



Original Research—CME

Beneficial Effects of Dry Needling for Treatment of Chronic Myofascial Pain Persist for 6 Weeks After Treatment Completion

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Abstract

Background: Dry needling is an effective treatment for reducing pain associated with active myofascial trigger points (a-MTrPs) in the short term. The duration of the benefits of this treatment have not been fully assessed.

Objective: To determine whether the benefits of dry needling (DN) of a-MTrPs are sustained 6 weeks posttreatment.

Design: Follow-up of a prospective study.

Setting: University.

Participants: A total of 45 patients (13 male and 32 female) with cervical pain >3 months and a-MTrPs in the upper trapezius who completed 3 DN treatments and who were evaluated 6 weeks posttreatment.

Interventions: None.

Main Outcome Measures: Primary outcomes were changes from baseline to follow-up in scores for the verbal analogue scale (VAS), Brief Pain Inventory (BPI), and MTrP status. MTrPs were rated as active (spontaneously painful), latent (painful only on compression), and nonpalpable nodule. Responders were patients whose MTrP status changed from active to latent or nonpalpable nodule (resolved). Secondary outcomes were pain pressure threshold (PPT), Profile of Mood States, Oswestry Disability Index (ODI), MOS 36-Item Short-Form Health Survey (SF-36), and cervical range of motion.

Results: Pain measures remained significantly improved 6 weeks posttreatment ($P < .003$), as did the SF-36 physical functioning score (0.01) and ODI ($P = .002$). Side bending and PPT for subjects with unilateral MTrPs had sustained improvement ($P = .002$). The number of subjects with sustained MTrP response at 6 weeks was significant ($P < .001$). Comparing responders to nonresponders, the changes in VAS and BPI were statistically significant ($P = .006$, $P = .03$) but the change in PPT was not. Patients with higher baseline VAS scores had a higher risk of not responding to DN; those with a greater drop in VAS score from baseline had a higher probability of sustained response. A 1-unit decrease in VAS at baseline resulted in a 6.3-fold increase in the odds of being a responder versus a nonresponder ($P = .008$).

Conclusions: In this study, there was sustained reduction of pain scores after completion of DN, which is more likely with a greater drop in VAS score. Patients with higher baseline VAS scores are less likely to respond to DN. Early intervention toward significant pain reduction is likely to be associated with sustained clinical response.

Level of Evidence: IV

Introduction

Myofascial pain syndrome (MPS) is often a chronic condition with a prevalence that varies from 15% to a lifelong prevalence of 85% [1-3]. There is lack of consensus about what constitutes criteria for diagnosis [4-7] and which measures are appropriate for clinical research outcomes [8]. Recently, researchers have reported the results of surveys among health care providers to assess whether there is even a working consensus about diagnostic criteria, which tissue should be the treatment

target, which measures are best for determining efficacy, and whether they should be objective and/or self-reported outcomes [3,9]. The results of these surveys show a lack of agreement among clinicians and investigators on many of these points. Accordingly, there is a need for standardizing patient evaluations, interventions, and treatment outcomes if we are to generate reliable evidence and influence practice. With these standards, treatment guidelines can be generated.

One approach, which is one that our research team has selected, is to devise a standard evaluation for

patients with MPS that is used for determining the diagnosis, the level of severity of the condition, and outcomes sought to assign effectiveness [2,10]. Toward this end, pain as well as the status of the myofascial trigger point (MTrP) were considered important. We posited that the MTrP was central to the MPS. Standardizing the examination of the MTrP, assessing its status in terms of being active or latent, and evaluating its responsiveness (or lack thereof) to treatment provides an opportunity for obtaining reliable data about the effectiveness of treatment. The Travell and Simons description states that an MTrP is a discrete, palpable nodule located within a taut band of skeletal muscle [6]. When the MTrP is spontaneously painful, it is known as an active MTrP (a-MTrP). Strong digital pressure on an a-MTrP exacerbates the patient's spontaneous pain complaint and mimics the patient's familiar pain experience. When the MTrP is not spontaneously painful but typical pain can be elicited when palpated or disturbed, it is regarded as a latent MTrP (l-MTrP). The l-MTrP is a nodule with the same physical characteristics as an a-MTrP but requires palpation to elicit pain. Electromyographic, biochemical, and imaging studies have demonstrated notable differences between these 2 MTrP classifications, and also distinguish them from normal, nonpainful tissue [11,12].

It has not yet been established that the MTrP is necessary for the pain syndrome. It is also not yet known whether the MPS "causes" the development of the MTrP. These questions remain of significant interest to the research community and to practitioners. If it could be shown that the MTrP is a necessary condition for MPS, it would serve as an objective, identifiable target for pain relief. Thus, a clear, definitive relationship between the MTrP and MPS would help to advance the field.

Our research group has reported the results of a prospective intervention trial of dry needling for the treatment of patients with chronic shoulder girdle/neck pain secondary to a-MTrPs in the upper trapezius muscle [13]. In this study, we applied standardized evaluations that assessed objective findings and self-reported outcomes, including pain, mood, health status and disability measures, and cervical range of motion. We also used ultrasound, Doppler imaging, and elastography to determine tissue properties of the upper trapezius muscle and the characteristics of MTrPs at baseline and after treatment. Our results demonstrated that 3 treatments (once per week for 3 weeks) of a-MTrPs using a standardized dry needling technique significantly reduced myofascial pain and changed the status of the MTrP from active to either latent or resolved [13]. Other investigators have used a similar approach with positive therapeutic results. Several recent reviews and meta-analyses are available [14-17]. This article reports the findings of the same cohort 6 weeks after completion of the 3 dry needling treatments. Our aim was to assess the long-term treatment

outcome of dry needling in a cohort of patients receiving 1 course of treatment without any intervening subsequent treatment for MPS.

Methods

Participants and Procedures

The study was approved by the Chesapeake Institutional Review Board. All participants were consented by 1 of 3 of the authors. Participants were recruited from a university campus and surrounding area, received no remuneration for participation, and ranged in age from 18 to 65 years. This was a convenience sample. All patients who participated in the intervention trial were asked to return for a follow-up evaluation 6 weeks after completion of their initial 3 dry needling treatments and were seen at the same clinic. Entry criteria included at least 1 palpable a-MTrP in the upper trapezius. Exclusion criteria included 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. A general history and physical examination were completed in all participants and included inquiry about medication, dietary supplements, and regular exercise (defined by at least 3 episodes of exercise for at least 30 minutes in duration [2]. A repeat of all baseline and treatment outcome measures was performed by 2 clinicians. There were 2 measures of pain used as primary outcome measures. These were a verbal analogue scale (VAS) [18], scored from 0 to 10 (0 = no pain, 10 = unbearable pain), and the Brief Pain Inventory (BPI) [19]. Another primary outcome was change in trigger point status, as determined by palpation of the upper trapezius muscle by 2 clinicians (J.P.S., L.H.G.), who have achieved good interrater reliability [13]. MTrP status was scored as a change from a-MTrP to either l-MTrP or no palpable nodule. A secondary outcome measure used for pain was the pain pressure threshold (PPT) [20]. 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>). Additional secondary outcomes included cervical range of motion (ROM) (flexion/extension, side bending, and rotation). Cervical ROM was determined using the Deluxe Cervical Range of Motion Instrument (CROM), model 12-1156 (Fabrication Enterprises, White Plains, NY) for determination of asymmetry. Patient-reported outcomes included the Oswestry Disability Index [21], the MOS 36-Item Short-Form Health Survey (SF-36) [22], as well as a short version of the Profile of Mood States (POMS) [23]. A full description of the instruments used and methods followed is available [2,13].

Data Analysis

Analyses of covariance models were conducted to determine the impact of responder/nonresponder status on pain metrics, adjusted for important variables. The adjustment variables are the baseline pain measure, side, age, gender, and exercise status. As these variables are deemed a priori important, they are included in all models whether or not the results were significant. Least-squares means were computed for VAS, BPI, and PPT for the change from baseline to 8 weeks and the change from 3 weeks to 8 weeks. A full longitudinal assessment was not performed, as data were available only at 2 points in time. Appropriate regression diagnostics were performed, including Q-Q plots to assess the normality assumption of the regression model, and residual plots to assess homoscedasticity. All models were deemed appropriate. Results for *P* values are reported without adjustment for multiple testing.

We conducted an adjusted logistic regression analysis to determine how the baseline and change from

baseline pain scores would affect the odds ratio of being a responder versus a nonresponder. It should be noted that the study was not designed to answer this question.

Results

There were 45 patients, 13 male and 32 female, with a mean age of 37 years, who completed follow-up at 8 weeks. All had received 3 dry needling treatments, which were completed 6 weeks prior, and had no intervening treatment for MPS.

Subject characteristics at baseline and 8 weeks are given in Table 1 as well as the *P* value of the *t* test of comparison of means. Asymmetry of side bending for subjects with unilateral trigger points and PPT at the treated site were significantly improved from baseline (*P* = .002). Pain measures were all significantly improved except for VAS at the untreated site in patients with unilateral trigger points (*P* < .003). The SF-36 physical functioning score (*P* = .012) and the

Table 1
Patient characteristics at baseline and 8 weeks

Characteristic	n	Baseline	Follow-up	<i>P</i> Value
Physical findings (mean ± SD)				
Cervical ROM extension	33	73.4 ± 10.3	73.5 ± 10.9	.91
Cervical ROM flexion	33	52.8 ± 9.5	54.7 ± 8.2	.28
Rotation asymmetry unilateral	16	6.6 ± 5.7	3.6 ± 4.1	.12
Rotation asymmetry bilateral	17	4.6 ± 3.6	1.9 ± 3.2	.06
Side bending unilateral	16	6.4 ± 3.7	1.9 ± 2.6	.002
Side bending bilateral	17	4.1 ± 4.7	2.6 ± 4.2	.31
PPT treated site unilateral	16	7.4 ± 4.0	8.8 ± 4.1	.009
PPT treated site bilateral	17	6.7 ± 3.0	7.6 ± 2.8	.39
PPT untreated site unilateral	16	9.1 ± 4.0	8.8 ± 4.8	.77
PPT untreated site bilateral	17	8.3 ± 3.5	7.1 ± 2.2	.27
Pain (mean ± SD)				
BPI	27	3.2 ± 1.1	1.9 ± 1.4	<.001
VAS treated site unilateral	24	3.3 ± 2.0	1.3 ± 1.8	.002
VAS treated site bilateral	21	3.0 ± 1.4	1.2 ± 1.7	.001
VAS untreated site unilateral	23	1.1 ± 2.0	0.78 ± 1.44	.41
VAS untreated site bilateral	21	2.7 ± 1.2	1.1 ± 1.3	<.001
SF-36 pain	29	61.5 ± 15.5	72.6 ± 15.2	.003
Self-reported outcomes (mean ± SD)				
POMS confusion	29	0.21 ± 0.34	0.17 ± 0.21	.51
POMS depression	29	0.08 ± 0.20	0.08 ± 0.22	>.99
POMS fatigue	29	0.74 ± 0.78	0.49 ± 0.56	.13
POMS tension	29	0.43 ± 0.40	0.34 ± 0.62	.50
POMS mood	29	-0.06 ± 1.50	-0.58 ± 1.88	.20
POMS vigor	29	1.63 ± 0.90	1.75 ± 0.92	.34
POMS anger	29	0.10 ± 0.24	0.06 ± 0.13	.39
SF-36 general health	29	78.4 ± 18.4	80.5 ± 14.3	.24
SF-36 mental health	29	79.3 ± 9.4	79.3 ± 14.5	>.99
SF-36 physical functioning	29	92.8 ± 9.8	95.9 ± 6.1	.01
SF-36 emotional	29	85.6 ± 22.9	93.4 ± 9.3	.08
SF-36 physical role	29	87.9 ± 15.2	91.2 ± 12.2	.28
SF-36 Social functioning	29	91.4 ± 12.5	93.1 ± 11.4	.35
SF-36 vitality	29	60.3 ± 16.0	63.1 ± 16.9	.30
Disability (mean ± SD)				
ODI score	28	8.7 ± 5.4	6.2 ± 4.7	.002

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

Table 2
Primary outcomes for treated subjects with bilateral active trigger points

Site	Baseline	Follow-up	No. of Subjects With This Status
Treated	Active	Active	6
	Active	Latent	8
	Active	Normal	7
Untreated	Active	Active	6
	Active	Latent	8
	Active	Normal	3

Oswestry Disability Index ($P = .002$) remained significantly improved from baseline.

The change in status of the MTrP from baseline was also measured (Tables 2 and 3). When the status of the MTrP changed from active to latent or nonpalpable, the patient was identified as a “responder.” The number of subjects who were responders at 3 weeks was highly significant, and this was sustained at 8 weeks ($P < .001$).

Figures 1, 2, and 3 show the mean and standard deviation of VAS, BPI, and PPT, respectively, from baseline to 3 weeks, and 8 weeks. These results indicate that the VAS decreases from baseline at 3 weeks, and this decrease is sustained at 8 weeks for responders. Among responders, the VAS at 8 weeks was significantly lower than baseline. For nonresponders, the VAS at 8 weeks is not significantly different from baseline. A similar pattern was observed for BPI. No significant trends were observed for PPT, although the PPT for responders at 8 weeks was significantly higher (less pain for the same amount of pressure) than that of nonresponders. We proceeded to model the baseline to 8 week data and 3 week to 8 week data to report adjusted least-squares means.

Change from baseline to 8 weeks in VAS was statistically significant ($P = .006$) between responders (-2.29 , standard error [SE] = 0.27) and nonresponders (-0.46 , SE = 0.60) (Table 4). Of the adjustment variables, only baseline VAS ($P < .001$) and age ($P = .02$) were statistically significant (Table 5). Change from baseline to 8 weeks in BPI was also statistically significant ($P = .026$) between responders (-1.43 , SE = 0.22) and nonresponders (0.08 , SE = 0.60) (Table 6), and with no significant adjustment variables (Table 7). Change from baseline to 8 weeks in

Table 3
Primary outcomes for treated subjects with unilateral active trigger points

Side	Baseline	Follow-up	No. of Subjects With This Status
Treated	Active	Active	7
	Active	Latent	9
	Active	Normal	8
Untreated	Latent	Active	0
	Latent	Latent	11
	Latent	Normal	3
	Normal	Active	1
	Normal	Latent	1
	Normal	Normal	2

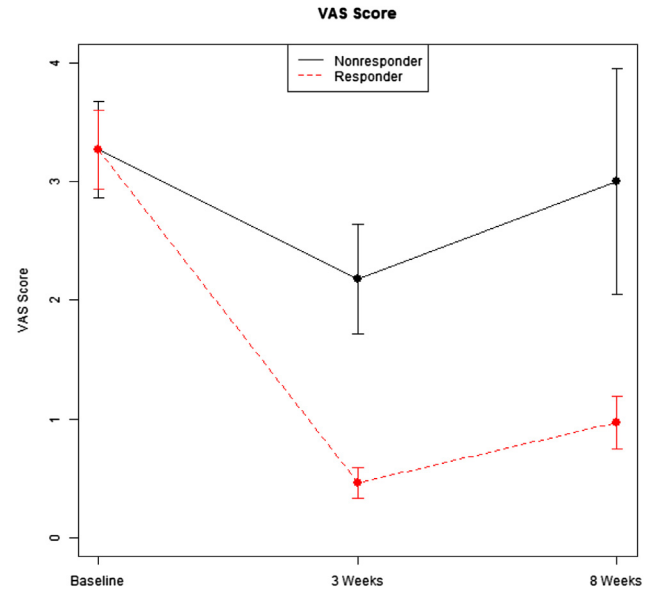


Figure 1. Trends for verbal analogue scale (VAS) scores at baseline, 3 weeks, and 8 weeks among responders and nonresponders.

PPT was not statistically significant ($P = .17$; data not shown) between responders (1.11 , SE = 0.57) and nonresponders (-1.14 , SE = 1.57).

Change from 3 weeks to 8 weeks in VAS was not statistically significant ($P = .57$) between responders (0.47 , SE = 0.26) and nonresponders (0.14 , SE = 0.57). Similarly, change from 3 weeks to 8 weeks in BPI was not statistically significant ($P = .91$) between responders (-0.14 , SE = 0.24) and nonresponders (-0.06 , SE = 0.62).

Our results, viewed from the perspective of baseline and change from baseline in pain scores, show that patients with higher baseline scores had a higher risk of not responding to dry needling. They also show that patients with a higher reduction in VAS pain scores from

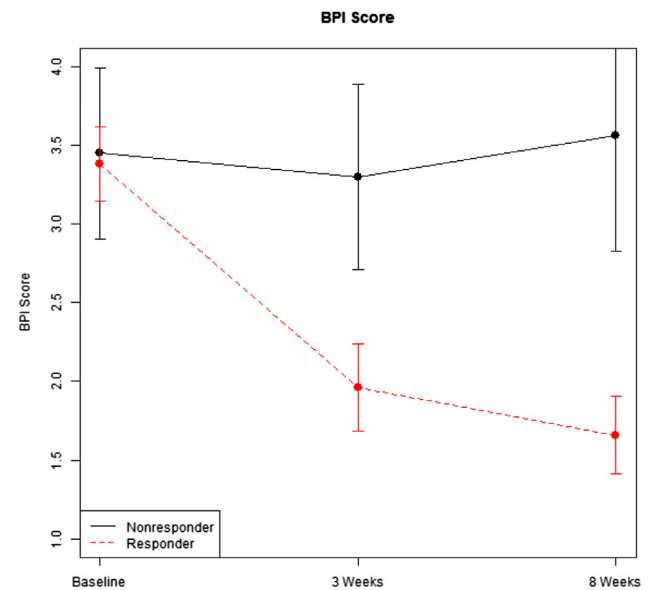


Figure 2. Trends for Brief Pain Inventory (BPI) at baseline, 3 weeks, and 8 weeks among responders and nonresponders.

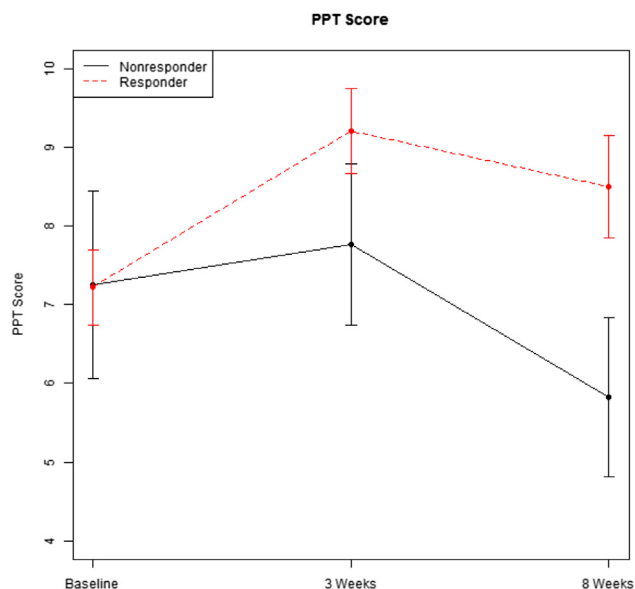


Figure 3. Trends for pain pressure threshold (PPT) at baseline, 3 weeks, and 8 weeks among responders and nonresponders.

baseline score have a higher probability of responding to treatment. A 1-unit decrease in VAS score at baseline resulted in a 6.3-fold increase in the odds of being a responder versus a nonresponder ($P = .008$). For VAS scores measured at 3 weeks, a 1-unit increase in change from baseline resulted in an 8-fold increase in the odds of being a responder versus a nonresponder ($P = .004$). For VAS scores measured at 8 weeks, a 1-unit increase in change from baseline resulted in a doubling of the odds of being a responder versus a nonresponder ($P = .02$).

Discussion

MPS is a pain syndrome the etiology of which is still being debated. Many, but not all, clinicians and investigators agree that an MPS diagnosis must include the presence of an MTrP [6,24,25]. This study supports the view that MPS and a-MTrPs have a significant relationship, but their pathophysiology and mechanisms need to be better understood.

Pieces of the puzzle about MTrPs are unfolding. However, the relationships between the biochemical findings and tissue properties of MTrPs, and whether they play a key role in clinical response to treatment, remain subjects of debate. In addition, the idea that a-MTrPs are the “pain generators” within MPS remains contested. Some groups suggest that the tissue in need of treatment is not

Table 4

Least squares means of change in verbal analogue scale (VAS) score at 8 weeks from baseline, adjusted for baseline, site, gender, age, and exercise status

Response	Mean Change in VAS	SE
Responders	-2.29	0.27
Nonresponders	-0.46	0.60

SE = standard error.

Table 5

Regression estimates with change in verbal analogue scale (VAS) score at 8 weeks from baseline as response variable

Parameter	Estimate	SE	t	P Value
Intercept	3.41	0.91	3.74	.001
VAS baseline	-0.73	0.14	-5.27	<.001
Responders	-1.83	0.62	-2.97	.005
Nonresponders	Baseline			
Bilateral	0.17	0.45	0.39	.70
Unilateral	Baseline			
Age	-0.05	0.02	-2.45	.02
Gender, female	0.49	0.53	0.94	.36
Gender, male	Baseline			
Exercise status, no	-0.45	0.51	-0.88	.38
Exercise status, yes	Baseline			

SE = standard error.

necessarily the MTrP but, rather, the muscle [26,27]. Another group suggests that it is the fascia [28]. Still others suggest that there are several contributors to pain development, such as arthritis or nerve root irritation [29,30]. Regardless, the persistence of pain typically requires the development of central sensitization and/or changes in the dorsal horn [31]. Sensitization of both peripheral and central afferents is responsible for the transition from normal to aberrant pain perception in the central nervous system that outlasts a noxious peripheral stimulus. Biochemical data suggest that a-MTrPs involve mechanisms of muscle nociception and sensitization and are therefore potential sources of persistent pain [32,33]. Our group has previously reported a link in pain reduction in patients with a-MTrPs and MPS to a change in status of the MTrP. The conversion of an a-MTrP from a spontaneously painful state to one requiring perturbation (latent) or a resolution of the finding is associated with a significant reduction in pain [13]. A reduction in pain is accompanied by a decrease in central sensitization. The follow-up data reported in this article demonstrate that the relationship between MTrP status change and pain reduction remains significant at 8 weeks. This observation supports the view that, on average, persistent pain reduction is likely related to sustained MTrP status change or vice versa, but a causal relationship has not been established; that is, the change in status of the MTrP does not necessarily cause the decrease in pain but, rather, both occur together.

Many have reported abnormalities of MTrPs associated with MPS that might help explain their pathophysiology and underlying association with the pain syndrome. These include increased spontaneous electrical activity [34], which is an indication of excessive acetylcholine (ACh)

Table 6

Least squares means of change in Brief Pain Inventory (BPI) score at 8 weeks from baseline adjusted for baseline, site, gender, age, and exercise status

Response	Mean Change in BPI	SE
Responders	-1.43	0.22
Nonresponders	0.08	0.60

SE = standard error.

Table 7

Regression estimates with change in Brief Pain Inventory (BPI) score at 8 weeks from baseline as response variable

Parameter	Estimate	SE	<i>t</i>	<i>P</i> Value
Intercept	1.21	1.26	0.96	.35
BPI baseline	-0.28	0.21	-1.36	.19
Responders	-1.51	0.63	-2.41	.03
Nonresponders	Baseline			
Bilateral	0.11	0.44	0.24	.81
Unilateral	Baseline			
Age	-0.01	0.02	-0.69	.50
Gender, female	-0.05	0.47	-0.1	.92
Gender, male	Baseline			
Exercise status, no	0.34	0.46	0.74	.47
Exercise status, yes	Baseline			

SE = standard error.

release at the motor endplate. This would lead to sarcomere contracture that could, in turn, produce local ischemia and hypoxia, resulting in the release of algogenic and vasoactive substances (eg, inflammatory cytokines, neuropeptides, and catecholamines) capable of activating and sensitizing peripheral nociceptors in a-MTrPs and surrounding soft tissue [9,35-37].

The mechanism(s) by which dry needling of MTrPs might reduce myofascial pain is also somewhat speculative. These include its effects on the taut band, local ischemia and hypoxia, and peripheral and central sensitization via neural mechanisms, increase in local milieu blood flow and oxygenation, change in the milieu of endogenous opioids, endorphins, cholinergic anti-inflammatory mediators, and a modulatory effect on sensory neural impulses at the central nervous system level [38-45].

One study demonstrated that dry needling of active (but not latent) MTrPs can elicit motor unit potentials (MUPs), in a time-locked manner, on the contralateral side of the body. Because only a-MTrPs featured contralateral MUPs, it suggests that 1 difference between a-MTrPs versus l-MTrPs is due to maladaptive neuroplastic changes in the central nervous system of both the sensory and motor arms. In addition, 38% of a-MTrPs did not feature contralateral MUPs, suggesting that there may be degrees of central sensitization. This likely depends upon the chronicity of pain and maybe even the degree of neuroplasticity. This study demonstrated another recordable pathophysiological distinction that emphasizes the validity and importance of clinically differentiating active from latent MTrPs [46]. Only a few articles have been published about the natural history of a-MTrPs and their relationship to MPS [30,47]. None, to our knowledge, have addressed the natural history of treating a-MTrPs. In other words, if one has an effective treatment, how long does the treatment effect persist? In addition, are there any potential patient or syndrome characteristics that might inform us about the posttreatment trajectory of patients? Such syndrome characteristics could include intensity and chronicity of pain, the presence of segmental and/or supraspinal sensitization, the amount of pain reduction

following treatment, whether pain reduction is associated with a low PPT and improvements in self-reported outcome measures, the number and size of a-MTrPs, and whether the patient presented with bilateral a-MTrPs or only unilateral a-MTrPs at baseline. Which patients are at risk for recurrence, and what are their patient profiles?

The findings from the prospective treatment trial reported here suggest that patients receiving 3 weekly treatments of dry needling for an active MTrP have sustained benefit for at least 6 weeks after treatment. We believe that this can be attributed to the effect of the dry needling that they received, as no intervening treatment was obtained to the best of our knowledge. Dry needling, the use of a high-gauge, solid, filiform, no-bore needle without the addition of solutions or local pharmacological agents has had therapeutic application for nearly 50 years [24,47].

In this study, pain is the primary outcome, and the VAS and BPI were used to measure pain. The VAS is a unidimensional measure of pain intensity that we selected because of validity, ease, and frequency of use in many different clinical settings [48,49]. However, we also measured pain using 2 different multi-dimensional instruments, the BPI and the Bodily Pain Score of the SF-36. Results from these measures were similar to those of the VAS. Pain response was sustained during the follow-up period and remained statistically significantly different from baseline scores.

The algometer measurements remained significantly different from baseline only in the patients who had a unilateral a-MTrP. Patients with bilateral involvement were only slightly better at 8 weeks (PPT score of 6.7 at baseline and 7.6 at 8 weeks). This was not statistically significantly different from baseline scores. Algometry and the recording of the PPT has been shown to be a very reliable measure and correlates well with MTrP sensitivity [20,50,51]. It uses an instrument (algometer) and hence is objective, but requires patient reporting of when the pain threshold is reached. The interpretation of what the PPT represents and whether it is providing the same information as a self-reported pain score is worthy of discussion. Fischer suggested that this type of measurement can be used reliably to quantify tenderness and to "diagnose pathological tenderness in muscles." He further notes that there is a strong correlation between the PPT and tissue resistance, as evidenced by the taut muscular band, "muscle tone or consistency," and tissue compliance meters. This latter is a method of determining the "depth of penetration achieved by a unit of force applied" [50]. These observations suggest that the PPT may provide information that is different from a self-report of pain.

Mood and health-related quality of life measures were not significantly different from baseline measures. However, the disability measure (Oswestry Disability Index) and the physical functioning score of the SF-36 demonstrated sustained improvement over baseline.

There were no statistically significant differences between baseline VAS or BPI scores between the responders and nonresponders. The drop in VAS for the responders was clinically significant (>2.0 points). This suggests that a sustained response to treatment is more likely to be observed if there is a clinically meaningful drop in pain level at initial treatment. A decrease in VAS of 2 points is thought reach this threshold. The standard for clinically meaningful improvement for the PPT is an increase of 4 kg/cm^2 [52]. The clinical response of the cohort improved by 2 kg/cm^2 . In the earlier report about the efficacy of 3 dry needling techniques [13], there were no significant differences in PPT scores between patients with unilateral versus bilateral trigger points.

This study has several limitations. The follow-up phase began initially as a clinical assessment during which patients were asked to return for a physical examination and an assessment of their pain status. The research team decided to perform all of the baseline and posttreatment assessments, which was approved. The first 8 subjects had only limited data for this follow-up and are not included in the 8-week evaluation. In addition, although we requested that study participants not initiate new treatments for myofascial pain and they reported that they did not obtain such treatments, we did not confirm this through means other than self-reports. The cohort in this study was recruited primarily from a university campus and may not reflect the usual population of individuals with myofascial pain. In general, they were young, spent much time at computer terminals, and may have a lifestyle different from that of an older population. We did perform regression estimates for changes in VAS and found that age was a significant variable, although participation in exercise was not, and we did not inquire about diet.

Conclusion

In this study, 3 dry needling treatments for patients with chronic myofascial pain and active myofascial trigger points resulted in a sustained reduction of pain, as measured by VAS and BPI, at 6-week follow-up after completion of treatment. Only patients with unilateral active MTrPs had sustained improvement in PPT scores and cervical side bending. Patients achieving a lower pain score after treatment had a sustained response to treatment.

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, Durmus O, Cakar E, Dincer U, Kiralp 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 fibrositis. *Proc R Soc Med* 1913;6:27-35.
5. Schade H. Untersuchungen in der Erkältungstrage: 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. Rathbone AT, Kumbhare DA. Re: Signs and symptoms of myofascial pain: An international survey of pain management providers and proposed preliminary set of diagnostic criteria. *Pain Med* 2016;17:620.
11. 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* 2015;34:2149-2161.
12. Hubbard DR, Berkoff GM. Myofascial trigger points show spontaneous needle EMG activity. *Spine* 1993;18:1803-1807.
13. Gerber LH, Shah J, Rosenberger W, et al. Dry needling alters trigger points in the upper trapezius muscle and reduces pain in subjects with chronic myofascial pain. *PM R* 2015;7:711-718.
14. Kalichman L, Vulfsons S. Dry needling in the management of musculoskeletal pain. *J Am Board Fam Med* 2010;25:5640-5646.
15. Kietrys DM, Palombaro KM, Mannheimer JS. Dry needling for management of pain in the upper quarter and craniofacial region. *Curr Pain Headache Rep* 2014;18:437.
16. Dunning J, Butts R, Mourad F, Young I, Flannagan S, Perreault T. Dry needling: A literature review with implications for clinical guidelines. *Phys Ther Rev* 2014;19:252-265.
17. Cagnie B, Castelein B, Pollie F, Steelan L, Verhoeyen H, Cools A. Evidence for the use of ischemic compression and dry needling in the management of myofascial trigger points in the upper trapezius in patients with neck pain: A systematic review. *Am J Phys Med Rehabil* 2015;94:573-583.
18. Jensen MP, Karoly P, Braver S. The measurement of clinical pain intensity: A comparison of six methods. *Pain* 1986;27:117-126.
19. 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.
20. Fischer AA. Pressure algometry over normal muscles. Standard values, validity and reproducibility of pressure threshold. *Pain* 1987;30:115-126.
21. Fairbank JC, Couper J, Davies JB, O'Brien JP. The Oswestry Low Back Pain Disability Questionnaire. *Physiotherapy* 1980;66:271-273.
22. 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.
23. Shacham S. A shortened version of the Profile of Mood States. *J Pers Assess* 1983;47:305-306.
24. Simons DG, Travell JG, Simons LS. *Myofascial Pain and Dysfunction: The Trigger Point Manual*. 2nd ed., vol. 1. Baltimore, MD: Williams & Wilkins; 1999.
25. Cailliet R. *Soft Tissue Pain and Disability*. Philadelphia, PA: F. A. Davis; 1977.
26. Mense S. Muscle pain: Mechanisms and clinical significance. *Deutsches Arzt Int* 2008;105:214-219.
27. Chen Q, Wang H, Gay R, et al. Quantitation of myofascial taut bands. *Arch PMR* 2016;97:67-72.
28. Stecco A, Gesi M, Stecco C, Stern R. Fascial components of the myofascial pain syndrome. *Curr Pain Headache Rep* 2013;17:352.
29. Huntley AH, Srbely JZ, Zettel JL. Experimentally induced central sensitization in the cervical spine evokes postural stiffening strategies in healthy young adults. *Gait Posture* 2015;41:652-657.
30. Deitos A, Dussán-Sarria JA, Souza A, et al. Clinical value of serum neuroplasticity mediators in identifying the central sensitivity

- syndrome in patients with chronic pain with and without structural pathology. *Clin J Pain* 2015;31:959-967.
31. Melzack R. Myofascial trigger points: Relation to acupuncture and mechanisms of pain. *Arch Phys Med Rehabil* 1981;62:114-117.
 32. 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* 2005;99:1977-1984.
 33. 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.
 34. Ge HY, Monterde S, Graven-Nielsen T, Arendt-Nielsen L. Latent myofascial trigger points are associated with an increased intramuscular electromyographic activity during synergistic muscle activation. *J Pain* 2014;15:181-187.
 35. Awad E. Interstitial myofibrosis: Hypothesis of the mechanism. *Arch Phys Med Rehabil* 1973;54:449-453.
 36. Brendstrup P, Jespersen K, Asboe H. Morphological and chemical connective tissue changes in fibrositic muscles. *Ann Rheum Dis* 1957;16:438-440.
 37. Rosendal L, Larsson B, Kristiansen J, et al. Increase in muscle nociceptive substances and anaerobic metabolism in patients with trapezius myalgia: Microdialysis in rest and during exercise. *Pain* 2004;112:324-334.
 38. Lewit K. The needle effect in the relief of myofascial pain. *Pain* 1979;6:83-90.
 39. 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.
 40. 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 randomised controlled trials. *Eur J Pain* 2009;13:3-10.
 41. Gerwin RD, Dommerholt J, Shah JP. An expansion of Simons' integrated hypothesis of trigger point formation. *Curr Pain Headache Rep* 2004;8:468-475.
 42. Srbely JZ, Dickey JP, Lowerison M, Edwards AM, Nolet PS, Wong LL. Stimulation of myofascial trigger points with ultrasound induces segmental anti-nociceptive effects: A randomized controlled study. *Pain* 2008;139:260-266.
 43. Srbely JZ, Dickey JP, Lee D, Lowerison M. Dry needle stimulation of myofascial trigger points evokes segmental anti-nociceptive effects. *J Rehabil Med* 2010;42:463-468.
 44. Chu J. Dry needling (intramuscular stimulation) in myofascial pain related to lumbosacral radiculopathy. *Eur J Phys Med Rehabil* 1995;5:106-121.
 45. Cagnie B, Dewitte V, Barbe T, Timmermans F, Delrue N, Meeus M. Physiologic effects of dry needling. *Curr Pain Headache Rep* 2013;17:34.
 46. Audette JF, Wang F, Smith H. Bilateral activation of motor unit potentials with unilateral needle stimulation of active myofascial trigger points. *Am J Phys Med Rehabil* 2004;83:368-374.
 47. Shah JP, Thaker N, Heimur J, Aredo JV, Sikdar S, Gerber L. Myofascial trigger points then and now: A historical and scientific perspective. *PM R* 2015;7:746-761.
 48. McCormack HM, Horne DJ, Sheather S. Clinical applications of visual analogue scales: A critical review. *Psychol Med* 1988;18:1007-1019.
 49. Huskisson EC. Measurement of pain. *Lancet* 1974;2:1127-1131.
 50. Fischer AA. Pressure threshold meter: Its use for quantification of tender spots. *Arch Phys Med Rehabil* 1986;67:836-838.
 51. Jaeger B, Reeves JL. Quantification of changes in myofascial trigger point sensitivity with the pressure algometer following passive stretch. *Pain* 1986;27:203-210.
 52. Todd KH. Clinical versus statistical significance in the assessment of pain relief. *Ann Emerg Med* 1996;27:439-441.

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CME Question

Which of the following prognostic factors will favor a sustained therapeutic response to dry needling?

- a. Higher baseline verbal analogue scale (VAS).
- b. Greater drop in VAS at follow up.
- c. Limited cervical range of motion.
- d. Female gender.

Answer online at me.aapmr.org