





A Comprehensive Protocol to Diagnose and Treat Pain of Muscular Origin May Successfully and Reliably Decrease or Eliminate Pain in a Chronic Pain Population

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Abstract

Objective. A comprehensive protocol is presented to identify muscular causes of regional pain syndromes utilizing an electrical stimulus in lieu of palpation, and combining elements of Prolotherapy with trigger point injections.

Methods. One hundred seventy-six consecutive patients were evaluated for the presence of muscle pain by utilizing an electrical stimulus produced by the Muscle Pain Detection Device. The diagnosis of "Muscle Pain Amenable to Injection" (MPAI), rather than trigger points, was made if pain was produced for the duration of the stimulation. If MPAI was found, muscle tendon injections (MTI) were offered to patients along with post-MTI physical therapy, providing neuromuscular electrical stimulation followed by a validated exercise program [1]. A control group, evaluated 1 month prior to their actual consultation/evaluation when muscle pain was identified but not yet treated, was used for comparison.

Results. Forty-five patients who met criteria completed treatment. Patients' scores on the Brief Pain Inventory decreased an average of 62%; median 70% (P < 0.001) for pain severity and 68%; median 85% (P < 0.001) for pain interference one month following treatment. These changes were significantly greater (P < 0.001) than those observed in the untreated controls.

Conclusion. A protocol incorporating an easily reproducible electrical stimulus to diagnose a muscle causing pain in a region of the body followed by an injection technique that involves the entirety of the muscle, and post injection restoration of muscle function, can successfully eliminate or significantly reduce regional pain present for years.

Key Words. Muscle Pain; Myofascial Pain Syndrome (MPS); Trigger Points (TrPs); Muscle Pain Amenable to Injection (MPAI); Muscle Tendon Injections (MTI); Low Back Pain; Muscle Pain Detection Device; MPDD

Introduction

The study of pain in the past 40 years has evolved into an important field of scientific inquiry leading to improved understanding of the mechanisms of various painful conditions and the development of commensurate treatment interventions. One potential area of promise however is alaringly overlooked. Muscle involvement in common clinical regional pain syndromes is neither generally studied nor considered as an important source of treatable pain. Many factors have made it difficult to conceptualize muscles as an integral part of the standard of evaluation and treatment protocols. Muscle exercise programs may be effective [1] but are rarely taught in medical training. Even when muscle involvement is considered, approaches to diagnosis and treatment are highly variable, leading to unexplainable results and suboptimal outcomes.

"Myofascial Pain" Lacks Universal Understanding

Varying concepts of myofascial pain [2–12] and oversimplification of the nomenclature are responsible for difficulties in the diagnosis and treatment of muscle pain. According to the core curriculum of the International Association for the Study of Pain, myofascial pain is defined as pain emanating from muscle and connective tissue that causes pain in common clinical regional pain syndromes and "lacks reliable means [for physicians] to identify, categorize, and treat such pain" [13] Studies of clinicians attempting to identify painful muscles demonstrate poor inter-rater reliability in the identification of myofascial trigger points (TrPs) [14–19] Clinicians will frequently and mistakenly use the terms, "myofascial trigger point" and "myofascial pain," interchangeably. "Myofascial trigger

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point" is only one of the four various potential etiologies of myofascial pain. The other three suggested etiologies of myofascial pain-deficiency (weakness and or stiffness) spasm, and tension [20]—are not typically appreciated by clinicians, leading to the disregard of critical causes of muscle pain, and the ensuing suboptimal outcomes. These issues collectively have contributed to the absence of muscle pain as a putative source of investigation in published pain treatment guidelines, such as the 2007 Low Back Pain Guidelines from the American Pain Society and the American College of Physicians [21]. The failure to create an agreed methodology for muscle pain assessment and treatment has contributed to the rejection of trigger point injections (TPIs) and sclerosant injections, as recommended treatment options for low back pain [22]. Ignoring muscles facilitates an overemphasis on structural abnormalities demonstrated on imaging and not necessarily identifying the true source of the patient's pain. Subsequent inappropriate treatments contribute to the \$86 Billion spent in 2005 on neck and back pain in the United States [23].

Possible Etiologies of Myofascial Pain are not Fully Recognized by Clinicians

Myofascial pain can be caused by various etiologies. However, the current community standard of establishing the diagnosis is limited to only palpating the putative muscle causing regional pain and identifying any TrPs. The standard treatment is to give TPIs to the putative muscle, injecting into a discrete area that includes only the TrPs and associated taut bands. The evaluation of TrPs without a complete assessment of muscle conditioning leads to unexplainable variability in treatment outcomes because diagnoses are confounded when clinicians fail to consider weakness, stiffness, spasm, or tension as a primary source of pain [24]. Therefore, even if the putative muscle is correctly identified and injected, failure to acknowledge, and/or appropriately treat pain from these other causes of myofascial pain may leave the patient with persistent discomfort and clinically unchanged.

Limits of Palpation as a Diagnostic Tool

Palpation alone used to detect areas of muscle pain introduces two confounding variables: First, varying amounts of pressure may be applied diminishing the reliability of the examination. Pressure-recording devices have been introduced to determine more accurately the amount of applied pressure necessary to elicit discomfort in the patient [25,26]. However, the accuracy of these devices is compromised because examiner preconceptions have been reported to influence the assessment [27]. Second, palpation to elicit a subjective experience of pain is done in a sedentary muscle. Most functional muscle pain is experienced with muscle activity vs rest. Therefore, an examination of a resting muscle is likely to be less accurate in determining the source of the muscle pain, frequently identifying a referred pain pattern, compared with an examination utilizing movement of discrete muscles [28,29].

It has been shown in humans with delayed onset muscle soreness that the presence of hardness in the muscle or muscular tendonous junction does not correlate with the presence of muscle hyperalgesia [30]. An unpublished observational study comparing traditional palpation with external electric stimulation of putative muscles demonstrated that among nearly 50% of the evaluated patients, the tenderness and taut band in the trapezius muscle was actually secondary to the primary source of pain in an adjacent muscle, which when injected eliminated the pain and taut band in the trapezius (data available upon request).

Since a patient is unable to isolate and move just one muscle, an electrical device was developed that can stimulate individual muscles to identify the involvement of one or more that are suspected as the source of pain in a clinical regional pain syndrome. We postulate that externally induced contraction of the putative muscle in the painful region produces pain by two means: 1) Muscle fiber contraction stimulating the density of sensitized nociceptors in the muscle-tendon and bony-tendon attachments, and 2) Stimulation of sensitized nociceptors in the muscle belly through deformation of the area where sensitized nociceptors in TrPs are located. The present study was conducted to test a new standardized evaluation and treatment algorithm that we believe accurately identifies and effectively treats pain from muscle involvement in common clinical regional pain syndromes.

Methods

Study Description and Patients

This nonrandomized, nonblinded, controlled study was designed to evaluate the effectiveness of a novel protocol for the diagnosis and treatment of myofascial pain. The study population was drawn from a pool of 176 consecutive patients seeking relief from a variety of chronic painful conditions at the lead author's (N.M.) pain practice. All patients were evaluated by the senior author (N.M.), a board certified pain medicine specialist, using a structured physical examination to detect potential muscle involvement as the cause of their pain syndromes. When muscle involvement was confirmed using the Muscle Pain Detection Device (MPDD) and the evaluation protocol suggested that injections were indicated, the muscle was identified as a "Muscle Pain Amenable to Injection" (MPAI), as opposed to "trigger point pain." Patients diagnosed with MPAI and without exclusionary criteria (see below) were offered treatment. This treatment consisted of muscle tendon injections (MTIs), followed by a structured physical therapy protocol that includes a validated set of exercises. Patients were excluded from the study if they had a concurrent physical diagnosis (including morbid obesity, severe deconditioning, Parkinson's disease, severe peripheral neuropathy or significant psychological co-morbidities) that discouraged aggressive treatment of the muscle pain. A control group was drawn from a pool of 79 patients using the same inclusion and exclusion criteria as in the treatment group of patients. The control group patients completed the Brief Pain Inventory (BPI) survey by mail or by phone at one month prior to their actual initial consultations, at which time they were retested with the BPI, now 1 month later, and assessed for MPAI. Informed consent was given for all procedures that patients received.

Structured Physical Examination

All 176 patients in the study pool and the 18 controls received a structured physical examination that consisted of the Kraus–Weber (KW) test for key trunk muscle strength and flexibility, Kraus examination protocol for neck and shoulder range of movement, neurological examination, standard palpation for muscle tenderness and resilience, and evaluation with the MPDD (SPOC, Inc. Stamford, CT), a hand-held biomedical device that locates muscle pain (the device picture is available to view in the supporting information link provided online only). Palpation for tenderness and resilience was performed only to identify presumptive sources of muscle pain, but the diagnosis of MPAI was only made with the MPDD.

Main Outcome Measures

The measurement of pain severity and interference with movement was assessed by the administration of the BPI [31,32]. The BPI assesses severity of pain and interference in normal movement as two dependent variables. The numerical results of the four severity questions were averaged to determine a "Severity Score" and the first seven interference questions were averaged to determine an "Interference Score." The BPI was administered to all patients in the study pool by the office manager at three time points: 1) initial visit, 2) last day of treatment, and 3) 1 month following the last day of treatment in person or by USPS mail.

Diagnosis of Myofascial Pain

See Figure 1.

For a detailed discussion of the evaluation technique including the KW test, see the supporting information link (provided online only).

Treatment Protocols

Injection Technique-Muscle Tendon Injections

See Figure 2.

For a detailed discussion of the injection technique, see the supporting information link (provided online only).

Patients who have MPAI diagnosed and no contraindications to MTI are injected in one or two muscles each day for a total of 1–5 muscles each week. The choice of which

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muscle to inject first is made according to the patient's complaint of their worst pain in a region coupled with the degree of discomfort experienced with the MPDD. Proximal and/or superficial muscles are injected prior to deep and/or distal muscles. Patients who travel long distances may receive multiple muscle injections (more than one muscle a day) in order to finish the treatment as quickly as possible and decrease the costs of food and lodging. Patients are reevaluated with the MPDD prior to each MTI to ensure that all previously identified muscles still test positive for MPAI.

Post Injection Physical Therapy

See Figure 3.

For more detailed discussion of the post injection physical therapy including the Kraus exercises, see the supporting information link (provided online only).

Statistical Methods

Treated and withdrawn groups were compared on baseline and treatment variables with chi-square tests (for nominal and yes/no variables) and either *t*-tests or Mann-Whitney *U*'s for numeric variables. The numeric variables are generally presented as mean (median) range, as many were quite non-normal in distribution. Within-group changes were analyzed with Wilcoxon tests. The control subjects only had data at baseline and one month later. Within group changes for this group were also analyzed with Wilcoxon tests. Mean changes were compared between controls and treated subjects with a Mann-Whitney *U*-test.

Results

Patient Characteristics

Of the 176 patients in the study pool, 106 patients were excluded because of the following reasons: 1) no primary muscle pain (n = 43); 2) primary muscle pain with MPAI was present, but patients had a concurrent physical diagnosis (n = 25) that included morbid obesity, severe deconditioning, Parkinson's disease and severe peripheral neuropathy, or significant psychological co-morbidities; and 3) primary muscle pain with MPAI was present, but patients refused suggested treatment (n = 38). Of the remaining 70 patients that began treatment, 45 patients (64.3%) completed the treatment and 25 patients (35.7%) chose to withdraw from the treatment program. As shown in Table 1, the average age of patients in the withdrawn group (51.3 years) was slightly older than the patients who completed treatment (44.7 years; P = 0.06), but there were few other differences. About half of the patients in the treatment group (58%) and withdrawn group (48%) were female, and both groups had a wide range of ages (age 12-76 years) and a wide range of duration of symptoms, with a median of 6 years of pain for both groups.

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I. Kraus–Weber Test \rightarrow Pass – No weakness or stiffness in key postural muscles \rightarrow Fail – Weakness and/or stiffness in key postural muscles \downarrow

(suggest administration of Kraus exercises)

II. Muscle Pain Detection Device applied to suspected muscles – used on initial consult and prior to each Muscle-Tendon Injections



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To each injected muscle for 3 consecutive post injection days: Rhythmic Sinusoidal Wave Neuromuscular Electrical Stimulation, 2 seconds on 2 seconds off, to a visible contraction with Ice x 20 minutes

Figure 3 Post injection physi-

cal therapy protocol.

First seven lower body exercises or eight upper body exercises

Patients Who Received Treatment

In the patients who completed treatment (Completers), the only gross neurological abnormality was a diminished lower extremity reflex in two patients. The vast majority of all patients reported pain for more than 1 year, with only two patients who completed treatment having pain duration of 3 months or less. Of the patients who withdrew, one patient had hyperreflexia of the lower extremities, and one patient had pain for 3 months or less. As shown in Table 1, some patients in both groups had undergone previous treatments, including TPIs. About 1/3 of both groups had evidence of depression. The withdrawn group had nonsignificantly higher rates of all prior diagnoses and treatments.

Discontinued Treatment

Patients in the withdrawn group were verbally asked for their primary reason for not continuing treatment and reported the following reasons: lack of time or distance from the pain center (n = 8); diagnosed with more urgent, prominent health condition (n = 7); felt the treatment was not working (n = 4); financial concerns prevented them from continuing the treatment (n = 3); elected to try another form of treatment rather than finish the prescribed treatment (n = 2); noncompliant with the post injection protocol (n = 1). Patients who chose to discontinue treatment were not reevaluated. At the beginning of treatment, all patients were informed in a written instruction sheet that "pain may not be relieved until all muscles that were identified as MPAI are injected, and treatment is completed." There were two patients with neurological abnormalities in the group of 38 patients who refused the suggested treatment. One patient had neurologic impairment of an arm and shoulder due to a car accident and one patient had diminished reflexes in one leg following a microdiscectomy. Pain duration was 14.8 years (median 13.5) with a range of 1.5 months to 51 years, with one patient having pain for less than 3 months. Similar to the treatment group and the withdrawn group, about 1/3 of the patients who chose not to have treatment had evidence of depression.

Main Outcome: Pain Severity and Interference Scores

Completers

Individual BPI scores for severity at initial consultation, completion of treatment, and 1-month follow up are

Ν	Treated 45	Withdrawn 25	Control 18
Age (v) ^a	44.7 (44) 12–71	51.32 (52) 26–76*d	51.61 (51) 20–79*d
Pain duration (y) ^a	9.2 (6) 0.1–37	8.34 (6) 0.25–30 ^{NS,b}	18.97 (18.7) 1.7–37** ^b
Sex: N (% female)	26 (58%)	12 (48%) ^{NS}	11 (61%) ^{№S}
Length of treatment (weeks)	2.1 (2) 1–8	1.8 (1) 1–7** ^b	N/A
Prior back surgery	7 (15.5%)	6 (24%) ^{NS}	7 (39%)*
Prev Dx Herniated disk	8 (17.8%)	7 (28%) ^{NS}	4 (22%) ^{NS}
Prev. Treatment with epidural steroids or facet blocks	14 (31.1%)	11 (44%) ^{NS}	10 (56%)*
Prior trigger point tx	4 (8.9%)	3 (12%) ^{NS}	1 (6%) ^{NS}
Depression ^c	13 (28.9%)	9 (36%) ^{NS}	7 (39%) ^{NS}
# Muscles identified with MPAI	6.7 (4.5) 1–35	7.1 (6) 1–27 ^{NS,b}	7.1 (6) 2–27 ^{NS,b}
# Muscles treated Pain location	5.6 (4) 1–31	4.6 (3) 1–18 ^{NS,b}	N/A
Upper body only	11 (24%)	4 (16%) ^{NS}	1 (6%) ^{NS}
Lower body only	24 (53%)	11 (44%)	11 (61%)
Both locations	10 (22%)	10 (40%)	6 (33%)

Table 1 Statistical analyses

* $P \le 0.10$ vs treated; ** $P \le 0.01$ vs treated. All others, P > 0.10.

^a Numeric variables are shown as mean (median) range.

^b Mann–Whitney U-test (those without a letter are chi-square, uncorrected or Yates corrected as appropriate).

^c Suffered with or medicated for.

^d Unpaired *t*-test.

NS = nonsignificantly different from treated.



Severity at 3 time points

Figure 4 Severity and interference at three times points in 45 patients who completed treatment. Each plots shows the median (dark bar), 75th and 25th percentile (top and bottom of box) and the range. P < 0.001 for post and one month vs initial (baseline) on each measure.

shown in Figure 4. The mean BPI score for severity at initial consultation was 4.95; median 5.00. After completion of treatment (MTIs and Physical Therapy) the mean BPI severity score had dropped to 2.02; median 1.25 (P < 0.001 vs baseline). The mean BPI score for severity at the 1-month follow up was 1.87; median 1.13 (also P < 0.001 vs baseline). Comparing completion of treatment with 1-month follow-up, no difference was observed (P = 1.0). The mean decrease in BPI severity of all patients was 62%; median 70%.

The distribution of BPI scores for pain Interference at all three time points in the patients who completed treatment is shown in Figure 5. At initial consultation the mean was 5.28; median 5.67. After completion of treatment (MTIs and Physical Therapy) the mean BPI interference score had dropped to 1.32; median 0.71, at the 1 month follow up the average was 1.57; median 0.57. As we found for severity, both posttest and 1 month were significantly different from baseline (P < 0.001 for each), but not from each other (P = 0.49). The average decrease in BPI rated pain interference of all patients was 68%; median 85%. As shown in the figure, prior to treatment, over 75% of the patients had values above 3 on severity and likewise on interference, whereas 75% of the patients were below 3 on these variables at post-treatment and 1 month.

Controls

For both severity and interference, there was effectively no average change in the control group (median change on



Figure 5 Change in severity pretreatment to 1 month post treatment (in 45 patients who completed treatment) or post evaluation (in the 18 untreated controls). Each figure shows the median (dark bar), 75th and 25th percentiles (top and bottom of box), and the range. P < 0.001 between groups on both change measures.

severity was –0.0625, a slight worsening, while the median change on interference was 0.1214. Mean changes were positive, but only 0.61 and 0.81, respectively). Neither change was even close to significant. The 95% confidence interval for the mean change suggests that the maximum plausible mean improvement in untreated controls would be 1.39 on severity and 2.00 on interference.

Completers vs Controls

Median changes on severity and interference in the completer group are both >2.5 (means are >3). Both are highly significant (P < 0.001), and both are significantly greater than the changes in the controls (P < 0.001). The lower bound of the 95% confidence interval, indicating the minimum plausible mean changes in the completer group are 2.37 for severity and 2.83 for interference. These minimums are greater that the upper end of the 95% confidence interval in the controls—in other words, the 95% confidence intervals are nonoverlapping.

Discussion

We present for the first time a muscle pain algorithm with a unique combination of features. It incorporates a diagnostic technique that utilizes an electrical stimulus, standardized for each patient, that detects multiple causes of functional muscle pain, a specific injection technique for identified painful muscles diagnosed with MPAI, and an aftercare program which includes specific exercises that have been demonstrated to be effective on patients with back pain.

For relevant physiology of nerve conduction and associated putative mechanism of MPDD effect, please go to the supporting information link (provided online only).

Physical Examination

An important aspect of the examination is the production of pain along the entire course of suspected muscle from origin to insertion in order to unambiguously identify a muscle as a source of pain. A partially blinded randomized controlled validation study has been completed demonstrating that the MPDD is significantly better than palpation in determining a muscle thought to be the source of pain in a region of the body [28]. For MPAI to be diagnosed in a muscle the entire course of the muscle from the origin to insertion must be experienced as painful (tender, aching, or soreness). Sustained pain produced by MPDD in only a portion of the muscle suggests that another muscle is the true source of the pain.

Injection Technique

Our premise is that proper injection technique and aftercare could lead to the elimination of the source of the pain and therefore the need for reinjection. We found that patients' completing our treatment protocol obtained both substantial pain relief and diminished interference in function as measured on the last day of treatment, both of

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which persisted at least 1 month, with our treatment protocol. We recognize that a longer follow-up period would be desirable. Of 45 patients, 11 (24.4%) exhibited complete elimination of pain that was present for years. The average duration of pain in 10 of the 11 patients who had complete elimination of pain was 8.9 years, with a range from 1 year to 18 years. The median decrease in pain severity for all patients was 70%, and the median decrease in pain interference was 85%, both highly significant statistically and little changed between the end of treatment and 1 month later.

Changes in a sample of untreated controls were far less, and the difference between completers and controls in change to 1 month was highly statistically significant. Our finding of a decrease in the total number of muscles identified as having MPAI over time in the course of the treatment may be related to central sensitization [33]. The elimination of the most painful muscles may result in diminished centrally facilitated pain and that therefore some of our identified MPAIs may be false positives. We believe therefore that the routine re-evaluation prior to each MTI for MPAI is an important part of our protocol.

We postulate that a longer duration of relief is achievable with this approach, vs palpation only, which generally results in transient pain relief. At least one published study used the return of 75% of the pre-injection pain as the dependent time variable in studying TPIs using different injectates [34] and other studies have commented on the need to reinject TrPs [35,36].

Published studies address the specific number of trigger points in a muscle [37], the importance of eliciting a "twitch response" [35], or of thoroughly injecting the "taut band" [38]. Our approach is modeled on that of HK. He had originally thought that injecting TrPs when present could successfully diminish or eliminate muscle pain. He observed that 50% of patients treated with TPIs would repeatedly return with the need for reinjection in the same muscle. He speculated that as the muscle-tendon and bone-tendon attachments had the least blood supply and in animal studies were most prone to rupture, vs the muscle belly, that these areas might also be the source of the recurrent pain pattern. He therefore modified his injection technique so that it always included the origin and the insertion of the identified painful muscle. Gibson et al. [39] reported that the Proximal Tendon Bone Junction and tendon sites are more sensitive and susceptible to sensitization by hypertonic saline than muscle belly. This observation is consistent with our clinical impression of the importance of the tendon bone junction in the course of muscle needling. The injection into the bony attachment of the muscle identified as having MPAI has similarities to prolotherapy injections in which a variety of injectates may be utilized. A sclerosing solution is frequently injected into tendons or ligaments that are found to be painful on palpation with the assumption that the injectate will tighten lax connective tissue or promote healing in damaged tissue. The treatment is always coupled with some form of exercise or spinal manipulation therapy in any of the pub-

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lished studies demonstrating success in relieving pain in a region of the body [40]. We agree that pain does originate in the tendon bone attachments but we suggest that the effect of prolotherapy is based on the placement of the needle into this area rather than the injectate [41].

The utility of exercise in the treatment paradigm makes sense, but prolotherapy (and TPI) protocols that suggest the use of exercise following injections, do not identify which specific exercise are indicated. Varying injectate, as well as exercise protocols may confound outcome data and eliminate the possibility of valid systematic review or meta-analysis.

Exercise is defined as a "series of movements to promote good physical health." This definition is problematic in that it allows almost any activity to be defined as an exercise protocol, thus accounting for the wide variety of outcomes achieved through "exercise" [42,43]. In 2007, van Tulder et al. [44] found that of 43 Cochrane-reviewed trials on exercise for the treatment of low back pain, 18 of the trials reported a positive response but only four showed any statistically significant reduction of pain. We believe that the nonspecific nature of the physical therapy programs provided in conjunction with muscle injections contributes to the inconsistent outcomes, even when apparently similar injection techniques are used.

The aftercare we provide is not generic stretching or a choice of techniques, but a structured, rigorous, albeit simple, exercise program performed following passive movement of the injected muscle using a neuromuscular electrical stimulator (NMES). NMES interestingly, following MTIs, reduces discomfort whereas electrical stimulation to the same muscle prior to MTI causes discomfort. We specifically prescribe an exercise program developed on 3,700 patients over the course of $4^{1}/_{2}$ years and given to 300,000 participants at the YMCA with an 80% success rate in minimizing or eliminating low back pain [1]. The exercises provide relaxation (to address tension), limbering (to address stiffness), and gentle stretching (to address shortened muscles) after the muscle with MPAI is needled, vs a generic instruction to stretch the effected muscle and or do home exercises [30], or the suggestion to do Kraus or McKenzie exercises [42]. Studies discussing the low quality of heterogeneous outcome measures overlook the role of exercising.

Our study does not address the various injectates that are typically utilized in TPIs. We agree with the Cochrane group that there is no significant difference in outcomes when using various injectates and that the needle itself is the critical factor in the various muscle and ligamentous injection techniques [45].

There are a number of limitations in our study. An "intent to treat" model was not used for this analysis, since 1-month data was not obtained for patients who withdrew. An "intent to treat" model would require using an extremely conservative strategy such as last observation carried forward, and would introduce more bias than simply excluding these patients. We asked why the patients were leaving the study and lack of efficacy was not the typical reason. We assume a patient would be less likely to withdraw if the treatment was wonderfully effective. However, patients are informed in writing that full treatment efficacy is not attained until after the complete course of treatment, and these patients withdrew before completion.

The data are preliminary in that there is no randomized control group or blinded treatment. In addition, a substantial number of subjects withdrew from the treatment group and have no follow-up data. We also note that the diagnostic and treatment protocol incorporates multiple variables, each of which should be investigated for their individual contributions to the overall outcome: The electrical detection device to identify MP and to determine possible etiologies of such pain (MPAI, TrPs, tension, stiffness, and/or spasm as the cause of pain. [We have noted above a successful RCT comparing MPDD to palpation in identifying MPAI]) [28], injections only of muscles identified as containing trigger points vs tension, stiffness or spasm through the use of the electrical device, the muscle injection procedure itself (entire muscle including the origin and insertion down to the bony attachment) and the injectate, 0.5% Lidocaine. The post-injection protocol variables that should be studied for their role in the total outcome are: use of a neuromuscular electrical stimulation to provide a rhythmic contraction and use of a specific structured exercise protocol.

The presented protocol was utilized successfully to avoid surgery in a group of pain patients whose pain was attributed to a variety of nonmuscle diagnoses and for whom surgery was recommended [46].

Conclusion

A protocol has been developed for the identification and treatment of muscle pain, utilizing electrical stimulation for potentially more precise identification of painful muscles, which appears to be successful at reducing and perhaps eliminating muscle based pain. Our data on 176 consecutive patients presenting to a comprehensive pain center indicate that 133 (76%) had MPAI, based on our model of identification of painful muscles with the use of electrical stimulation. It is estimated that 70-85% [21,47] of patients with low back pain have nonspecific low back pain. We suggest that a large percentage of these patients with not only low back pain, but upper back and shoulder pain as well, have a muscular component to their pain [28,29,48]. Although we only specifically address those patients with MPAI as a cause of pain, many of the patients had pain also from weakness stiffness, and tension, which we did not specifically analyze. The study of patients treated at the YMCA suggests that the Kraus exercises are an effective method to address these causes of muscle pain. Randomized controlled studies need to be performed to substantiate the suggested effectiveness of this comprehensive approach.

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References

- 1 Kraus H, Nagler W, Melleby A. Evaluation of an exercise program for back pain. Am Fam Physician 1983;28(3):153–8.
- 2 Gunn CC. Radiculopathic pain: Diagnosis and treatment of segmental irritation or sensitization. J Musculoskeletal Pain 1997;5(4):119–43.
- 3 Gunn CC. Reply to Chang-Zern Hong. J Musculoskeletal Pain 2000;8(3):137–42.
- 4 Gunn CC, Milbrandt WE, Little AS, Mason KE. Dry needling of muscle motor points for chronic low-back pain: A randomized clinical trial with long-term follow-up. Spine 1980;5(3):279–91.
- 5 Hong CZ. Comment on gunn's radiculopathy model of myofascial trigger points. J O Musculoskeletal Pain 2000;8(3):133–5.
- 6 Hong CZ. Treatment of myofascial pain syndrome. Curr Pain Headache Rep 2006;10(5):345–9.
- 7 Hopwood MB, Abram SE. Factors associated with failure of trigger point injections. Clin J Pain 1994; 10(3):227–34.
- 8 Kraus H, Fischer AA. Diagnosis and treatment of myofascial pain. Mt Sinai J Med 1991;58(3):235–9.
- 9 Marcus N, Kraus H. Letter to the editor in response to article by hopwood and abram. Clin J Pain 1995; 11(1):84.
- 10 Rachlin E, Rachlin I. Myofascial Pain and Fibromyalgia. St Louis, MO: Mosby; 2002:234–44.
- Simons DG, Travell J. Myofascial Pain and Dysfunction (The Trigger Point Manual), 2nd edition. Baltimore, MD: Lippincott Williams & Wilkins; 1999:5.
- 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(3):278–86.
- 13 International Association for the Study of Pain (IASP). Core Curriculum, 3rd edition. Seattle: IASP Press; 2005.

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- 14 Christensen HW, Vach W, Manniche C, et al. Palpation for muscular tenderness in the anterior chest wall: An observer reliability study. J Manipulative Physiol Ther 2003;26(8):469–75.
- 15 Levoska S. Manual palpation and pain threshold in female office employees with and without neck-shoulder symptoms. Clin J Pain 1993;9(4):236–41.
- 16 Maher C, Adams R. Reliability of pain and stiffness assessments in clinical manual lumbar spine examination. Phys Ther 1994;74(9):801–9; discussion 9–11.
- 17 Marcus N, Kraus H, Rachlin E. Comments on K.H. Njoo and E. Van der Does, Pain, 58 (1994) 317–323. Pain 1995;61(1):159.
- 18 Njoo KH, Van der Does E. The occurrence and interrater reliability of myofascial trigger points in the quadratus lumborum and gluteus medius: A prospective study in non-specific low back pain patients and controls in general practice. Pain 1994;58(3): 317–23.
- 19 Wolfe F. Stop using the American College of Rheumatology criteria in the clinic. J Rheumatol 2003;30(8): 1671–2.
- 20 Kraus H. Diagnosis and Treatment of Muscle Pain. Chicago, IL: Quintessence Books; 1988:11–37.
- 21 Chou R, Qaseem A, Snow V, et al. Diagnosis and treatment of low back pain: A joint clinical practice guideline from the American College of Physicians and the American Pain Society. Ann Intern Med 2007; 147(7):478–91.
- 22 van Tulder MW, Koes B, Seitsalo S, Malmivaara A. Outcome of invasive treatment modalities on back pain and sciatica: An evidence-based review. Eur Spine J. 2006;15(suppl 1):S82–92.
- 23 Martin BI, Deyo RA, Mirza SK, et al. Expenditures and health status among adults with back and neck problems. JAMA 2008;299(6):656–64.
- 24 Rachlin E, Rachlin I. Myofascial Pain and Fibromyalgia, 2nd edition. St. Louis, MO: Mosby; 2002:438.
- 25 Fischer AA. Documentation of myofascial trigger points. Arch Phys Med Rehabil 1988;69(4):286–91.
- 26 Jensen K, Andersen HO, Olesen J, Lindblom U. Pressure-pain threshold in human temporal region. Evaluation of a new pressure algometer. Pain 1986; 25(3):313–23.
- 27 Orbach R, Crow H. Examiner expectancy effects in the measurement of pressure pain thresholds. Pain 1998;74:163–70.

Marcus et al.

- 28 Hunter C, Dubois MD, Zou S, et al. A RCT of a new muscle pain detection device to diagnose muscle as a source of back and or neck pain. Pain Med 2010 (in press).
- 29 Simons D, Travell J. Myofascial Pain and Dysfunction (The Trigger Point Manual). Baltimore, MD: Lippincott Williams & Wilkins; 1999:166.
- 30 Andersen H, Arendt-Nielsen L, Danneskiold-Samsoe B, Graven-Nielsen T. Pressure pain sensitivity and hardness along human normal and sensitized muscle. Somatosens Mot Res 2006;23(3–4):97–109.
- 31 Keller S, Bann CM, Dodd SL, et al. Validity of the brief pain inventory for use in documenting the outcomes of patients with noncancer pain. Clin J Pain 2004;20(5):309–18.
- 32 Tan G, Jensen MP, Thornby JI, Shanti BF. Validation of the Brief Pain Inventory for chronic non-malignant pain. J Pain 2004;5(2):133–7.
- 33 Mense S, Hoheisel U. Mechanisms of central nervous hyperexcitability due to activation of muscle nociceptors. In: Graven-Nielsen T, Arendt-Nielsen L, Mense S, eds. Fundamentals of Musculoskeletal Pain. Seattle, WA: IASP Press; 2008:61–73.
- 34 Graboski CL, Gray DS, Burnham RS. Botulinum toxin A versus bupivacaine trigger point injections for the treatment of myofascial pain syndrome: A randomised double blind crossover study. Pain 2005;118(1– 2):170–5.
- 35 Hong CZ. Lidocaine injection versus dry needling to myofascial trigger point. The importance of the local twitch response. Am J Phys Med Rehabil 1994;73(4): 256–63.
- 36 Hong CZ. Consideration and recommendation of myofacial trigger point injections. J Musculoskeletal Pain 1994;2:29–59.
- 37 Kamanli A, Kaya A, Ardicoglu O, et al. Comparison of lidocaine injection, botulinum toxin injection, and dry needling to trigger points in myofascial pain syndrome. Rheumatol Int 2005;25(8):604–11.
- 38 Fischer AA. New Injection Techniques for Treatment of Musculoskeletal Pain. Philadelphia, PA: Mosby; 2002.
- 39 Gibson W, Arendt-Nielsen L, Graven-Nielsen T. Referred pain and hyperalgesia in human tendon and muscle belly tissue. Pain 2006;120(1–2):113–23.
- 40 Dagenais S, Mayer J, Haldeman S, Borg-Stein J. Evidence-informed management of chronic low back pain with prolotherapy. Spine J 2008;8(1):203–12.

- 41 Peloso P, Gross A, Haines T, et al. Medicinal and injection therapies for mechanical neck disorders. Cochrane Database Syst Rev 2007;3:CD000319.
- 42 Hayden JA, van Tulder MW, Malmivaara A, Koes BW. Exercise therapy for treatment of non-specific low back pain. Cochrane Database Syst Rev 2005;3: CD000335.
- 43 Abenhaim L, Rossignol M, Valat JP, et al. The role of activity in the therapeutic management of back pain. Report of the International Paris Task Force on Back Pain. Spine. 2000;25(suppl 4):1S–33S.
- 44 van Tulder M, Malmivaara A, Hayden J, Koes B. Statistical significance versus clinical importance: Trials on exercise therapy for chronic low back pain as example. Spine 2007;32(16):1785–90.
- 45 Peloso PM, Gross AR, Haines TA, et al. Medicinal and injection therapies for mechanical neck disorders: A Cochrane systematic review. J Rheumatol 2006;33(5): 957–67.
- 46 Marcus NJ. Failure to diagnose pain of muscular origin leads to unnecessary surgery. Pain Med 2002; 3(2):161–6.
- 47 Deyo RA, Weinstein JN. Low back pain. N Engl J Med 2001;344(5):363–70.
- 48 Rosomoff HL, Fishbain DA, Goldberg M, Santana R, Rosomoff RS. Physical findings in patients with chronic intractable benign pain of the neck and/or back. Pain 1989;37(3):279–87.

Supporting Information

Additional Supporting Information may be found in the online version of this article:

Physiology of Muscle Nociceptors Related to Effects of MPDD Diagnostic Technique.

Image 1. Muscle pain detection device.

Evaluation Technique. Injection Technique.

Image 2. Infraspinatus muscle tendon injection protocol. **Post Injection Physical Therapy.**

Appendix 1. Kraus-Weber test.

Appendix 2. Instruction sheet for patients receiving muscle tendon injections.

Appendix 3. Kraus–Weber program level 1.

Appendix 4. Kraus–Weber program upper body. **References.**

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Physiology of Muscle Nociceptors Related to Effects of MPDD Diagnostic Technique Animal models of slowly conducting afferent (bringing back info to brain) units in muscle provide a possible explanation of how externally generated contraction (using our device) could help identify a painful muscle. Studies of muscle fiber physiology indicate that type III and IV slowly conducting afferents can be divided into four distinct functional groups(1) one of which is contraction sensitive. These fibers are not true nociceptors. However, some Group IV receptors excited during ischemic contraction(2) are probably true nociceptors. In addition, it has been shown that there is a greater density of muscle nociceptors(3) and greater sensitivity to hypertonic saline(4) in muscle-tendon junction and in bony tendinous attachments than in muscle belly. The nociceptors in the cat gastrocnemeus found on the calcaneal tendon and surrounding connective tissue are sensitive to contraction particularly when sensitized.

Graven-Neilsen reports that in humans thick myelinated (non-nociceptive) afferent fibers are activated at lower electrical stimulus intensities than unmyelinated afferents. The effects of various anesthetic blockades do differentiate between sensitivity of skin vs. muscle to electrical stimuli(5) and in our experience electrical stimuli that are just strong enough to produce a non-painful contraction in a neutral muscle may produce a mildly unpleasant stinging or burning sensation which we believe is from skin in contrast to the deep ache, soreness, or tenderness that patients report when a muscle thought to be the source of the pain is stimulated.

2

The device although applied on the surface is engineered to focus the electrical stimulation beneath the skin (interferential current). The MPDD encompasses technology based on Interferential Therapy (IFT), which allows for stimulation to deep tissues unlike other surface stimulation devices (i.e. TENS) that stimulate sensory fibers. It is well documented that at kilohertz frequencies skin impedance is minimized(6, 7) therefore at low frequencies, skin impedance is high, whereas at high frequencies, the skin impedance is lower.(8, 9)

Interferential currents (IFC) are produced by crossing two frequencies of alternating current with one another and creating an amplitude-modulated, low-frequency current (beat frequency).(10) Normally, this is done through two pairs of electrodes where one pair of electrodes would be set at a medium frequency and the other pair is set at a different medium frequency, the resultant being a lower frequency. The MPDD eliminates the use of four electrodes by producing amplitude modulated current within the unit and the output is known as premodulated IFC.(10) The MPDD also incorporates a metal electrode and standard electrode versus two standard electrodes. The metal electrode allows for a physician to move the MPDD along a muscle while the standard electrode completes the circuit.

Image 1: Muscle Pain Detection Device



Evaluation Technique

The MPDD was used to provide electrical stimulation to an individual muscle along the entire length of the muscle from the origin to the insertion. The device is calibrated for each individual patient using an uninvolved muscle. The electrical current is adjusted to produce a mild visible contraction, generally 12-20 milliamps at 150 hertz, while moving the MPDD over the uninvolved muscle. In regions where muscles overlap, testing of individual muscles was continued until all suspected muscles were evaluated. If continued muscle stimulation resulted in continuous pain, the muscle received the MPAI diagnosis. If continued stimulation resulted in diminution and subsequent disappearance of the pain in one minute or less, the diagnosis of MPAI was ruled out and the cause of the pain was assumed to be from one or more other causes (tension, stiffness, or spasm) described by the Kraus model for functional muscle pain.(11)

Injection Technique (12, 13)

Only one muscle is injected at any injection treatment. A needle that is long enough to reach the bony attachment of the muscle, between 25 gauge x 5/8 inch and 20 gauge x 3 ¹/₂ inch, is used depending on the size of the identified muscle. The treatment consists of using the needle to disrupt the muscle tissue with particular attention to the origin and insertion. The injection is referred to as a muscle tendon injection (MTI) because of the significant difference in location of the injections vs. TPIs. An entire muscle versus just a "point or taut band" is injected. The patient may first receive an opioid to decrease the discomfort of the procedure; the dose is determined by the patient's past response to opioids, generally meperidine 20-100 mg intravenously. The area is swabbed with iodine. Then, 10 cc's of 0.5 % lidocaine is injected into the subcutis overlying the indexed muscle. After 5-8 minutes, the muscle is needled from the origin to its insertion point, including the muscle belly with an additional 10 cc's of 0.5 % lidocaine for comfort, injected down to the bony attachment. To illustrate the treatment technique, consider the example of giving an MTI to the infraspinatus (Image2): After instilling SC lidocaine, the muscle is injected at the vertex of the scapula with a 22 gauge, $1\frac{1}{2}$ inch needle and with the needle still inserted, moving the needle along the medial and lateral borders of the scapula and withdrawing and reinserting the needle as one proceeds up towards the spine of the scapula and the rotator cuff. Ice is applied for 4 minutes after the injection. The area is cleansed and when all bleeding stops, the stable patient is released.

Image 2 Infraspinatus Muscle Tendon Injection Protocol

The numbers represent the suggested sequence in the injection procedure



Post Injection Physical Therapy

The MTI procedure causes pain during the procedure, and also afterwards to some degree. In order to facilitate additional injections and mobilization afterwards, the patient receives physical therapy on the day following the MTI, or 2 hours after the MTI if a second MTI is given on the same day. The physical therapy consists of the patient receiving neuromuscular sine wave stimulation, 2 seconds on and 2 seconds off, with ice, to a visible contraction for 15 to 20 minutes followed by the first seven Kraus exercises for the lower body or the eight exercises for the upper body. Treatment always commences on a Monday to allow more than one muscle to be injected per week and allow time for the required three post-MTI physical therapy sessions to be completed for each MTI. Therefore, treatment is considered "complete" on the final day of the post-MTI physical therapy session of the last week that injections are given. Patients are given further instructions on the final day of physical therapy for the remaining 14 additional exercises.

Appendix 1

Kraus-Weber Test

SIX BASIC MUSCLE TESTS

These six standardized tests of muscular function may help to "pinpoint" deficiencies of strength or flexibility (Test 6). They are done as slowly and smoothly as possible. Avoid jerky movements. Do not strain. Stop and rest briefly after each test.



Scoring Key:

A failed test occurs with any one of the following: 1. <10 seconds, 2. <90°, 3. <90°, 4. <10 seconds , 5. <10 seconds, 6. < toe touch.

<u>Appendix 2</u> Instruction Sheet for Patients Receiving Muscle Tendon Injections

- 1. Nothing to eat or drink for 4 hours before your appointment.
- 2. You should not be on Aspirin, Coumadin, or any other anticoagulant medication or herbal supplements. If you are, this needs to be discussed before treatments are begun.
- 3. It is not advisable for you to use public transportation or to drive yourself after you are injected. Therefore, you should arrange for transportation (i.e., cab, car service, friend to drive...).
- 4. You may have some increased discomfort initially after the injection. Use an ice pack to the area every hour as needed. Apply it directly to the skin, for 8 minutes only (to prevent a burn). We can give you a prescription for medication if needed.
- 5. You must return for Physical Therapy for the 3 days following each injection. Transportation restrictions to and from your appointment will still apply. Please schedule the physical therapy appointments at the same time that you schedule your appointment for the injection(s).
- 6. Do not perform any exercise except for the ones you will be shown by the physical therapist. For three days, avoid remaining in any one position for any extended period of time; for example,
 - a. Do not sit, stand, or lie down for more than 15 minutes *during waking hours*; changing position is important for the success of the treatment.
 - b. Do not walk for more than two blocks without taking a 30 sec break.
 - c. Carry as little as possible. Do not carry heavy handbags.
- 7. You may continue to work if you are able to abide by these restrictions. If your work is contraindicated, a letter may be addressed to your employer explaining the medical necessity of time off from work.

Your ability to abide by these restrictions is important for success of this treatment.

Please note that often a number of injection sessions are needed. Treatment may take one to two months. Dr. Marcus will be able to give you an approximation as to the number of treatments needed. However, an exact number cannot be predicted because of the nature of this treatment.

YOUR PAIN MAY NOT BE RELIEVED UNTIL ALL INJECTIONS ARE COMPLETED.

Appendix 5 Krause Exercise i rogram Level 1		
C.	1. <u>Diaphragmatic Breathing</u> Inhale through your nose (belly gets round). Exhale slowly through your mouth.	
	2. <u>Shoulder Shrugs</u> Pull your shoulders up toward your ears. Inhale and exhale as you let go.	
	3. <u>Leg Slides</u> Breathing out, let one knee fall to the side and then slide your leg all the way down. Relax and let it go. Slide it back to the basic position. Repeat with the opposite leg.	
	4. <u>Head Rotations</u> Drop your head to the right side, return back to neutral and let go, then drop the head to the left side and back to the center.	
	5. <u>Single Knee To Chest</u> Begin in the basic position. Bring one knee to the chest as far as you can comfortably. Lower the foot to the floor, then breathing out, let one knee fall to the side and then slide your leg down and let it go. Slide the leg back to the basic position. Repeat with the opposite leg.	
	6. <u>Side lying Knee To Chest</u> Lie on your side in the fetal position. Bring your upper leg toward your chest, letting the knee drop toward the floor like dead weight. Then slide the leg back, extending the knee. Bring the leg back to starting position and let it go. Do this 3 times, then roll onto your other side and repeat with opposite leg.	
	7. <u>Buttocks Squeeze</u> Turn onto your stomach and place a pillow under your belly. Tighten your seat muscles and hold for 2 seconds. Relax and let go.	

Appendix 3 Krause Exercise Program Level 1

 pres Bowl
1. <u>Diaphragmatic Breathing</u> Inhale through your nose (the belly gets round), exhale slowly through your mouth.
2. <u>Shoulder Shrugs</u> As you are inhaling pull your shoulders up toward your ears, exhale and let go.
3. <u>Head Rotations</u> Drop your head to the right side, return back to neutral and let go, then drop the head to the left side and back to center.
4. <u>Elbow Bend</u> As you are inhaling close fists and bend elbows, as you are exhaling - let go.
5. <u>"Chicken Wings"</u> Basic position with hands on the chest. Slide the arms out to the sides as you inhaling and bring them back on the exhale.
6. <u>Horizontal Abduction - Adduction</u> Bring the arm across the chest and bring it back (out to the side).
7. <u>Shoulder Rotation</u> Abduct the arm with elbow flexed. Rotate in and out.
8. <u>Shoulder Bend</u>
In basic position elevate your arms with elbows straight, up above the head during the inhale. Return back to basic position on the exhale. If it is too difficult or painful, lower both hands and let the strong/less painful side assist with raising and lowering the arms.

Appendix 4 Krause Exercise Program Upper Body

References

1. Mense S, Meyer H. Different types of slowly conducting afferent units in cat skeletal muscle and tendon. J Physiol. 1985 Jun;363:403-17.

2. Mense S, Stahnke M. Responses in muscle afferent fibres of slow conduction velocity to contractions and ischaemia in the cat. J Physiol. 1983 Sep;342:383-97.

3. Mense S, Simons DG. Muscle Pain Understanding its Nature, Diagnosis, and Treatment. Baltimore: Lippincott Williams & Wilkins; 2001.

4. Gibson W, Arendt-Nielsen L, Graven-Nielsen T. Referred pain and hyperalgesia in human tendon and muscle belly tissue. Pain. 2006 Jan;120(1-2):113-23.

5. Graven-Nielsen T, Arendt-Nielsen L, Mense S. Fundamentals of Musculoskeletal Pain. Seattle: IASP Press; 2008.

6. Low J, Reed A. Electrotherapy explained. 2nd ed. Oxford: Butterworth-Heinemann Ltd.; 1994.

7. Ward AR, Robertson VJ. Sensory, motor, and pain thresholds for stimulation with medium frequency alternating current. Arch Phys Med Rehabil. 1998 Mar;79(3):273-8.

8. Keller S, Bann CM, Dodd SL, Schein J, Mendoza TR, Cleeland CS. Validity of the brief pain inventory for use in documenting the outcomes of patients with noncancer pain. Clin J Pain. 2004 Sep-Oct;20(5):309-18.

9. Ozcan J, Ward AR, Robertson VJ. A comparison of true and premodulated interferential currents. Arch Phys Med Rehabil. 2004 Mar;85(3):409-15.

10. Tan G, Jensen MP, Thornby JI, Shanti BF. Validation of the Brief Pain Inventory for chronic nonmalignant pain. J Pain. 2004 Mar;5(2):133-7.

11. Rachlin E, Rachlin I. Myofascial Pain and Fibromyalgia. 2nd ed. St Louis: Mosby; 2002:437.

12. Rachlin E, Rachlin I. Myofascial Pain and Fibromyalgia. St.Louis: Mosby; 2002: 234-244.

13. Marcus NJ. Failure to diagnose pain of muscular origin leads to unnecessary surgery. Pain Med. 2002 Jun;3(2):161-6.