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ARTICLE

Reduction of Spontaneous Electrical Activity and Pain Perception of Trigger Points in the Upper Trapezius Muscle through Trigger Point Compression and Passive Stretching

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ABSTRACT. Objectives: Investigate the effects of ischemic compression [IC] technique and passive stretching [PS] in isolation and in combination on the reduction of spontaneous electrical activity [SEA] and perceived pain in trigger points [TrPs] located in the upper trapezius muscle.

Methods: Ninety participants with TrPs in the upper trapezius muscle were randomly assigned to three treatment groups: IC, PS, and IC + PS. TrP compression was applied on the TrP for three applications of 60 seconds each, followed by a 30-second rest period. PS was applied for three 45-second applications, with 30-second rest intervals. All patients received the same amount of therapy.

Results: Significant decreases were found in pain perception and on SEA for all study participants. The IC + PS group evidenced greater declines in pain perception and SEA when compared to the IC and PS groups.

Conclusion: Because of ethical considerations, a control group design was not possible, thereby limiting the robustness of the findings. Although each technique significantly reduced pain perception and SEA, the combination of IC and PS was superior, apparently because of the complementary nature of the therapeutic interventions.

KEYWORDS. Pain perception, ischemic compression, stretching, trapezius, myofascial pain

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Journal of Musculoskeletal Pain, Vol. 16(4), 2008
Available online at http://jmp.haworthpress.com

doi: 10.1080/10582450802479594

INTRODUCTION

Approximately 45 million Americans have chronic headaches; this is approximately 17 percent of the United States population (1). Tension headaches can have their etiology in trigger points [TrPs] in the upper trapezius muscle (2). Other symptoms generated by TrPs in the upper trapezius muscle include dizziness, tinnitus, and pain in the jaw, shoulder, upper arm, back of the neck, and mastoids. Because the upper trapezius muscle is a common source of pain, it is the object of this study.

Although researchers have mapped out TrPs in the body and knowledge about their structure, pathologies, and interventions are expanding, there has been relatively little research on appropriate interventions for TrPs in the trapezius muscle. In addition, such treatment modalities as ischemic TrP compression [IC] and passive stretching [PS], while commonly used in the treatment of TrPs, have not been subject to rigorous empirical investigation, although studies using impressionistic data have been published (3, 4). TrP compression is a therapeutic manipulative technique designed to release muscle tension by inactivating the TrPs that cause taut bands that increase muscle tension. The procedure is also called the TrP release technique (2). PS, also known as myofascial stretching (5), is directed at a specific muscle under treatment that avoids overstretching and requires absolute relaxation of the muscle. The target muscle is placed where tension is sensed at the end of the range of motion [ROM]. The muscle is allowed to relax while stretching is increased and the subject exhales. The newly gained position is held while the subject exhales. In subsequent movements, further gain is obtained by holding the position for 20–45 seconds at a rate of 3–4 mm/second, and then allowing the muscle to relax (6).

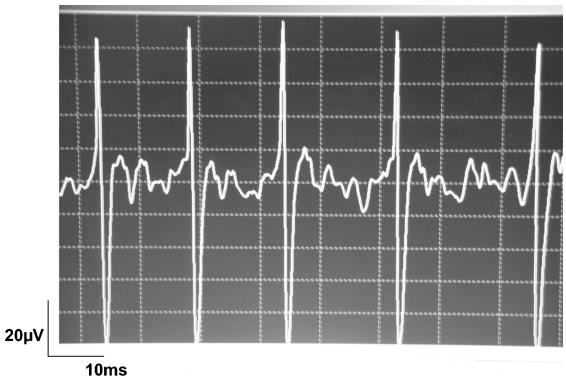
The theory that underlies this study is that TrPs are the consequence of microtraumas resulting from overstretching, overloading, or overshortening of skeletal muscles (2, 5, 7). The criteria for a TrP include a palpable taut band of muscle, a local tender spot located within the taut band, a pattern of referred pain resulting from pressure on the TrP, a local twitch response [LTR] resulting from snapping palpation or needle insertion in the TrP, and a reduction in the muscular ROM (7). Electromyography [EMG] has been used to confirm TrPs through the measurement of spontaneous electrical activity [SEA] and to explore the pathophysiologic mechanisms of TrPs (8, 9).

TrPs in the upper trapezius muscle are most commonly associated with tension headaches (2). In this case, intense, referred pain can be felt upward along the neck to the mastoid process, centering in the lateral wings of the sphenoid bone and behind the eye globes. Tension may be experienced in the angle of the neck when cradling a phone or using a pinching motion between the neck and shoulder.

The presence of local pain at the TrP and referred pain can lead to muscle guarding and loss of flexibility in the patient. Compensatory mechanisms on the part of the patient may result in further injury and creation of additional microtraumas, creating a vicious cycle of trauma, compensation, and further trauma (2, 5). A diagram of the process is presented in Figure 1.

The purpose of this investigation is to examine the effects IC and PS in isolation and in combination have on the reduction of SEA and perceived pain in TrPs located in the upper trapezius muscle. The following hypotheses were tested in this study: (a) The IC will reduce SEA and perceived pain in a TrP region in the upper trapezius muscle; (b) PS will reduce SEA and perceived pain in a TrP region in the upper trapezius muscle; (c) A combination of IC and PS will reduce SEA and perceived pain more than either individually.

FIGURE 1. Significantly increased SEA of a patient's upper trapezius muscle before treatment.



METHODS

The Sample

The sample contained participants who were presenting for neck pain or headaches and who had confirmatory evidence of at least one TrP in the upper trapezius muscle. The inclusion criteria included a positive response of at least two of the following essential criteria and at least one of the confirmatory criteria listed below (2, 5):

Essential Criteria

- Palpable taut band
- Exquisite spot tenderness of a nodule in a taut band
- Pain pattern recognition by patient
- Painful limit to full stretch ROM

Confirmatory Criteria

- Visual or tactile identification of LTR
- Imaging of an LTR induced by needle
- Pain or altered sensation [in the expected TrP distribution] with IC
- EMG spontaneous activity

On the basis of prior research (10–16), we expected substantial changes in SEA and perceived pain from pretest to posttest. Therefore, we expected a moderate-to-large effect size of 0.35.

Any potential participant who was taking medication other than nonsteroidal anti-inflammatory drugs for pain or had other neuromuscular pathology was excluded from the study. All participants were patients of Hands-On Physical Therapy, P.C., which has eight centers located in New York City metropolitan area. Participants were randomly assigned to one of three treatment groups: IC only, PS only, and IC combined with PS [IC + PC]. Group assignment was conducted serially with the first patient assigned to IC, the second patient assigned to PS, the third patient assigned to IC + PC, and so on, until all participants had been assigned a group. Although subjects were told the type of treatment they were to receive, they did not know that other subjects were assigned to alternative groups receiving other forms of invention. Though random assignment is expected to randomly distribute potentially confounding variables, checks were made comparing the three groups on gender, age, educational background, and ethnicity. No significant differences were found between the groups.

Instrumentation

Pain Perception

A CommanderTM Algometer [JTECH Medical Industries, Salt Lake City, UT] was used to assess the pressure–pain threshold [PPT] of TrPs and pressure tolerance. It consists of a 0.5-cm^2 rubber tipped plunger mounted on a calibrated spring. The gauge is calibrated in pounds per square centimeter. The JTECH device was designed in accordance with Fischer's (17) validity protocols. The PPT is the minimum pressure required to cause pain. Pressure tolerance is the maximum pressure that can be tolerated under clinical conditions. Both measures were conducted on the left and right upper trapezius muscle.

Several researchers have conducted reliability studies of pressure algometry, all of which demonstrated high levels of reliability for a variety of muscles (17–20). Sciotti et al. (20) assessed the reliability and validity of pressure algometry on the upper trapezius muscle using four physicians and 20 subjects who had prior diagnosis of latent TrPs or no TrPs; the latter subjects were used as controls. The authors concluded that palpation with pressure algometry reliably and validly identified TrPs in the upper trapezius muscle.

Perception of pain was assessed using the pain visual analog scale [PVAS]. The PVAS consists of a 10-cm line on which a respondent indicates the intensity of pain. Respondents were asked to indicate the intensity by responding to the direction, "On the scale below please indicate how intense or strong the pain you are now experiencing from your shoulder, neck, or head is." The 10-cm line spanned two polar opposites from "None" to "The worst pain ever."

The PVAS has been in use by researchers for many years and is used as a standard criterion for the assessment of pain when researchers attempt to validate other pain assessment tools. For example, Turchin et al. (21) used it to assess elbow pain, Paice and Cohen (22) used it to validate a rating of pain among cancer patients, and Gragg et al. (23) employed it as a criterion for

assessing a pediatric pain questionnaire. Flandry et al. (24) compared the PVAS with the Noyes (25) Knee Scale. In a test of predictive validity, correlations were low to moderate with two high [running and climbing stairs] and negative, indicating that the higher the pain perception, the lower the knee function, indicating predictive validity of the PVAS. These were a few of many studies that employed the PVAS as a criterion for validity of other pain indicators.

Spontaneous Electrical Activity

Spontaneous electrical activity (SEA) was measured using a Cadwell Sierra II EMG/EP [Cadwell Laboratories, Kennawick, WA] neurodiagnostic instrument and a Cadwell monopolar disposable needle. The TrP was probed in accordance with the directions of Hong and Simons (8), "A special technique using high-sensitive recordings and a very gentle insertion movement of the recording needle is required to record SEA. The recording needle should be moved slowly and gently [by fractions of a millimeter] during the search for SEA, because a fast movement may miss this small signal or may elicit an LTR instead. As the recording needle approaches responsive locus where SEA can be recorded, the amplitude of SEA progressively increases and the . . . noise becomes louder during that needle movement."

SEA data were analyzed using the technique reported by Chen et al. (26). Intramuscular electrical activity was recorded using Cadwell 25-mm, disposable, monopolar Teflon-coated EMG needle electrodes. The EMG unit was set with the following parameters: Low-cut frequency filter at 100 Hz and high-cut frequency filter at 1,000 Hz. These levels were selected in order to improve baseline stability and reduce baseline noise level. The gain was generally set at 20 μV per division and the sweep speed at 10 ms per division. Room temperature was maintained at 21 $\pm$ 1°C. The needle electrode was connected to the preamplifier. Reference and ground electrodes were placed to adjacent tissues. The needle insertion was done at an extremely slow rate to avoid any LTR.

Researchers have questioned whether SEA is normal or pathological. In 1957, Weeks and Travell (27) reported high-frequency spike-like EMG discharges on TrP areas while adjacent areas were electrically silent. Hubbard and Berfoff (9) reported similar activity identifying it as a characteristic of TrPs. Simons et al. (28), using higher amplification and larger sweep speed than the previous researchers, identified both a lower amplitude [<60 μV] noise-like activity along with high-amplitude spike-like potentials. These potentials were identified in areas with TrPs and corresponded to the potentials that are recognized by electromyographers as normal motor endplate potentials and endplate spikes. Wiederholt (29) identified these potentials as normal electrical activity.

Simons et al. (28), however, concluded that Wiederholt's (29) observation that these potentials derive from normal muscle endplates is correct only for the low-amplitude monophasic potentials. The continuous noise-like endplate potentials that Wiederholt (29) also suggested were normal, presented an entirely different noise-like configuration, and were the products of an abnormal origin. Liley (30), Heuser and Miledi (31), and Ertekin et al. (32) reported that mechanical, chemical, and thermal stimuli near the endplate region can cause such abnormal noise-like continuous potentials. These potentials can be the result of excessive release of ACh packets. When sufficient packets of ACh are released during depolarization of the postjunctional membrane, production of spike-like potentials may be observed. Hong (33) has demonstrated that clinically identified TrPs consist of multiple sensitive spots. These sensitive spots are abnormal endplates evincing SEA that can be scattered among uninvolved normal endplates (34).

The presence of SEA was identified if noise-like potentials persisted continuously for at least 300 ms and the amplitude was greater than 20 μV. This was at least four times the instrumentation noise level which averaged lower than 5 μV, observed in control recordings that took place before and after the SEA investigation. To assess the SEA, the maximum amplitude of a set of SEA potentials [in one screen with duration of 100 ms] with a stable baseline was measured. All the peaks of both positive and negative identifiable potentials were connected to form an envelope to wrap the SEA of one EMG screen. The maximal height of the envelope was measured as the maximal amplitude of the SEA. If the maximal amplitude was less than 20 μV, it did not fit our definition for SEA and a value of

FIGURE 2. Reduced but still significant SEA in a patient's upper trapezius after the delivery of six treatment sessions.

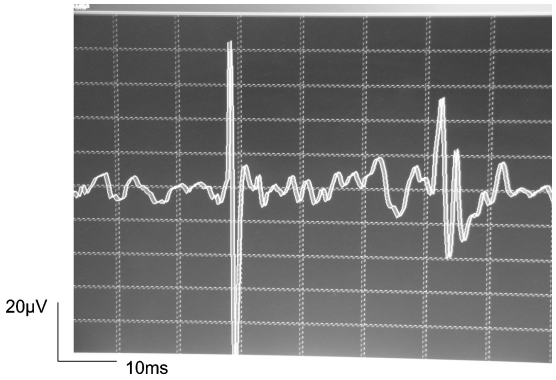
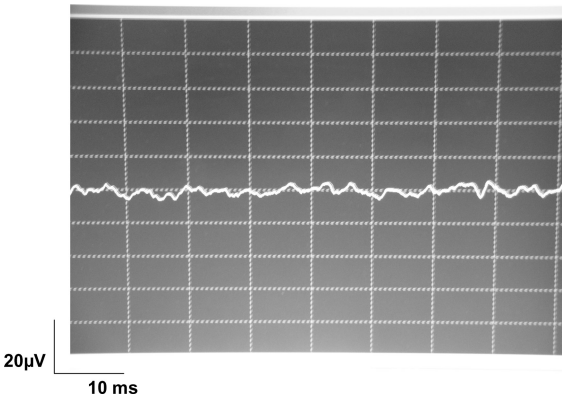


FIGURE 3. Zero qualified SEA posttreatment.



0 was given. Figure 2 shows SEA in a typical patient prior to treatment. Figure 3 demonstrates a reduction in SEA following treatment. Figure 4 presents zero-qualified SEA in a patient following treatment.

Procedures

Research Design

The design of the study was reviewed and approved by the Committee on the Protection of Human Subjects of the Rocky Mountain University of Health Professions. Random assignment to three treatment groups without a control group was mandated by ethical considerations. In some cases, a control group can be constructed by wait listing potential participants and providing them services upon completion of the research protocols. However, in the case of the emergent pain, a wait listing is unfeasible and unethical because of the potential harm to wait-listed patients who would not receive appropriate services in a timely fashion. Due to ethical considerations, the design of the study is weakened because no controls exist for history or maturation, both of which are threats to internal validity. Therefore, pretest/posttest differences may be due to the natural course of the disorder or to placebo effects.

Preliminary Activities

Prior to assessment, each research participant was required to fill out a demographic form and the PVAS. TrPs on the upper trapezius muscle were identified using the essential and confirmatory criteria as described by Simons and Travell (2). A doctoral-level trained physical therapist with more than 15 years of experience treating myofascial pain palpated the TrP. Once identified through palpation, the skin was marked using a fine skin marker with a circle of 2 mm diameter, as shown in Figure 5. A fanning needle approach was used to identify the locus of the greatest amount of SEA.

PPT and pressure tolerance were measured using the pressure algometer by pressing it directly on the TrP, as indicated by Hong and Simons (8). After the identification and marking of the TrP area, patients lay down in a prone position, exposing the areas of upper trapezius muscle to be tested. Supporting pillows were used to help the patients relax the upper

FIGURE 4. EMG setup including amplifier and reference and active electrodes

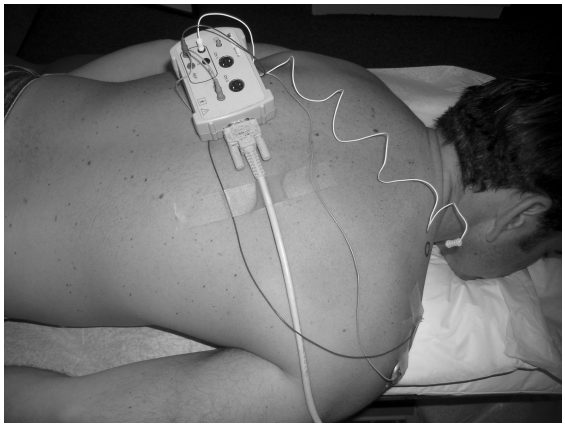
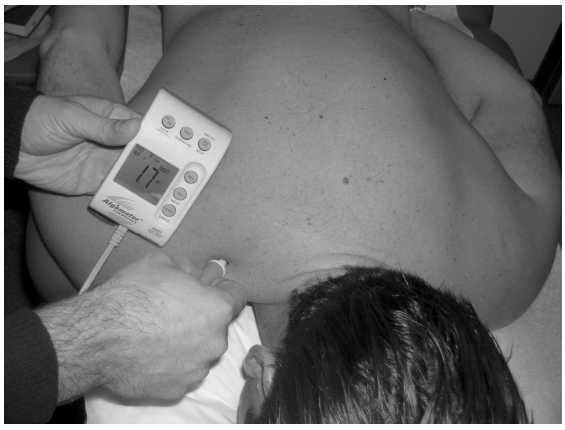


FIGURE 5. Position and application of pressure algometry.



trapezius muscle. Patients were instructed to immediately indicate the onset of pain with the verbal cue, "Now," indicating the PPT. The force was applied perpendicular to the skin's surface at a gradual rate of 2.2 pounds [1 kg] per second. The tip of the algometer was removed from the skin and the test was repeated three times. The average measurement for PPT was obtained. Immediately after, patients were instructed to indicate the maximum tolerable pain with the verbal cue, "Stop," indicating pressure tolerance. The tip of the algometer was removed from the skin and the test was repeated three times. The average measurement for pressure tolerance was obtained. Figure 6 demonstrates pressure on commentary on a patient.

Therapeutic Procedures

Two therapeutic procedures, IC and PS, were used, either individually or in combination. TrP compression was applied on the TrP by a physical therapist using the thumb and forefinger in a pinching motion for three applications of 60 seconds each, followed by a 30-second rest period. Practitioners have successfully used compression and stretching separately and in combination in the treatment of the tibialis posterior muscle (35–40). Simons et al. (2) defined IC as "TrP pressure release" and described as follows, "Application of slowly increasing, nonpainful pressure over a TrP until a barrier of tissue resistance is encountered. Contact is then maintained until the tissue barrier releases, and pressure is increased to reach a new barrier to eliminate the TrP tension and tenderness."

PS is specific to the muscle and requires a narrow therapeutic range 6. The targeted muscle is stretched until tension is sensed at the end of the ROM. The patient exhales allowing the muscle to relax, increasing the stretch. The newly gained position is held while the patient inhales. Further length is gained through succeeding exhalations, allowing the muscle to relax out rather than push through, moving at the rate of 3–4 mm/second for 45 seconds. This procedure was repeated three times with 30-second rest intervals in between. For the IC + PC group, the two techniques were alternated with 30-second rest intervals as follows: IC, rest, PS, rest, and so forth, for three repetitions. Participants in the IC and PS groups followed a similar regimen: Treatment [either IC or PS], rest, light massage, rest, for three repetitions.

Study participants attended six 15-minute therapeutic sessions over a two-week period. Participants attended sessions either on a Monday, Wednesday, Friday schedule or a Tuesday, Thursday, Saturday schedule. They were not engaged in any other interventions during this period. After the completion of the therapeutic protocol, research subjects were retested on the PVAS, SEA at the TrP, PPT, and pressure tolerance using pressure algometry. They were thanked for their participation and provided with instructions for home stretching exercises.

Analysis of Data

In order to meet the assumption of the analysis of variance model that dependent variables must be normally distributed, the distributions of the dependent variables of posttest scores on the PVAS, PPT, pressure tolerance, and SEA were tested for approximations to normality using the Kolmogorov–Smirnov test. A significant z-score on the Kolmogorov–Smirnov test indicates that the distribution violates the assumption of normal distribution. All z-scores were nonsignificant, indicating that none of the variables violated the assumption of approximation to normality.

In order to test the hypotheses, a repeated-measures multivariate analysis of variance [MANOVA] was computed with posttest scores as the dependent variables and treatment group

as the factor. The assumption of equality of variances [homoscedasticity] was met. A repeated-measures MANOVA was used because the hypotheses were formed around two major questions: (a) Did the treatments reduce pain [within-subjects measures over time]? (b) Did differences exist between treatment modalities [within-subjects differences by group as indicated by treatment by group interaction factors]? The MANOVA could answer both questions simultaneously.

RESULTS

Table 1 presents descriptive statistics on the demographic background variables of the 90 study participants. This number was deemed feasible within the context of the study and the capacity of Hands-On Physical Therapy. The patients were randomly distributed to have 30 in each group. Overall, females outnumbered males 60 percent to 40 percent–74.4 percent were between the ages of 30 and 59; 18.9 percent were 60 years or older. Educational background showed wide variation: 33.4 percent were college graduates, 44.4 percent were high school graduates, and 22.2 percent did not complete high school. Among them 67.7 percent were white, 16.7 percent were Latino, 8.9 percent were Asian, and 6.7 percent were African-American. The modal client was a Caucasian female high school graduate in her 40s.

Table 2 presents data on the type of pain experienced by study participants. Participants were encouraged to indicate as many types of pain that they had experienced in the head and neck region. The most common was the experience of dull pain, reported by 53.3 percent of the study participants, followed by headache [46.7 percent] and sharp pain [21.1 percent]. Unspecified other pain was experienced by 27.8 percent of the study participants.

Table 3 presents data on other characteristics of the pain experienced by study participants. Study participants split into approximately equal groups on the length of the time they had been experiencing pain, with 37.8 percent experiencing pain three or fewer months, 31.1 percent experiencing pain between four and six months, and the remaining 31.1 percent reporting pain six months or longer. A majority [53.3 percent] experienced pain seven or more times a day, 78.9 percent experienced pain at least four times a day, and 21.2 percent experiencing pain three or fewer times per day. The vast majority [81.1 percent] experienced pain for at least 30 minutes at a time, with nearly a majority indicating

TABLE 1. Frequencies and Distributions of Study Participants on Demographic Background Variables [N= 90]

Variable	N	%
Gender		
Male	36	40.0
Female	54	60.0
Age		
< 20	1	1.1
20–29	5	5.6
30–39	22	24.4
40–49	23	25.6
50–59	22	24.4
60–69	12	13.3
70 +	5	5.6
Educational attainment		
< Seventh-grade	4	4.4
Junior high school	9	10.0
Partial high school	7	7.8
High school graduate or GED	27	30.0
Some college	13	14.4
College graduate	19	21.1
Graduate degree	11	12.2
Racial/ethnic background		
White	61	67.8
Hispanic/Latino	15	16.7
Asian/Asian Pacific	8	8.9
African-American	6	6.7

GED= General Education Development degree.

TABLE 2. Type of Pain Experienced [N= 90]

Pain	No	Yes
Headache		
N	48	42
%	53.3	46.7
Dull pain		
N	42	48
%	46.7	53.3
Sharp pain		
N	71	19
%	78.9	21.1
Other pain		
N	65	25
%	72.2	27.8

TABLE 3. Frequencies and Distributions on Characteristics of Pain [*N*= 90]

Variable	*N*	%
Length of time of pain experience		
< 1 month	19	21.1
1–3 months	15	16.7
4–6 months	28	31.1
6–11 months	13	14.4
1–2 years	8	8.9
2 years+	7	7.8
Frequency of pain experience		
< Once a day	5	5.6
1–3 times a day	14	15.6
4–6 times a day	23	25.6
7 + times a day	48	53.3
Duration of pain experience		
< 5 min	2	2.2
6–15 min	4	4.4
16–30 min	11	12.2
30 min–1 hr	43	47.8
More than 1 hr, intermittently	29	32.2
Continuously	1	1.1
What caused pain?		
Tension/psychological stress	6	6.7
Repetitive stress	9	10.0
Trauma	12	13.3
Overstretching	21	23.3
Overshortening	21	23.3
Do not know	7	7.8
Other	14	15.6

that their pain lasted between 30 minutes and an hour.

The most common causes of the pain were either overstretching or overshortening, each indicated by 23.3 percent of the participants. Trauma was a distant third, endorsed by 13.3 percent of the participants. Repetitive stress was reported by 10.0 percent and tension was indicated by 6.7 percent. A small minority [7.8 percent] did not know the cause of their pain, and twice that number [15.6 percent] listed other causes for their pain.

The hypotheses of the study were tested using a MANOVA. Table 4 contains the descriptive statistics on the four pain indicator variables on the pretest and posttest assessments by treatment group. The results of the MANOVA are presented in Table 5.

On the basis of the effect size, it was established that three groups of 30 subjects each would result in a power coefficient greater than 0.80. The findings indicate that therapeutic intervention, regardless of whether it was IC, PS, or IC + PS, generated significant changes on all four pain indicators. In addition, on all four indicators, significant between-group differences were also found. For the total sample, the pretest mean on the PVAS was 7.07 [standard deviation [SD] = 1.60]. The posttest mean was 3.25 [SD = 1.83]. The resultant $F[1,87] = 410.99$ [$P < 0.05$], accounting for 83 percent of the variance in the equation. Between-group differences were tested using the treatment by group interaction factor. On the posttest PVAS, the IC group had a mean of 3.58 [SD = 1.78], the PS group had a mean of 3.72 [SD = 1.95], and the IC + PS group had a mean of 2.45 [SD = 1.50]. The resultant $F[2,87] = 7.99$ [$P < 0.05$], which accounted for an additional 16 percent of the variance. Post hoc pairwise comparisons indicated that the IC + PS combination resulted in a significantly greater decline in PVAS scores than IC alone [$P < 0.05$] and PS alone [$P < 0.01$]. The overall decline in PVAS scores was 54.0 percent; the decline in IC and PS scores were slightly less than 50 percent [47.4 percent and 47.5 percent, respectively]. The decline in IC + PS scores was nearly two thirds [66.6 percent].

Similar results were found for pain threshold. A significant increase from pretest [$M = 4.63$, SD = 1.66] to posttest [$M = 7.40$, SD = 1.76] was found for the total sample [$F[1, 87] = 190.03$, $P < 0.01$], which accounted for 69 percent of the total variance. The treatment group had a significant effect as well. The IC group had a posttest mean of 7.09 [SD = 1.65]; the PS group had a mean of 6.89 [SD = 1.63]. The IC + PS group had a posttest mean of 8.20 [SD = 1.77]; the resultant $F[2, 87] = 3.92$, $P < 0.05$. Treatment group differences accounted for 8 percent of the variance in the equation. Post hoc analyses indicated that the posttest mean of the IC + PS group was significantly higher than the other two groups [IC, $P < 0.05$; PS, $P < 0.01$]. Pain threshold increased 45.3 percent for the IC group, 58.0 percent for the PS group, and 75.6 percent for the IC + PS group. The overall increase in pain threshold was 59.8 percent.

Therapeutic interventions accounted for 69 percent of the variance in pretest to posttest differences on pressure tolerance [$F[1, 87] = 197.02$, $P < 0.01$]. For the total sample, pressure tolerance increased from 8.64 [SD = 1.60] on the pretest to 11.33 [SD = 1.79] on the posttest, representing an increase of 31.1

TABLE 4. Pretest and Posttest Descriptive Statistics for Pain Perception and SEA

Pain indicator	Group	*N*	*M*	SD	*M*	SD	% Change
PVAS [Scale 1–10]	IC	30	6.80	1.77	3.58	1.78	−47.4
	PS	30	7.08	1.54	3.72	1.95	−47.5
	IC + PS	30	7.33	1.48	2.45	1.50	−66.6
	Total	90	7.07	1.60	3.25	1.83	−54.0
Pressure–pain threshold [ppi^2]	IC	30	4.88	1.79	7.09	1.65	45.3
	PS	30	4.36	1.67	6.89	1.63	58.0
	IC + PS	30	4.67	1.52	8.20	1.77	75.6
	Total	90	4.63	1.66	7.40	1.76	59.8
Pressure tolerance [ppi^2]	IC	30	8.93	1.47	11.51	1.60	28.9
	PS	30	8.70	1.48	10.90	1.82	25.3
	IC + PS	30	8.27	1.80	11.58	1.93	40.0
	Total	90	8.64	1.60	11.33	1.79	31.1
SEA [μm]	IC	30	220.10	111.39	125.10	90.05	−43.2
	PS	30	212.80	130.06	137.40	97.33	−35.4
	IC + PS	30	222.63	102.32	77.57	76.37	−65.2
	Total	90	218.51	113.94	113.36	91.12	−48.1

PVAS= Pain Visual Analog Scale, PPT= pressure–pain threshold, SEA= spontaneous electrical activity, IC= ischemic compression, PS= passive stretching.

percent. Although differences between treatment techniques accounted for an additional 6 percent of the variance [$F[2, 87] = 2.89$, $P < 0.05$], post hoc analysis indicated no significant between-group differences.

For SEA, therapeutic interventions accounted for 64 percent of the variance from pretest to posttest [$F[1, 87] = 156.06$, $P < 0.01$]. For the total sample, SEA declined from the pretest mean of 218.51 [SD = 113.94] to a posttest mean of 113.36 [SD = 91.12]. Between-group differences accounted for an additional 12 percent of the variance in SEA. The IC group had a posttest mean of 125.10 [SD = 90.05]; the PS group had a mean of 137.40 [SD = 97.33]. The IC + PS group had a posttest mean of 77.57 [SD = 76.37]; the resultant $F[2, 87] = 6.07$, $P < 0.01$. Post hoc analyses indicated that the posttest mean of the IC + PS group was significantly lower than the other two groups [$Ps < 0.01$]. The decline in SEA for the IC group was 43.2 percent, for the PS group, 35.4 percent, and for the IC + PS group, 65.2 percent. Overall, SEA was reduced by 48.1 percent.

Hypothesis 1, which stated that IC would significantly reduce SEA and pain perception, was

TABLE 5. Summary of Repeated-Measures MANOVA on Posttest Pain Indicators

Pain indicator	Source	SS	df	MS	F
PVAS	Treatment	657.04	1	657.04	410.99**
	Treatment X group	25.55	2	12.77	7.99**
	Error	139.09	87	1.60	
PPT	Treatment	343.07	1	343.07	190.03**
	Treatment X group	14.17	2	7.08	3.92**
	Error	157.07	87	1.81	
Pressure tolerance	Treatment	326.16	1	326.16	197.02**
	Treatment X group	9.56	2	4.78	2.89*
	Error	144.02	87	1.66	
SEA	Treatment	497,596.09	1	497596.09	156.06**
	Treatment X group	38,721.38	2	19360.69	6.07**
	Error	277,401.53	87	3188.52	

*$P < 0.05$, **$P < 0.01$.
PVAS= Pain Visual Analog Scale, PPT= pressure–pain threshold, SEA= spontaneous electrical activity.

supported strongly by the data. Hypothesis 2, which stated that PS would significantly reduce SEA and pain perception, was also supported strongly by the data. With the singular exception of lack of pairwise differences in pressure tolerance, Hypothesis 3 was supported by the data. All three intervention techniques significantly reduced SEA and pain perception. With the exception of pressure tolerance, the combination of IC + PS reduced pain perception and SEA more than did IC or PS in isolation.

DISCUSSION

The findings of the study confirm the outcomes of prior research demonstrating the positive effects of IC and PS. Several researchers have found that dry needling and injections of substances such as lidocaine or botulinum toxin into the TrP significantly reduces pain (2, 13, 26, 41–47). However, the major problem with such interventions is their invasiveness. Given that shortcoming, researchers have tried numerous other modalities including IC (14, 15), PS (10–14, 16), nerve stimulation (45), anesthetic sprays (15), exercise (11, 12, 48, 49), and laser therapy (46), with most techniques demonstrating reductions in pain.

Hanten et al. (14) studied the effects of IC and PS on neck and shoulder pain and found that the combination reduced the intensity of subjective pain but did not reduce the duration of pain. Ingber (3) used dry needling, IC, stretching, and an exercise regimen with three racquetball players who had shoulder pain, which resulted in a decline of pain. All three subjects were interviewed a year after the regimen and indicated that they were pain-free. The major problem with these studies is that in each case IC was used in combination with other treatment modalities, confounding any effects of IC with them. Therefore, prior research has not indicated whether TrP compression alone is effective in reducing pain. The findings of this study indicate that TrP compression significantly reduces pain; however, in combination with PS, pain is further reduced.

As noted above, numerous researchers have incorporated PS into therapeutic regimens. Hanten et al. (14) combined PS with IC without isolating the two so that the independent contributions of each treatment modality could not be assessed. Similarly, Ingber's study (3) of racquetball players combined PS with several other techniques. In addition, data were anecdotal and sample sizes were small.

Although Taylor et al. (16) used PS as a single modality, their experiments were on rabbits and the dependent variable was muscle flexibility. Lewit and Simons (50) studied the effects of isometric relaxation and stretching exercises in 244 patients, who reported immediate pain relief in nearly all cases and lasting relief in approximately one quarter of the cases. Although the data were promising, reports were anecdotal and no control group was involved. Patla and Abbott (4) also used anecdotal data in case studies in which stretching was used to reduce pain in the tibialis posterior muscle of two patients. Neither patient evidenced pain in a two-month follow-up. Bandy et al. (10) conducted a study to examine the optimal length of stretching in order to diminish pain.

The findings of the study raise the question as to why TrP compression and PS reduced pain and why together they produced significant declines in pain. According to the theory of TrPs, pain at the site and referred pain are a consequence of trauma caused by overstretching, overshortening, or overloading muscles, which damages the sarcolemma and sarcoplasmic reticulum generating the release of excess Ca^{2+} into the muscle tissue, causing a sustained muscle contraction (2, 5, 15). The presence of SEA in the vicinity of a TrP is the result of excessive quanta of ACh released by dysfunctional endplates present in the synaptic cleft during rest causing partial depolarization of the postsynaptic membrane (2, 5). A decrease in length of the affected muscle fibers along with muscle spasm promotes vasoconstriction and induction of a hypoxic state at the affected area of the muscle. The individual experiencing the pain may attempt to compensate for it by restricting motion, generating further muscle shortening.

TrP compression and PS as well as their combination cause an increase in blood circulation helping the muscle to achieve an energetically adequate metabolic state. Restoration of aerobic metabolism increases adenosine triphosphate supply and enhances myofilament interaction in the previously myofascially active loci. This process of restoration of proper muscle cell metabolism and function may be responsible for the decrease of excess ACh in the synaptic cleft and postsynaptic EMG silence.

The findings of this study suggest that IC and PS have different functions in the treatment of myofascial pain. Otherwise, there would have been no difference between treatments in isolation and in combination. The theory of IC is that palpation at the TrP provides a gentle stretch to the muscle (2, 5, 15). The purpose of TrP compression is to inactivate the TrPs by lengthening the overshortened muscle fibers through a progressive pressure technique that is applied until resistance is felt. Steady force is applied against the tissue until resistance dissipates. The procedure is repeated until the contracted sarcomeres release.

TrP compression results in temporary further local ischemia during the application of the pressure, followed by reactive hyperemia when the pressure is released (2). This additional blood supply relieves the affected muscle locus from the hypoxic state and provides new resources of energy supply [aerobic metabolism for adenosine triphosphate formation] for the local tissue metabolic demands (2, 5). Pain relief from TrP compression may also result from counterirritant effects or a spinal reflex mechanism for the relief of muscle spasm (51).

Passive or myofascial stretching is also directed at lengthening the overshortened muscle fibers, but uses a different technique. It involves slow stretching because in a fast stretch, only healthy fibers will be extended (2, 5). Slow stretching with proper concentration, relaxation, and breathing will inhibit the gamma spindle response that causes the muscle to shorten when rapidly stretched. PS involves stretching the muscle to the end of the ROM and holding it there until the muscle relaxes. Whereas TrP compression works directly on the TrP, PS involves the whole muscle in a way that allows for the lengthening of the contracted sarcomeres. This research team hypothesizes that the two treatments in tandem provide superior results because of their complementary approaches to the problem of TrPs. TrP compression focuses directly on the TrP; PS treats the TrP in relation to the rest of the muscle. Because of that, the two treatment modalities seem to be complementary, reinforcing each other. In this study, the combination of the two treatment modalities greatly influenced the effectiveness of the therapeutic intervention; perceived pain dropped by two thirds compared to less than 50 percent for the two modalities in isolation; pain threshold increased by three fourths compared to about 50 percent of the other two modalities; SEA dropped by 65 percent compared to about 40 percent of the two modalities in isolation.

In addition, the use of EMG and the quantification of SEA as an adjunct diagnostic tool indicating activity of a TrP provided a physiological correlation to pain perception, providing stronger validity to the outcomes of this study. By achieving similar results using subjective [pain perception] and objective [SEA] measures, the indicators triangulate and cross-validate each other. In addition, levels of SEA could indicate whether a TrP is still active or has been deactivated with appropriate intervention.

As noted above, because of ethical considerations, the study was performed without a control group. Therefore, maturational and placebo effects could not be controlled. This constitutes a limitation on the generalizability of the findings. However, of concern is the extent to which such design limitations affect generalizability. Limitations of design can be mitigated by theory. That is, if findings conform to theoretical expectations, the possibility of maturation or placebo effects as an explanation of differences declines. In this study, the findings conform to theoretical expectations. However, the issue of maturation cannot be avoided. How do we know that the decline in pain perception and SEA from pretest to posttest was not an artifact of a natural healing process?

In addition, dry needling has been used as a therapeutic technique in the reduction of pain and SEA (13, 26, 46). Therefore, it is possible that the insertion of the needle at the TrP may have influenced the healing process. Despite the problems of the research design, several factors suggest that the findings represent actual changes in pain perception and SEA resulting from therapeutic interventions. First, pretest/posttest differences are very strong. Second, the findings conform to theoretical expectations. Third, the two therapeutic interventions manifested a multiplier effect, suggesting changes beyond those induced by maturation or needle insertion. It is important to reiterate that all participants experienced the same amount of therapeutic intervention regardless of the group to which they belonged.

Although these findings are robust and highly encouraging, they are limited to the treatment of TrPs in the upper trapezius muscle. On the basis

of the findings of the study, physical therapists, after verifying the existence of TrPs in the upper trapezius muscle, should provide a regimen of therapy that involves the combination of IC and PS.

This is the first study that has examined the effectiveness of TrP compression and PS in isolation and in combination on TrPs in the upper trapezius muscle. The major question facing researchers and the profession as a consequence of these findings is whether they are in evidence when examining therapeutic treatments of TrPs in other parts of the body. Therefore, future researchers might wish to replicate this study in other regions of the body where PS can be used. In addition, researchers need to explore the most efficacious combination of treatment modalities.

ACKNOWLEDGEMENTS

This study was sponsored by St. Matthew's University School of Medicine [http:// www.stmatthews.edu/].

REFERENCES

1. Wrongdiagnosis.com: Prevalence and incidence of headache. Retrieved January 9, 2004, from http://www.wrongdiagnosis.com/h/headache/prevalence.htm.

2. Simons DG, Travell JG, Simons LS: Myofascial Pain and Dysfunction: The Trigger Point Manual, Vol 1. Upper Half of Body. 2nd ed. Williams & Wilkins, Baltimore, 1999.

3. Ingber R: Shoulder in management in tennis/racquetball players treated with subcapularis myofascial treatments. Arc Phys Med Rehabil 81: 679–682, 2000.

4. Patla CE, Abbott JH: Tibialis posterior myofascial tightness as a source of heel pain: Diagnosis and treatment. J Orthop Sports Phys Ther 30(10): 624–632, 2000.

5. Kostopoulos D, Rizopoulos K: The Manual of Trigger Point and Myofascial Therapy. Slack, Inc., Thorofare, NJ, 2001.

6. Ingber R: Myofascial pain in lumbar dysfunction. Phys Med Rehabil State Art Rev 13(3): 473–493, 1999.

7. Rivner MH: The neurophysiology of myofascial pain syndrome. Curr Pain Headache Rep 5: 432–440, 2001.

8. Hong CZ, Simons DG: Pathophysiologic and electrophysiologic mechanisms of myofacial trigger points. Arc Phys Med Rehabil 79: 863–872, 1998.

9. Hubbard DR, Berkoff GM: Myofascial trigger points show spontaneous needle EMG activity. Spine 18: 1803–1807, 1993.

10. Bandy WD, Irion JM, Briggler M: The effect of time and frequency of static stretching on flexibility of the hamstring muscles. Phys Ther 77(10): 1090–1096, 1997.

11. Barnes WJ: Efficiency and costs of medical exercise therapy, conventional physiotherapy, and self-exercise in patients with chronic low back pain: A pragmatic, randomized, single-blinded, controlled trial with 1-year follow-up. Phys Ther 79(6): 609, 1999.

12. Blackburn SE, Portney LG: Electromyographic activity of back musculature during Williams' flexion exercises. Phys Ther 61(6): 878–885, 1981.

13. Edwards J, Knowles N: Superficial dry needling and active stretching in the treatment of myofascial pain—A randomised controlled trial. Acupunc Med 21(3): 80–86, 2003.

14. Hanten WP, Olson SL, Butts NL, Nowicki AL: Effectiveness of home program of ischemic pressure followed by sustained stretch for treatment of mild fascial trigger points. Phys Ther 80(10): 997–1003, 2000.

15. Hou CR, Tsai LC, Cheng KF, Chung KC, Hong CZ: Immediate effects of various physical therapeutic modalities on cervical myofascial pain and trigger-point sensitivity. Arch Phys Med Rehabil 83: 1406–1414, 2002.

16. Taylor DC, Dalton JD, Jr., Seaber AV, Garrett WJ: Viscoelastic properties of muscle-tendon units: The biomechanical effects of stretching. Am J Sports Med 5(3): 300–309, 1990.

17. Fischer AA: Pressure algometry over normal muscles. Standard values, validity, and reproducibility of pressure threshold. Pain 30: 115–126, 1987.

18. Antonaci F, Sand T, Lucas GA: Pressure or algometry in healthy subjects: Inter-examiner variability. Scand J Rehabil Med 30: 3–8, 1998.

19. Farella M, Michelotti A, Steenks MH, Romeo R, Cimino R: The diagnostic value of pressure algometry in myofascial pain of the jaw muscles. J Oral Rehab 27: 9–14, 2000.

20. Sciotti VM, Mittak VL, DiMarco L, et al.: Clinical precision of myofascial trigger point location in the trapezius muscle. Pain 93: 2001, 259–266.

21. Turchin DC, Beaton DE, Richards RR: Validity of observer-based aggregate scoring systems as descriptors of elbow pain, function, and disability. J Bone Joint Surg 82(2): 154–162, 1998.

22. Paice JA, Cohen FL: Validity of a verbally administered numeric rating scale to measure cancer pain intensity. Cancer Nurs 20(2): 88–93, 1997.

23. Gragg RA, Rapoff MA, Danovsky MB, et al.: Assessing chronic musculoskeletal pain associated with rheumatic disease: Further validation of the Pediatric Pain Questionnaire. J Pediatr Psychol 21(2): 237–250, 1996.

24. Flandry F, Hunt JP, Terry GC, Hughston JC: Analysis of subjective knee complaints using visual analog scales. Am J Sports Med 19(2): 112–118, 1991.

25. Noyes FR, McGinniss GH: Controversy about treatment of the knee with anterior cruciate laxity. Clin Orthop 198: 61–76, 1985.

26. Chen JT, Chung KC, Hou CR, Kuan CR, Chen SM, Hong CZ: Inhibitory effect of dry needling on the spontaneous electrical activity recorded from myofascial trigger spots of rabbit skeletal muscle. Am J Phys Med Rehabil 80(10): 729–736, 2001.

27. Weeks VD, Travell JG: How to give painless injections. AMA Scientific Exhibits 1957. Grune & Stratton, New York, 1957, pp. 318–322.

28. Simons DG, Hong CZ, Simons LS: Prevalence of spontaneous electrical activity at trigger spots and at control sites in rabbit skeletal muscle. J Musculoske Pain 3(Supp 1): 35–48, 1995.

29. Wiederholt WC: "End-plate noise" in electromyography. Neurology 20: 214–224, 1997.

30. Liley AW: An investigation of spontaneous activity at the neuromuscular junction of the rat. J Physiol 132: 650–666, 1956.

31. Heuser J, Miledi R: Effect of lanthanum ions on function and structure of frog neuromuscular junctions. Proc R Soc Lond (Biol) 179: 247–260, 1971.

32. Ertekin C, Arac N, Uluda B: Enhancement of "end-plate monophasic waves" during an attack of hypokalemic periodic paralysis. Muscle Nerve 19(5): 680–681, 1996.

33. Hong CZ: Myofascial trigger point injection. Crit Rev Phys Med Rehabil 5: 203–217, 1993.

34. Mense S, Simons DG, Russell IJ: Muscle Pain: Understanding its Nature, Diagnosis, and Treatment. Lippincott Williams & Wilkins, Philadelphia, 2001.

35. Ballas MT, Tytko J, Cookson D: Common overuse running injuries: Diagnosis and management. Am Fam Physician 55(7): 2473–2484, 1997.

36. Englund J: Chronic compartment syndrome: tips on recognizing and treating: The patient's history offers more important clues than physical exam. J Fam Pract 54(11): 955–961, 2005.

37. Kostopoulos D, Rizopoulos K: Shin splint pain: The runner's nemesis. ADVANCE for Physical Therapists & PT Assistants 33–34, 1999.

38. Patla CE: Tibialis posterior myofascial tightness as a source of heel pain: Diagnosis and treatment. J Orthop Sports Phys Ther 30(10): 624–632, 2000.

39. Newman J: Radiologic technology's role in sports medicine. Radiol Technol 69(6): 535–554, 1998.

40. Veenema KR: Ankle sprain: Primary care evaluation and rehabilitation. J Musculoske Med 17(9): 563–572, 2000.

41. Chen JT, Chen SM, Kuan TS, Chung KC, Hong CZ: Phentolamine effect on the spontaneous electrical activity of active loci in the myofascial trigger spot of rabbit skeletal muscle. Arch Phys Med Rehabil 79: 790–794, 1998.

42. Couppe C, Jorgensen U, Midttun A, Oxholm P, HIlden J, Fuglsang-Frederiksen A: Spontaneous needle electromyelographic activity in myofascial trigger points in the infraspinatus muscle: A blinded assessment. J Musculoske Pain 9(3): 7–16, 2001.

43. Greenwald R: Clinicians' pearls: Trigger point injections. Am J Pain Manag 10: 79–81, 2000.

44. Hou CR, Chung KC, Chen JT, Hong CZ: Effects of a calcium channel blocker on electrical activity in myofascial trigger spots of rabbits. Am J Phys Med Rehabil 81(5): 342–349, 2002.

45. Hsueh TC, Cheng PT, Kuan TS, Hong CZ: The immediate effectiveness of electrical nerve stimulation and electrical muscle stimulation on myofascial trigger points. Am J Phys Med Rehabil 76(6): 471–476, 1997.

46. Ilbuldu E, Cakmak A, Disci R, Aydin R: Comparison of laser, dry needling, and placebo laser treatments in myofascial pain syndrome. Photomed Laser Surg 22(4): 306–311, 2004.

47. Kuan TS, Chen JT, Chen SM, Chien CH, Hong CZ: Effect of transaction of innervating nerve on spontaneous electrical activity of myofascial trigger spot in rabbit skeletal muscle. J Rehabil Med Assoc Repub China 29(2): 65–75, 2001.

48. Decker MJ, Hintermeister RA, Faber KJ, Hawkins RJ: Serratus anterior muscle activity during selected rehabilitation exercises. Am J Sports Med 27: 784–791, 1999.

49. Hintermeister RA, Lange GW, Schultheis JM, Bey MJ, Hawkins RJ: Electromyographic activity in applied load during shoulder rehabilitation exercises using elastic resistance. Am J Sports Med 26: 210–220, 1998.

50. Lewit K, Simons DG: Myofascial pain: Relief by post-isometric relaxation. Arch Phys Med Rehabil 65(8): 452–456, 1984.

51. Wall PW, Melzack R: Textbook of Pain. Churchill Livingston, New York, 1984.

Submitted: October 19, 2005
Revisions Accepted: August 8, 2006