected for their pain before study entry.

**Table 2** presents the frequencies for the primary outcome in bilateral and unilateral groups, respectively. There were 41 responders and 11 nonresponders

**Table 1**  
Characteristics of study subjects

Characteristic	n	Active Myofascial Trigger Points
Gender		
Male	23	41.1
Female	33	58.9
Age, y		
Mean (range)	35.8 (20-62)	
Pain distribution		
Bilateral	42	75
Unilateral (right/left)	9/5	16.1/8.9
Pain duration, y		
<3	21	37.5
>3	35	62.5
Use of medication		
Analgesic	37	66
Mood	11	19.6
Sleep	1	1.8
Opioid/Narcotic	0	0
Supplements/vitamins	30	53.6
Use of nonpharmacological treatment		
Exercise	43	76.6
Physical Modalities (heat, cold, electrical stimulation)	32	57
Massage	17	30
Chiropractic	8	14

( $P < .001$ ). A conditional power analysis was conducted. Under the current trend of the data, under the hypothetical trend of the data, and under the null hypothesis, the conditional power was 1, meaning that there was no positive probability of a nonsignificant result using the full sample size.

**Table 3** presents baseline and follow-up characteristics for physical findings, pain, and self-reports. We measured a significant improvement in rotational asymmetry in both the unilateral and bilateral groups ( $P = .001$  and  $P = .021$ , respectively). ROM extension and flexion had not improved. There was a significant change in side-bending ROM in the unilateral group only ( $P = .001$ ) and a significant improvement in PPT at the treated site in both groups ( $P = .006$  and  $P = .012$ , respectively). The baseline and follow-up characteristics for pain measurements and self-reports showed a significant reduction in BPI scores ( $P < .001$ ). There was a significant reduction in VAS on the treated side in both the unilateral and bilateral groups ( $P < .001$ ), and on the untreated side only in the bilateral group ( $P < .001$ ). There was a significant increase in the SF-36 pain subscale score ( $P = .002$ ) and a decrease in the POMS tension and mood scores ( $P = .012$  and  $P = .013$ , respectively). These represented improvements. There was significant improvement in the scores of the SF-36 mental health and physical functioning subscales ( $P = .019$  and  $P = .03$ , respectively) and the Oswestry Disability Index scores ( $P = .003$ ). The regression model was significant for VAS scores (model  $F = 32.37$ ,  $P < .001$ ,  $R^2 = 0.81$ ,  $n = 52$ ). Baseline values for VAS were also significant ( $P < .001$ ). Other adjustment variables were not significant. For BPI scores, the regression model was marginally significant (model  $F = 2.36$ ,  $P = .047$ ,  $R^2 = 0.25$ ,  $n = 49$ ). For PPT, the regression model was not significant (model  $F = 2.13$ ,  $P = .069$ ,  $R^2 = 0.22$ ,  $n = 51$ ). Only baseline PPT was significant in the model.

**Table 4** presents the least-squares means (standard errors) for change from baseline in VAS, BPI, and PPT among responders and nonresponders from the adjusted regression models. The mean change from baseline in VAS score was  $-2.87 \pm 0.16$  for responders and  $-1.00 \pm 0.30$  for nonresponders. The means were significantly different ( $P < .001$ ). The mean change from baseline in BPI score was  $-1.32 \pm 0.22$  for responders and  $0.04 \pm 0.38$  for nonresponders. The means were significantly different ( $P = .002$ ). The mean change from baseline in PPT was not statistically significantly different in responders and nonresponders.

**Table 2**  
Primary outcome for treated subjects with bilateral and unilateral active trigger points

Bilateral Active Trigger Points			Unilateral Active Trigger Points		
Baseline	Follow-up	Count	Baseline	Follow-up	Count
Active	Active	7	Active	Active	4
Active	Latent	12	Active	Latent	14
Active	Normal	6	Active	Normal	9

**Table 3**Baseline and follow-up characteristics: Physical findings, pain and self-reported outcomes (mean  $\pm$  SD)

Characteristic	n	Baseline	Follow-up	P value
<b>Physical finding</b>				
Cervical ROM extension (°)	51	73.8 $\pm$ 12.8	74.3 $\pm$ 12.0	.741
Cervical ROM flexion (°)	51	55.2 $\pm$ 11.0	57.1 $\pm$ 8.3	.192
Rotation asymmetry unilateral (°)	27	8.1 $\pm$ 6.3	3.1 $\pm$ 5.4	.001
Rotation asymmetry bilateral (°)	24	5.4 $\pm$ 4.4	2.4 $\pm$ 3.2	.021
Side bending unilateral (°)	27	5.6 $\pm$ 3.8	2.7 $\pm$ 2.9	.001
Side bending bilateral (°)	24	5.5 $\pm$ 6.4	3.1 $\pm$ 3.2	.109
PPT treated site unilateral (lb)	27	7.6 $\pm$ 3.3	9.4 $\pm$ 3.7	.006
PPT treated site bilateral (lb)	24	6.7 $\pm$ 3.0	8.4 $\pm$ 3.1	.012
<b>Pain (scores)</b>				
BPI	49	3.4 $\pm$ 1.6	2.3 $\pm$ 1.9	<.001
VAS treated side unilateral	27	3.5 $\pm$ 2.4	0.9 $\pm$ 1.3	<.001
VAS treated side bilateral	25	3.0 $\pm$ 1.4	0.9 $\pm$ 1.2	<.001
VAS untreated side unilateral	27	1.0 $\pm$ 1.9	0.4 $\pm$ 1.1	.203
VAS untreated side bilateral	25	2.6 $\pm$ 1.2	0.9 $\pm$ 1.2	<.001
SF-36 pain	50	62.5 $\pm$ 18.4	69.3 $\pm$ 16.5	.002
<b>Self-reported outcomes</b>				
POMS confusion	49	0.28 $\pm$ 0.39	0.23 $\pm$ 0.35	.418
POMS depression	49	0.11 $\pm$ 0.23	0.07 $\pm$ 0.18	.151
POMS fatigue	49	0.77 $\pm$ 0.81	0.54 $\pm$ 0.69	.056
POMS tension	49	0.47 $\pm$ 0.50	0.28 $\pm$ 0.33	.012
POMS mood	49	0.29 $\pm$ 1.91	-0.38 $\pm$ 1.79	.013
POMS vigor	49	1.49 $\pm$ 0.94	1.58 $\pm$ 0.93	.261
POMS anger	49	0.15 $\pm$ 0.35	0.08 $\pm$ 0.27	.12
SF-36 general health	50	76.9 $\pm$ 19.1	76.8 $\pm$ 18.6	.913
SF-36 mental health	50	75.9 $\pm$ 11.8	79.1 $\pm$ 11.4	.017
SF-36 physical functioning	50	88.5 $\pm$ 14.3	91.4 $\pm$ 11.3	.03
SF-36 emotional	50	83.4 $\pm$ 21.5	88.8 $\pm$ 16.3	.051
SF-36 physical role	50	85.1 $\pm$ 17.0	86.9 $\pm$ 16.7	.471
SF-36 social functioning	50	87.8 $\pm$ 16.9	89.7 $\pm$ 15.9	.253
SF-36 vitality	50	58.7 $\pm$ 17.0	60.7 $\pm$ 16.9	.258
Oswestry Disability Index score	50	10.8 $\pm$ 6.0	8.5 $\pm$ 7.1	.004

BPI = Brief Pain Inventory; PPT = pressure pain threshold; POMS = Profile of Mood States; ROM = range of motion; SF-36 = MOS 36-Item Short-Form Health Survey; VAS = verbal analogue scale.

## Discussion

Much has been written about MTrPs and their possible relationship to MPS [21-24]. The contribution of the MTrP in the pathogenesis of MPS is an area of active investigation and has raised important questions about muscle and fascia in inciting and perpetuating soft-tissue pain [25-27]. Debate continues as to whether the MTrP is necessary for MPS diagnosis and whether it needs to be the target of treatment.

The pathogenesis of the MTrP is elusive, and current explanations about its relationship to MPS remain

incomplete. Trauma, muscle overload, and muscle overuse have been cited as etiologic agents, with trauma being 1 of the leading contenders [24,25]. Tissue injuries result in the release of noxious substances that bind to, sensitize, and/or activate nociceptors. This leads to the transmission of signals that indicate tissue damage and inflammation, and may set up persistent pain states [26]. The relative contributions of the central and peripheral nervous systems in generating and perpetuating pain are not yet fully understood, although there is preliminary evidence for pain dysregulation in MPS [28-30]. Disrupted descending inhibition in individuals with chronic musculoskeletal pain may lead to a muscle pain complaint, irrespective of peripheral tissue damage [30].

To explore relationships between MTrPs and MPS, we reasoned that if treatment directed at the MTrP was shown to improve myofascial pain [3,14,31-34], we could measure changes in pain and MTrP status at the same time. We elected to use a single MTrP in a defined anatomical area and to use experienced "calibrated" examiners to study it carefully. The examiners participated in a test of interrater reliability that demonstrated no statistically significant differences between

**Table 4**

Change from baseline on VAS, BPI, and PPT

	VAS score	BPI score	PPT (lb)
Responders	-2.87 $\pm$ 0.16	-1.32 $\pm$ 0.22	2.12 $\pm$ 0.50
Nonresponders	-1.00 $\pm$ 0.30	0.04 $\pm$ 0.38	0.85 $\pm$ 0.96

Data are least-squares means  $\pm$  standard errors of change from baseline of VAS, BPI, and PPT, and are adjusted for baseline, site, gender, age, and exercise status.

VAS = verbal analogue scale; BPI = Brief Pain Inventory; PPT = pain pressure threshold.

their clinical assessments. This approach would provide an opportunity to assess pain related to the MTrP and would allow us to determine the relationship, if any, between pain reduction and MTrP status change. We used objective measures of the MTrP (palpation and size) [35], and correlated these with patient self-reports of pain, mood, health status, and disability. One review article addressing the reliability of palpation suggests that it varies widely [34]. However, none of these 9 studies used examiners who had demonstrated inter-rater reliability and performed evaluation and treatment on a single muscle.

Our major findings were that pain reduction, as measured using all 3 of the pain assessments, is significantly correlated with change in the MTrP status as determined by MTrP palpation from active to latent or normal (no palpable nodule) after dry needling. We noted that there was a clinically significant improvement in pain scores (a drop of  $\geq 2$ ) on the VAS [36]. Treatment was correlated with a significant, clinically relevant reduction in pain compared with baseline values, as well as improvements in mood and function. Needling was also positively correlated with a significant increase in cervical ROM attributable to the upper trapezius (i.e., side bending and rotation). There was a significant decrease in asymmetry between the left and right sides after treatment.

We are aware of some concerns about the reliability of pain measures and therefore used 3 instruments. One of these, the PPT, is an instrumented measure. All showed significant reduction after treatment.

The mean baseline measurement of pain for subjects with unilateral MTrP was VAS 3.5 ( $\pm SD$  2.4), which is considered moderate pain [37]. The group with bilateral MTrPs had VAS score of 3.0 ( $\pm 1.4$ ), indicating mild pain. Some clinicians may not wish to treat MTrP and myofascial pain if the level is mild. The decision to treat often depends upon several factors, including frequency and persistence, intrusion into daily activities, and peak pain levels. The measure at baseline was determined at a moment in time, and the entry criterion was reportable pain; the clinical severity did not determine whether the research subject was to receive dry needling. After criteria were met, our primary outcome was a change in pain score, and the change was significant.

Appropriate measurement is critical to ensure the validity and reliability of this clinical study. Pain evaluations are not objective assessments, and consensus about which pain assessment tools are best to use for this study group has not been reached. This study used standard, systematically applied, and frequently used evaluations to assess patients with MPS. We used the BPI and algometry to assess the level and nature of pain. Although both the BPI and VAS measured pain intensity at the time of administration, the BPI also measured the impact of pain on daily functions, pain relief, pain quality, and the patient's perception of the cause of the

pain. The statistical analysis showed that VAS and BPI adjusted means scores were significantly different after treatment, and that PPT scores were not. In the regression model, VAS was significant, BPI was marginally significant, and PPT was not. We support the use of the VAS for assessment of treatment of MPS. PPT may be useful and has been shown to be reliable in evaluating MPS, but it lacks sensitivity [38]. In this study, we have defined a positive response to treatment as a statistically significant decrease in pain from baseline and improvement in MTrP status. The change was also clinically significant, that is a decrease in 2 points on the VAS.

We recommend a careful, systematic, and comprehensive approach to the evaluation of patients with MPS. This approach should include objective measures of cervical spine ROM, trigger point palpation and self-reports of pain, fatigue, mood, disability and health status, which have been shown to be sensitive to change and to provide important information about the impact of MPS on issues of importance to patients.

One review examined the level of evidence for dry needling in MPS [14]. The authors identified the data as level 1a because the reports were randomized, placebo-controlled trials. The outcomes were self-reports, did not include objective measures, and did not link response to trigger point status.

To the best of our knowledge, this is the first report to demonstrate that there is a significant, contemporaneous change in the level of both pain and the status of the MTrP after dry needling. Dry needling is likely to provide pain reduction and resolution of the a-MTrP. We report that dry needling has a significant effect in reducing pain as measured by VAS, BPI, and PPT; and in decreasing disability as measured by the Oswestry Disability Index in individuals with MPS and a-MTrPs. A randomized, placebo-controlled, blinded trial is the gold standard and is required to definitively demonstrate effectiveness. Our group is planning to conduct such a trial.

There are some limitations to this study. MPS has long been considered a local or regional pain syndrome, implying that the inciting factors for pain are local rather than resulting from central sensitization [39,40]. This study did not address this question. The results of this study do not answer questions about pathogenesis, etiology, and relative contributions of various regulatory mechanisms for developing or resolving MPS or MTrPs. However, the data advance our understanding of this complex syndrome by linking improvement in symptoms with objective measures of MTrP and establish a relationship between MTrP and MPS.

Subjects for this study were recruited on a university campus, and possibly represent an atypical cross-section of people with MPS. Nonetheless, computer-based activity is used by most of our subjects, and has been reported to be a significant risk factor for developing MPS [41].

Finally, this study was not a randomized, placebo-controlled, blinded clinical trial; hence it cannot prove

effectiveness. The treating clinicians also evaluated the subjects, creating potential bias despite their being experienced and standardizing their technique [42,43]. The 2 treating physicians evaluated and treated the subjects based on scheduling convenience, and any bias introduced as a result cannot be ruled out. Not all subjects responded with a twitch to the dry needling. Some investigators believe that this is an important part of the therapeutic effect [3,42]. The elicitation of the twitch response did not distinguish the responders from the nonresponders in this study.

This study was also had advantages. The study was a carefully conducted, systematic prospective study using valid instruments designed to measure soft tissue pain and disability to which objective measures were also applied. This permitted us to develop a properly sized and designed clinical effectiveness trial for dry needling using self-reported outcomes and objective measures.

## Conclusion

A 3-week course of dry needling had a significant effect on pain reduction in MPS. Pain reduction was significantly related to change in trigger point status from active (spontaneously painful) to latent or resolution. Importantly, pain reduction was significantly correlated with improvement in cervical spine side bending and rotation, in patient self-reports of improved physical and emotional well-being and mood; and reduction in disability.

## References

1. Skootsky SA, Jaeger B, Oye RK. Prevalence of myofascial pain in general internal medicine practice. *West J Med* 1989;151:157-160.
2. Gerber LH, Sikdar S, Armstrong K, et al. A systematic comparison between subjects with no pain and pain associated with active myofascial trigger points. *PM R* 2013;5:931-938.
3. Tekin L, Akarsu S, Durmuş O, Cakar E, Dinçer U, Kıralkı MZ. The effect of dry needling in the treatment of myofascial pain syndrome: A randomized double-blinded placebo-controlled trial. *Clin Rheumatol* 2013;32:309-315.
4. Llewellyn LJ. A discussion of fibrosis. *Proc R Soc Med* 1913; 6:27-35.
5. Schade H. Untersuchungen in der Erkältungsträge: III. Über den Rheumatismus, insbesondere den Muskelrheumatismus (myogelose). *Münch Med Wochenschr* 1921;68:95-99.
6. Travell JG, Simons DG. Myofascial Pain and Dysfunction: The Trigger Point Manual. Baltimore, MD: Williams & Wilkins; 1983.
7. Borg-Stein J, Simons DG. Focused review: Myofascial pain. *Arch Phys Med Rehabil* 2002;83(3 Suppl 1):S40-S49.
8. Bennett R. Myofascial pain syndromes and their evaluation. *Best Pract Res Clin Rheumatol* 2007;21:427-445.
9. Simons DG. Review of enigmatic MTrPs as a common cause of enigmatic musculoskeletal pain and dysfunction. *J Electromyogr Kinesiol* 2004;14:95-107.
10. Simons DG, Travell JG, Simons LS. Travell & Simons' Myofascial Pain and Dysfunction: The Trigger Point Manual. 2nd ed. Baltimore, MD: Williams & Wilkins; 1999.
11. Mense S, Masi AT. Increased muscle tone as a cause of muscle pain. In: Mense S, Gerwin RD, eds. *Muscle Pain: Understanding the Mechanisms*. Heidelberg: Springer; 2011; 207-249.
12. Tough EA, White AR, Richards S, Campbell J. Variability of criteria used to diagnose myofascial trigger point pain syndrome—evidence from a review of the literature. *Clin J Pain* 2007;23:278-286.
13. Tough EA, White AR, Cummings TM, Richards SH, Campbell JL. Acupuncture and dry needling in the management of myofascial trigger point pain: A systematic review and meta-analysis of randomized controlled trials. *Eur J Pain* 2009;13:3-10.
14. Kietrys DM, Palombaro KM, Azzaretto E, et al. Effectiveness of dry needling for upper-quarter myofascial pain: A systematic review and meta-analysis. *J Orthop Sports Phys Ther* 2013;43:620-634.
15. Daut RL, Cleeland CS, Flanery RC. Development of the Wisconsin Brief Pain Questionnaire to Assess Pain in Cancer and Other Diseases. *Pain* 1983;17:197-210.
16. Fairbank JC, Couper J, Davies JB, O'Brien JP. The Oswestry Low Back Pain Disability Questionnaire. *Physiotherapy* 1980;66:271-273.
17. Ware JE Jr, Sherbourne CD. The MOS 36-Item Short-Form Health Survey (SF-36). I. Conceptual framework and item selection. *Med Care* 1992;30:473-483.
18. Shacham S. A shortened version of the Profile of Mood States. *J Pers Assess* 1983;47:305-306.
19. Proschan MA, Lan KKG, Wittes JT, eds. *Statistical Manual of Clinical Trials*. New York: Springer; 2007.
20. Cytel Statistical Software and Services (2014). StatXact with Cytel Studio, Version 10. Cambridge, MA.
21. Friction JR, Kroening R, Haley D, Siegert R. Myofascial pain syndrome of the head and neck: A review of clinical characteristics of 164 patients. *Oral Surg Oral Med Oral Pathol* 1985;60:615-623.
22. Bron C, Dommerholt JD. Etiology of myofascial trigger points. *Curr Pain Headache Rep* 2012;16:439-444.
23. Gerwin RD, Dommerholt J, Shah JP. An expansion of Simons' integrated hypothesis of trigger point formation. *Curr Pain Headache Rep* 2004;8:468-475.
24. Stecco A, Gesi M, Stecco C, Stern R. Fascial components of the myofascial pain syndrome. *Curr Pain Headache Rep* 2013;17:352.
25. Mense S. The pathogenesis of muscle pain. *Curr Pain Headache Rep* 2003;7:419-425.
26. Mense S, Simons DG, Russell IJ. *Muscle Pain: Understanding Its Nature, Diagnosis and Treatment*. Baltimore, MD: Lippincott Williams & Wilkins; 2000.
27. Mense S. How do muscle lesions such as latent and active trigger points influence central nociceptive neurons? *J Musculoskelet Pain* 2010;18:348-353.
28. Sluka KA, Kalra A, Moore SA. Unilateral intramuscular injections of acidic saline produce a bilateral, long-lasting hyperalgesia. *Muscle Nerve* 2001;24:37-46.
29. Niddam DM, Chan RC, Lee SH, Yeh TC, Hsieh JC. Central modulation of pain evoked from myofascial trigger point. *Clin J Pain* 2007;23:440-448.
30. Ge HY, Fernández-de-las-Peñas C, Yue SW. Myofascial trigger points: Spontaneous electrical activity and its consequences for pain induction and propagation. *Chin Med* 2011;6:13.
31. Baldry P. Superficial dry needling at myofascial trigger point sites. *J Musculoskelet Pain* 1995;3:117-126.
32. Dommerholt J. Dry needling—peripheral and central considerations. *J Man Manip Ther* 2011;19:223-237.
33. Cummings TM, White AR. Needling therapies in the management of myofascial trigger point pain: A systematic review. *Arch Phys Med Rehabil* 2001;82:986-992.
34. Lucas N, Macaskill P, Irwiq L, Moran R, Boqdruk N. Reliability of physical examination for diagnosis of myofascial trigger points: A systematic review of the literature. *Clin J Pain* 2009;25:80-89.
35. Ballyns JJ, Shah JP, Hammond J, Gebreab T, Gerber LH, Sikdar S. Objective sonographic measures for characterizing myofascial trigger points associated with cervical pain. *J Ultrasound Med* 2011;30:1331-1340.

36. Todd KH. Clinical versus statistical significance in the assessment of pain relief. *Ann Emerg Med* 1996;27:439-441.
37. Collins SL, Moore RA, McQuay HJ. The Visual Analogue Pain Intensity Scale: What is moderate pain in millimetres? *Pain* 1997;72:95-97.
38. Park G, Kim CW, Park SB, Kim MW, Jang SH. Reliability and usefulness of pain pressure threshold measurement in patients with myofascial pain. *Ann Rehabil Med* 2011;35:412-417.
39. Treaster D, Marras WS, Burr D, Sheedy JE, Hart D. Myofascial trigger point development from visual and postural stressors during computer work. *J Electromyogr Kinesiol* 2006;16:115-124.
40. Fernández-de-las-Peñas C, Dommerholt J. Myofascial trigger points: Peripheral or central phenomenon? *Curr Rheumatol Rep* 2014;16:395.
41. Hoyle JA, Marras WS, Sheedy JE, Hart DE. Effects of postural and visual stressors on myofascial trigger point development and motor unit rotation during computer work. *J Electromyogr Kinesiol* 2011; 21:41-48.
42. Chu J, Schwartz I. The muscle twitch in myofascial pain relief: Effects of acupuncture and other needling methods. *Electromyogr Clin Neurophysiol* 2002;42:307-311.
43. Kalichman L, Vulfsons S. Dry needling in the management of musculoskeletal pain. *J Am Board Fam Med* 2010;23:640-646.

---

**This journal-based CME activity is designated for 1.0 AMA PRA Category 1 Credit™ and can be completed online at [www.me.aapmr.org](http://www.me.aapmr.org). This activity is FREE to AAPM&R members and available to non-members for a nominal fee. For assistance with claiming CME for this activity, please contact (847) 737-6000.**

---

## Disclosure

L.H.G. Center for the Study of Chronic Illness and Disability, George Mason University, CCID, 4400 University Dr, Fairfax, VA 22030. Address correspondence to: L.H.G.; e-mail: [negerber1@gmu.edu](mailto:negerber1@gmu.edu)

Disclosure: nothing to disclose

J.S. Rehabilitation Medicine Department, Clinical Center, National Institutes of Health, Bethesda, MD

Disclosure: nothing to disclose

W.R. Department of Statistics, George Mason University, Fairfax, VA

Disclosure: nothing to disclose

K.A. Center for the Study of Chronic Illness and Disability, George Mason University, Fairfax, VA

Disclosures related to this publication: grant from National Institutes of Health

D.T. Department of Bioengineering, George Mason University, Fairfax, VA

Disclosure: nothing to disclose

P.O. Department of Bioengineering, George Mason University, Fairfax, VA

Disclosure: nothing to disclose

J.H. Rehabilitation Medicine Department, Clinical Center, National Institutes of Health, Bethesda, MD

Disclosure: nothing to disclose

N.T. Rehabilitation Medicine Department, Clinical Center, National Institutes of Health, Bethesda, MD

Disclosure: nothing to disclose

S.S. ~~Siddhartha Sikdar~~, Department of Bioengineering, George Mason University, Fairfax, VA

Disclosures related to this publication: grant support for study from National Institute of Arthritis and Musculoskeletal and Skin Diseases, National Institutes of Health

Disclosures outside this publication: travel support for workshop presentation from Canadian Academy of Physical Medicine and Rehabilitation, International Symposium of Ultrasound for Regional Anesthesia and Pain, and American Academy of Physical Medicine & Rehabilitation

Funding for this study was provided by the National Institute of Arthritis and Musculoskeletal and Skin Diseases, National Institutes of Health (grant #1R01AR057348).

Peer reviewers and all others who control content have no relevant financial relationships to disclose.

Submitted for publication September 20, 2014; accepted January 18, 2015.

## CME Question

The primary outcome measure of the dry needling technique was the

- visual analog scale (VAS)
- myofascial trigger point (MTrP) response
- Oswestry Disability Index (ODI)
- Brief Pain Inventory (BPI)

Answer online at [me.aapmr.org](http://me.aapmr.org)

## Ask the Authors

**Senior Editor: Heidi Prather, DO**

**Lynn H. Gerber, MD, Jay Shah, MD, Siddhartha Sikdar, PhD**

1. When I read research studies describing latent and active trigger points, the importance of the distinction is clear and resonates with me. However, when I am behind in my clinic schedule, the time necessary to

make the distinction seems lost, and I often treat whatever seems to correlate best with the patient's history, pain pattern, and physical examination, including palpation. This study has "triggered" me to

**consider the possibility that distinguishing and documenting active and latent trigger points, as well as documenting change over time, may have an impact on overall treatment recommendations.**

This is a most interesting observation. The authors would probably agree that in a busy practice, the time spent identifying active versus latent trigger points is probably minimal. On the other hand, little has been written about the natural history of trigger points, and hence we know little about whether they are likely to evolve into active trigger points or resolve completely without treatment. Nonetheless, the data we report suggest that the status of the trigger point is likely related to the pain level, and therefore the status (active or latent) is likely to be useful in guiding treatment.

**2. For example, an active myofascial trigger point (MTrP) that remains active over time may represent such significant centralization of pain that the usual rehabilitation endeavors to improve muscle function (such as stretching and strengthening) may actually enhance continued activity. In such cases, should the rehabilitation efforts be focused on desensitization of the muscle with active trigger points and improving resting muscle resting tone, length, neuromuscular control, and strength in muscles with latent or no trigger points?**

Local myofascial desensitization techniques (eg, dry needling, MTrP injections, and stretching) are sometimes painful. However, they are often clinically beneficial and theoretically may improve circulation in and around the MTrP, permitting the clearance of accumulated noxious biochemicals such as inflammatory mediators, proinflammatory cytokines, catecholamines, and neuropeptides [1,2]. Elevated levels of these substances, if left untreated, may result in persistent activation of nociceptors in that region, and ultimately central sensitization via bombardment of primary afferent neurons in the dorsal horn.

Alternative treatments such as biofeedback, breathing and relaxation exercises, cognitive retraining, and mindfulness meditation may facilitate the process of central desensitization via a “top down” approach. However, to most effectively address central sensitization, techniques that target both the peripheral and central contributors to central sensitization should be considered.

At the same time, it is important to strengthen and improve the function of both uninvolved muscles and those containing latent MTrPs. According to the “Cinderella hypothesis,” fibers containing active MTrPs tend to be overloaded and remain in a persistent state of contracture [3]. Re-educating and strengthening the surrounding musculature may decrease the load on these overused fibers, which allows them to be substituted and become relaxed, thus preventing their

progression into a potentially painful, contracted state.

**3. In patients with continued active trigger points that are consistent with significant pain sensitization and centralization, we often focus inherently on the muscle itself. Because of its primary sympathetic innervation, the fascia often becomes a contributor to a pain complex as well. Do you believe that the use of ultrasound to distinguish the active trigger points in the muscle from fascia pain should be advocated in the clinical setting and be a focus of further clinical research?**

This is an excellent point. Current evidence is not clear on this issue, and therefore we cannot recommend that this approach be attempted routinely in the clinical setting; however, it should certainly be a focus of further research. Although in this article we have not reported the ultrasound imaging results from this study, they have been reported elsewhere [4,5]. One of our observations is that the regions of the muscle that we suspect correspond to active MTrPs often occur near the deeper fascial border of the upper trapezius, which perhaps provides some indirect evidence of the role of the fascia. Other studies in the literature have pointed to the role of fascia in myofascial pain syndrome (eg, see reference 6) [6]. Further, while performing dry needling, it may be the case that the needle perturbs the fascial border as well as the muscle. Thus further careful studies are needed to separate the muscle and fascial contributors before any definitive conclusions can be drawn.

**4. Your results were phenomenal: 79% (41/52) of your subjects had improvement in self-reported pain, range of motion, outlook on health and physical function, and reduced disability after 3 sessions of dry needling. There are drugs and procedures that cannot deliver results like these for disorders that are more easily objectively defined. Further, the outcome required an expert, time, and a needle—all in all, a relatively low-cost intervention. Do you plan to track health care utilization in future studies and assess utilization over time?**

A. This is an excellent question. We have not performed cost analysis for treatment of myofascial pain syndrome (MPS) or examined whether there is a better cost/benefit ratio for one treatment over another. The prevalence of MPS in the United States is high; Simons [7] reports a prevalence of 85%. Further, there is not consistent agreement on the effectiveness of dry needling or its superiority when compared with wet needling, analgesics, and manual therapies [8]. In other words, one would wish to choose the most effective treatment for MPS, and consensus is lacking about which treatment would best be selected for a cost analysis.

**5. Can you offer us more insight into the technique you performed? For example, on average, how long did the treatment take, and how much time was spent per muscle?**

The study we report was a prospective interventional study. Evaluation was systematically performed with use of a very specific protocol, and the initial comprehensive first visit required approximately 45 minutes, which is not comparable to the usual practice in a busy pain clinic. We performed careful manual palpation. Part of the examination required careful palpation across the left and right upper trapezius muscles, focusing on identifying trigger points and distinguishing which one was most symptomatic. The trigger point survey alone took approximately 10 minutes. Preparation of the trigger point site for injection took 1 minute using a alcohol swab. The needle was inserted at the site of the trigger point, advanced to about 1 inch, rotated, and partially withdrawn to try to elicit a twitch response. If none was obtained, the needle was moved to 3 or 4 different points surrounding the trigger point. The elapsed time for a typical needling was a few minutes.

**6. What common mistakes do you see clinicians make with regard to treating MTrPs? Is it what we use (eg, dry needling, anesthetics, and steroids), or perhaps lack of recognition of the trigger points? Are we overtreating? What metrics would you recommend that clinicians track over time as they treat these patient populations?**

Our clinicians have spent time calibrating their physical examination with each other to ensure that we properly identify MPS and MTrPs. Our reliability is high, but we did have to spend time testing and retesting patients to arrive at standard nomenclature of active/latent trigger points and confirm the findings. Clinical competence must be achieved through practice. We believe some clinicians may improperly classify the pain syndrome, include radicular and neuropathic pain, and treat this as myofascial pain. We also believe that some

clinicians may misclassify the status of the trigger point [9]. In our view, one must make the correct diagnosis before treating the patient.

It has been our experience that many treatments are effective in relieving myofascial pain. The literature has not produced a clear "winner" for effectiveness. Nonetheless, dry needling offers an excellent treatment choice, and our data suggest that pain and trigger point status are good outcome measures for the clinician to track over time. We will be reporting on data we have collected 6 weeks after the end of treatment, where we will discuss the outcome measures used to determine whether patients have had a sustained response to the initial treatment. Measuring cervical range of motion, mood, function, and disability may be of interest but are secondary to the status of the trigger point and the myofascial pain.

## References

- Shah JP, Phillips TM, Danoff JV, Gerber LH. An in vivo microanalytical technique for measuring the local biochemical milieu of human skeletal muscle. *J Appl Physiol* (1985) 2005;99:1977-1984.
- Shah JP, Danoff JV, Desai MJ, et al. Biochemicals associated with pain and inflammation are elevated in sites near to and remote from active myofascial trigger points. *Arch Phys Med Rehabil* 2008;89:16-23.
- Hägg G. Static work loads and occupational myalgia—a new explanation model. *J Electromyogr Kinesiol* 1991;949:141-144.
- Turo D, Otto P, Shah JP, et al. Ultrasonic characterization of the upper trapezius muscle in patients with chronic neck pain. *Ultrason Imaging* 2013;35:173-187.
- Turo D, Otto P, Hossain M, et al. Novel use of ultrasound elastography to quantify muscle tissue changes after dry needling of myofascial trigger points in patients with chronic myofascial pain. *J Ultrasound Med* (in press).
- Stecco A, Gesi M, Stecco C, Stern R. Fascial components of the myofascial pain syndrome. *Curr Pain Headache Rep* 2013;17:352.
- Simons DG. Clinical and etiological update of myofascial pain from trigger points. *J Musculoskelet Pain* 1996;4:93-122.
- Fleckenstein J, Zaps D, Rüger LJ, et al. Discrepancy between prevalence and perceived effectiveness of treatment methods in myofascial pain syndrome: Results of a cross-sectional, nationwide survey. *BMC Musculoskeletal Disorders* 2010;11:32.
- Lucas N, Macaskill P, Irwig L, Moran R, Bogduk N. Reliability of physical examination for diagnosis of myofascial trigger points: A systematic review of the literature. *Clin J Pain* 2009;25:80-89.

---

## Disclosure

H.P. Washington University School of Medicine, St. Louis, MO

L.H.G. Center for the Study of Chronic Illness and Disability, George Mason University, Fairfax, VA

J.S. Rehabilitation Medicine Department, Clinical Center, National Institutes of Health, Bethesda, MD

S.S. Department of Bioengineering, George Mason University, Fairfax, VA

---