Effects of Microcurrent Treatment on Perceived Pain and Muscle Strength Following Eccentric Exercise

Jeffrey A. Bonacci, MS, ATC; Elizabeth J. Higbie, PhD, PT, ATC

Objective: The purpose of this study was to assess the effect of low-volt, microamperage stimulation (LVMAS) on perceived pain and muscle strength following an intense bout of eccentric exercise.

Design and Setting: An experimental pretest-posttest control group design was used for the study. The experiment was conducted in the Lower Extremity Research Laboratory at Georgia State University.

Subjects: Twelve females and six males (mean age 27 ± 5 yr).

Measurements: Subjects, randomly assigned to experimental (EXP, $n = 6$), sham (SHAM, $n = 6$), and control (CON, $n = 6$) groups, were tested before, and at 24, 48, and 72 hours following, an intense bout of eccentric exercise.

Results: Three two-way (group × time) analyses of variance (ANOVAs) with repeated measures on the last factor were used to analyze the data. A significant time main effect was identified. Results indicated that perceived pain was not reduced in the EXP group as compared with the SHAM and CON groups. Muscle strength in the EXP group did not return to the initial baseline measure more rapidly than in the SHAM and CON groups.

Conclusions: We conclude that the use of LVMAS alone is not effective in reducing pain and increasing muscle function following an exhaustive bout of eccentric exercise.

Key Words: electrical stimulation, delayed-onset muscle soreness, musculoskeletal injury

For the past decade, athletic trainers and physical therapists have provided anecdotal praise for the use of low-volt, microamperage stimulation (LVMAS). LVMAS is used clinically to decrease pain precipitated by damaged muscles, tendons, and ligaments. Studies documenting successful pain reduction using LVMAS following musculoskeletal injury have been limited to a few clinical and experimental trials,[1–5] several nonexperimental trials[6] (L. Wallace, 1995, personal communication), and anecdotal recommendations (L. Wallace, 1995, personal communication; J. Halbach, 1995, personal communication). Collectively, these studies[3–5] have utilized a similar protocol: (1) polarity, biphasic mode; (2) intensity, 100 μamps; (3) frequency, 0.3 Hz; and (4) treatment time, 20 minutes (as described by Picker[7]). Results from several case studies have indicated that a long-term treatment protocol (> 8 hours) consisting of LVMAS, ice, and nonsteroidal anti-inflammatory drugs (NSAIDs) was effective in reducing pain and muscle soreness from musculoskeletal injuries[6,8] (J. Foley, 1995, personal communication). However, the effectiveness of LVMAS alone for treatment of perceived pain and muscle function has not been established.

Inducing delayed-onset muscle soreness (DOMS) in healthy subjects has been an acceptable approach for evaluating the treatment effects of therapeutic modalities such as LVMAS.[2] DOMS is commonly associated with an intense bout of unaccustomed exercise that involves eccentric actions and is characterized by pain, spasm, and weakness (ie, loss of force production) in the affected muscle(s).[9] The onset of pain resulting from DOMS is usually experienced 8 to 10 hours following intense eccentric exercise and peaks between 24 and 48 hours.[2,10,11] Since the symptoms associated with DOMS are similar to those experienced following musculoskeletal damage, we believe DOMS serves as an appropriate model for investigating the effects of LVMAS on musculoskeletal injuries.[2,8] Therefore, the purpose of this study was to assess the effects of a combined brief (20 minutes) and long-term (> 8 hours) application of LVMAS on perceived pain and eccentric isotonic muscle strength following muscle failure during eccentric exercise. We hypothesized that, following an exhaustive bout of eccentric exercise, the group receiving the LVMAS treatment would have significantly less perceived pain and a more rapid return of muscle strength than the groups not receiving LVMAS treatment.

METHODS

Subjects

Twelve female and six male right-hand-dominant subjects, 21 to 41 years of age (mean age 27 ± 5 years) volunteered to participate in this study. Subjects who had been involved in an upper-extremity weight-training program within the past year, used their arms regularly in strenuous activity, or presently had pain in their nondominant arm were excluded from the study. All subjects provided informed consent prior to testing in compliance with Georgia State University's Institutional Review Board.

Subjects were randomly assigned to one of the following three groups: (1) an experimental (EXP) group that received an LVMAS treatment following exercise ($n = 6$); (2) a sham

Jeffrey A. Bonacci is a doctoral student in the Department of Health, Physical Education, Recreation, and Safety at Middle Tennessee State University, Murfreesboro, TN 37132. **Elizabeth J. Higbie** is an assistant professor and director of graduate education in the Department of Physical Therapy at Georgia State University, Atlanta, GA 30303-3083.

(SHAM) group that received a sham treatment of LVMAS following exercise (n = 6); or (3) a control (CON) group that received no treatment following exercise (n = 6). Subjects were asked to refrain from taking any anti-inflammatory or pain-reducing medication and from applying heat, ice, massage, or stretching to the nondominant arm until the study was completed. Subjects were instructed to refrain from strenuous use of the nondominant arm. All subjects reported complying with the instructions each day.

Data Collection Protocol

Data collection for each subject in the EXP and SHAM groups consisted of the following: an orientation session, a baseline muscular strength measurement and a strenuous bout of eccentric exercise, four test sessions of muscle strength measures, and eight pain assessments using a modified graphic pain-rating scale[2,8] (Fig 1). During the orientation session, the subjects practiced applying the M.E.N.S. 2000S+ (Monad Corporation, Pomona, CA) electrodes to the skin over the nondominant biceps brachii muscle and adjusting the parameters on the unit. The baseline muscular strength session occurred during the orientation session. Data collection for each subject in the CON group consisted of the same test protocol as the one for the EXP and SHAM groups, except pain was measured at the pretest and at 24, 48, 72, and 96 (± 5) hours from the initiation of the muscle damage protocol for a total of five times. Each day after the exercise bout, pain was assessed using a graphic pain-rating scale before and after treatment for the EXP and SHAM groups. For the CON group, pain was assessed each morning for four consecutive days. An initial pain assessment was obtained to ensure that there was no pain in the nondominant arm prior to initiating DOMS for all three groups. Following the pain measurement, muscle strength was assessed with a one-repetition maximum (1RM) eccentric isotonic test using the nondominant arm of subjects in all three groups. Subjects were asked to perform a 1RM eccentric isotonic test using the nondominant arm to establish a baseline starting resistance. Following the baseline muscle strength assessment, the exercise bout was initiated. After measuring muscle strength, the EXP and SHAM groups received a 20-minute LVMAS treatment or sham LVMAS treatment, respectively.

Test Procedures

To determine initial muscle strength of the nondominant arm, each subject performed an eccentric isotonic 1RM of the elbow flexor group. Subjects were seated beside an exercise machine (Fig 2) designed to assess eccentric muscle strength of the elbow flexors. Subjects were instructed to sit up straight, place both feet flat on the floor, look straight ahead facing a full-length mirror, and keep the shoulders level. To ensure

Fig 1. Graphic pain-rating scale.

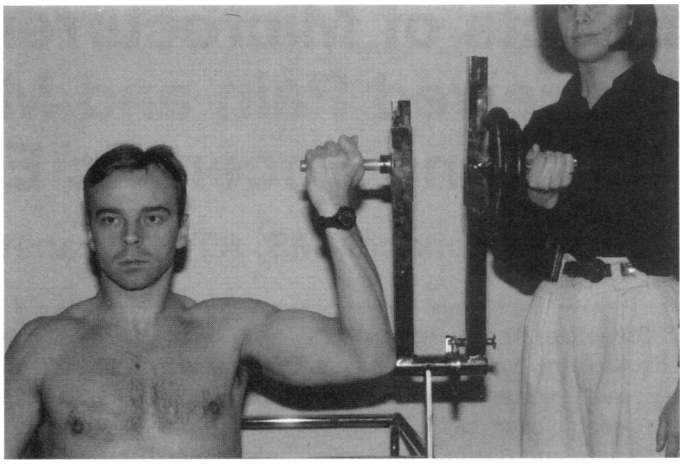

Fig 2. Subject seated at the designed exercise machine.

subject compliance with the exercise, another investigator, positioned behind the subject, manually stabilized the subject's shoulder and scapulae (Fig 3). The subject's lateral epicondyle was aligned with the axis of the exercise machine. The position of the lateral epicondyle was visually aligned and maintained by the investigator during the muscle damage protocol. DOMS was then induced in each subject's nondominant elbow flexor muscle group. All concentric lifting was performed by the investigator while another investigator stabilized the shoulder and the scapulae. Subjects lowered 90% of the 1RM until the weight could not be controlled for three seconds, at which time the resistance was decreased by 2.25 kg. The process continued in 2.25-kg decrements until the subject could not control 2.25 kg or had completed 10 repetitions with 2.25 kg. Perceived pain was assessed using a graphic pain-rating scale based on a verbal descriptive scale modified by Denegar et al.[8]

Treatment Procedures

The LVMAS was administered using a double-blind clinical protocol. Immediately following the assessment of the 1RM each day, subjects in the EXP and SHAM groups were given the LVMAS treatment through two oval (177-mm diameter) self-adhesive carbon-rubber electrodes. The electrodes were

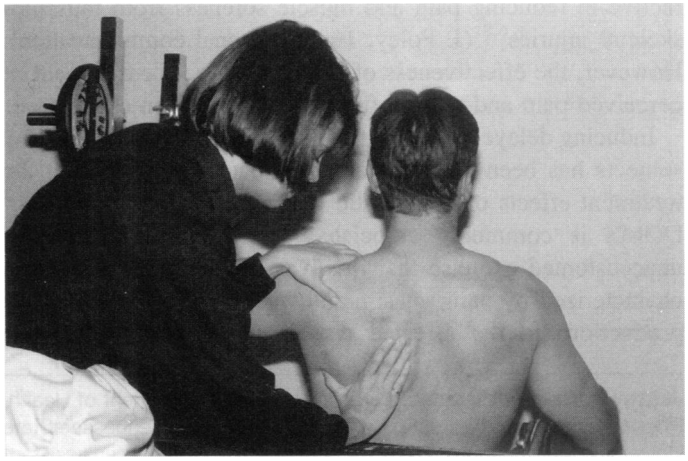

Fig 3. Stabilization of the subject's shoulder and scapulae by investigator.

placed on the skin over the medial and lateral aspects of the biceps brachii muscle at the initial point of pain and were held in place with athletic prewrap and 3.8-cm (1.5-inch) white adhesive athletic tape. Electrical stimulation was produced with a M.E.N.S. 2000S+ Microcurrent Stimulator (Monad Corporation). All six stimulation units used were designed to provide auditory and visual feedback to the subject. However, the electrical mechanism that delivered the stimulation was detached for three of the six units prior to initiation of the study. One investigator was responsible for identifying and distributing the units but did not administer any treatment. A different investigator, without knowledge of the functional status of the units, was responsible for applying treatment to the subjects.

With a permanent black marker, circles (25-mm diameter) were drawn on the skin over the biceps brachii muscle on each subject at the reported initial point of pain in order that pad placement would remain consistent throughout the study. Subjects were instructed not to remove the black circle on the skin over the biceps brachii muscle. The black circle was assessed daily for clarity and blackened as needed. The parameters on the stimulation unit were set at 100 μamps and 0.3 Hz with a biphasic polarity. Treatment time during the day was 20 minutes.

Subjects in the EXP and SHAM groups were issued the same M.E.N.S. 2000S+ unit each day for short- and long-term treatment. Subjects were instructed to treat the damaged arm for at least 8 continuous hours overnight using the same protocol that was used during the 20-minute treatment during the day. None of the subjects reported any malfunction in the application of the unit during the 8-hour treatment. Each subject was educated and retested verbally each day on the operation of the M.E.N.S. 2000S+ unit to assure proper knowledge of the operation of the unit.

Statistical Analysis

Muscle strength measures were analyzed with a 3 × 5 (group × time) ANOVA with repeated measures on one factor (time). Perceived pain measures were analyzed with a 3 × 5 (group × time) ANOVA with repeated measures on one factor (time) to compare measures from each morning group. A 2 × 8 (group × time) ANOVA with repeated measures on one factor (time) was used to assess changes in perceived pain between the EXP and SHAM groups. This analysis included a pain score following each daily microcurrent treatment session.[12]

A post hoc power analysis was conducted to interpret nonsignificant group × time interactions reported in all three two-way analyses of variance (ANOVAs). Power calculated for each group × time interaction for each of the three two-way ANOVAs used for data analysis was as follows: (1) 0.86 for the 3 × 5 (group × time) ANOVA for muscle strength data; (2) 0.73 for the 3 × 5 (group × time) ANOVA for perceived pain; and (3) 0.71 for the 2 × 8 (group × time) ANOVA for perceived pain. The power values indicate that the possibility is relatively small that a significant group × time interaction would be found with a larger sample size or higher alpha level.[12]

RESULTS

Changes in strength measures are presented in Table 1. No significant effect over time among the three groups was found for strength measures, indicated by a nonsignificant two-way interaction (group × time) ($F(8,75) = 0.07$, $p = .9997$). A significant main effect for time was found using an analysis for a main comparison ($F(4,75) = 19.72$, $p = .0483$). Strength measures determined daily using an eccentric 1RM test were significantly decreased following 24 ($F(2,75) = 14.24$, $p = .034$) and 48 ($F(2,75) = 10.33$, $p = .0468$) hours for all three groups. Each of the three groups returned to baseline strength measures within 72 hours following the initiation of DOMS.[12]

Changes in pain measures for the three groups are presented in Table 2. A nonsignificant two-way interaction (group × time) was found for pain measures, indicating no significant effect over time among the three groups when pain was measured each morning ($F(8,75) = 0.30$, $p = .9636$). A significant main effect for time was found using an analysis for a main comparison ($F(4,75) = 19.81$, $p < .0001$). Pairwise comparisons for the CON group indicated that the initial mean pain score was significantly lower than the pain scores measures at 24 ($F(1,75) = 13.79$, $p = .0004$), 48 ($F(1,75) = 22$, $p < .0001$), 72 ($F(1,75) = 12.07$, $p = .0009$), and 96 ($F(1,75) = 5.54$, $p = .0212$) hours. In addition, the mean pain score at 48 hours was significantly greater than the mean pain score at 96 hours ($F(1,75) = 5.46$, $p = .0221$). Pairwise comparisons for the EXP group indicated that the initial mean pain score was significantly lower than the pain scores measures at 24 ($F(1,75) = 25.43$, $p < .0001$), 48 ($F(1,75) = 17.19$, $p < .0001$), 72 ($F(1,75) = 9.15$, $p = .0034$) and 96 ($F(1,75) = 4.40$, $p = .0394$) hours. In addition, the mean pain score at 48 hours was significantly greater than the mean pain score at 72 ($F(1,75) = 4.07$, $p = .0473$) and 96 ($F(1,75) = 28.21$, $p = .0043$) hours. Pairwise comparisons for the SHAM group indicated that the initial mean pain score was significantly lower than the pain scores measures at 24 ($F(1,75) = 17.19$, $p < .0001$), 48 ($F(1,75) = 19.52$, $p < .0001$), 72 ($F(1,75) = 9.06$, $p = .0036$), and 96 ($F(1,75) = 4.60$, $p = .0352$) hours. In addition, the mean pain scores at 24 ($F(1,75) = 4.00$, $p = .0490$) and 48 ($F(1,75) = 5.17$, $p = .0259$) hours were significantly greater than the mean pain score at 96 hours.[12]

Changes in pain measures between the EXP and SHAM groups determined using a 2 × 8 (group × time) ANOVA with

Table 1. Muscle Strength Measures (kg) (Mean ± SD)

Time (Day)	Experimental Group ($n = 6$)	Sham Group ($n = 6$)	Control Group ($n = 6$)
0*	30.1 ± 18.6	33.6 ± 14.8	34.7 ± 18.2
1	29.6 ± 17.2†	28.6 ± 12.4†	26.3 ± 15.0†
2	26.3 ± 17.9†	28.7 ± 9.9†	26.8 ± 16.4†
3	29.0 ± 15.6	32.5 ± 11.1	30.0 ± 17.4
4	30.7 ± 16.1	36.1 ± 13.3	35.2 ± 18.1

* Baseline 1RM.
† Significantly different from the baseline 1RM ($p \leq .05$).

Table 2. Mean (±SD) of Pain Measures (cm)

Time (h)	Experimental Group (n = 6)	Sham Group (n = 6)	Control Group (n = 6)
0*	0.0†	0.0†	0.0†
24	4.4 ± 2.8	3.4 ± 1.9	3.0 ± 2.1‡
29	3.4 ± 3.1	3.3 ± 1.7	
48	3.4 ± 2.5‡	3.7 ± 1.3‡, §	4.0 ± 1.7‡
52	3.2 ± 3.1	3.0 ± 0.4	
72	2.3 ± 2.7	2.2 ± 1.5	2.7 ± 1.7
77	1.6 ± 2.2	1.9 ± 1.6	
96	1.3 ± 1.6	1.4 ± 1.6	1.3 ± 2.0

* Baseline pain measure.
† Significantly less than all other pain measures within groups ($p \leq .05$).
‡ Significantly greater than pain measured at 96 h within groups ($p \leq .05$).
§ Significantly greater than pain measured at 72 h within groups ($p \leq .05$).

repeated measures on one factor (time) are presented in Table 2. A nonsignificant two-way interaction (group × time) was found for pain measures, indicating no significant effect over time among the two groups when pain was measured each morning and following each daily treatment ($F(7,80) = 0.45$, $p = .9908$). A significant main effect for time was found using an analysis for a main comparison ($F(7,80) = 7.83$, $p < .0001$). Pairwise comparisons indicated that there were no differences following the 20-minute treatment each day for the EXP ($F(1,80) = 2.10$, $p = .8604$) or SHAM ($F(1,80) = 0.98$, $p = .9201$) groups.[12]

DISCUSSION

The purpose of this study was to assess the effects of a combined brief (20-minute) and long-term (> 8-hour) application of LVMAS on pain and eccentric muscle strength resulting from an intense bout of eccentric exercise. The results indicate that LVMAS does not reduce pain or facilitate more rapid return of eccentric strength. Based on these results, questions remain as to the clinical efficacy of LVMAS.

A combination of brief (20-minute) and long-term (>8-hour) LVMAS treatment did not produce a significant reduction in perceived pain in the EXP group compared with the SHAM and CON groups. Denegar et al[2] also reported that treatment with LVMAS did not produce a significant reduction in perceived pain caused by an eccentric bout of exercise. They did suggest, however, that a transient analgesic response may have been produced within 24 hours following a 20-minute LVMAS treatment. A transient analgesic response was not found in the EXP group in this study.

A significant reduction in pain following musculoskeletal injuries using the LVMAS treatment protocol described by Picker[7] has been reported[5,6] (J. Halbach, 1995, personal communication). According to the treatment protocols from these reports, ice and NSAIDs were also used simultaneously with the LVMAS treatment. Therefore, the combination of ice, NSAIDs, and LVMAS may reduce pain that results from musculoskeletal injuries. However, according to the results from our study, LVMAS alone is not effective in reducing pain following a heavy bout of eccentric exercise.

LVMAS treatment did not produce a more rapid return of eccentric isotonic muscle strength in the EXP group than it did in the SHAM or CON groups. A significant decrease in eccentric isotonic muscle strength was found in all three groups 24 and 48 hours following the muscle damage protocol, but there were no differences in muscle strength among the three groups. The decrease in muscle strength reported after 24 and 48 hours is consistent with results reported by Denegar et al[2] and Weber et al.[5]

Interestingly, eccentric isotonic muscle strength returned to baseline in the EXP, SHAM, and CON groups within 72 hours following the initiation of muscle damage. Weber et al[5] tested subjects only up to 48 hours following the initiation of muscle damage and did not determine the length of time necessary for isometric and concentric strength to return to baseline. In addition, Weber et al[5] did not measure eccentric muscle strength. Denegar et al[2] reported that concentric isokinetic strength had not returned to baseline levels when measured 7 days (168 ± 4 hours) following the initiation of muscle damage.

Other studies evaluating the effect of exercise on muscle strength following DOMS have utilized either isometric or concentric isokinetic testing to determine improvement in muscle strength following DOMS.[13,14] Donnelly et al[13] reported that light eccentric exercise performed 24 hours following an intense bout of eccentric exercise significantly reduced maximal isometric strength immediately after the exercise bout but did not hinder the return to baseline maximal voluntary strength measures. To date, no other study has utilized eccentric 1RM testing 24, 48, and 72 hours following an intense bout of eccentric exercise to determine changes in muscle strength. Results from this study suggest that the minimal amount of eccentric work (two to five trials) necessary to determine maximal eccentric isotonic strength of the elbow flexors may enhance the ability to return to baseline eccentric isotonic strength. However, the LVMAS treatment did not accelerate the return of eccentric isotonic muscle strength.

In summary, the results from our study substantiate previous findings that a combined brief (20-minute) and long-term (>8-hour) application of LVMAS alone is not effective in decreasing pain and restoring muscle function following an intense bout of eccentric exercise. With the extremely limited experimental research available concerning LVMAS and the treatment of pain, more controlled studies are warranted to validate the cause and effect of LVMAS and establish effective treatment protocols and parameters. Future studies include the need to determine (1) the efficacy between probes and electrode application for LVMAS treatment; (2) the cause-and-effect differences of other noninvasive methods in conjunction with LVMAS, such as ice, heat, or NSAIDs; and (3) the efficacy of LVMAS on edema or effusion from musculoskeletal injury.

ACKNOWLEDGMENTS

We thank Monad Corporation (Pomona, CA) for the use of the microcurrent units and also for providing the sham units, and David A. Rowe, PhD, for his help and guidance with the statistical analysis.

REFERENCES

1. Alon G, Fink AM, Anderson PA. The effect of subliminal stimulation on elbow flexors strength, fatigue, and soreness. *Phys Ther.* 1988;68:789. Abstract.
2. Denegar CR, Yoho AP, Borowicz AJ, Bifulco N. The effects of low-volt, microamperage stimulation on delayed onset muscle soreness. *J Sport Rehabil.* 1992;1:95–102.
3. Kulig K, Isles S, Rapaski D, Smith J. Comparison of three microcurrent stimulation protocols in reducing delayed onset muscle soreness and edema. *Abstracts from the 65th Annual Conference of the American Physical Therapy Association.* American Physical Therapy Association. 1990:R222. Abstract.
4. Rapaski D, Isles S, Kulig K. Microcurrent electrical stimulation: comparison of two protocols in reducing delayed onset muscle soreness. *Phys Ther.* 1991;71:S115–S116.
5. Weber MD, Servedio FJ, Woodall WR. The effects of three modalities on delayed onset muscle soreness. *J Orthop Sports Phys Ther.* 1994;20:236–241, 249.
6. Foley J. *M.E.N.S. Microcurrent Update.* Pomona, CA: Monad Corporation, 1993.
7. Picker RI. Low-volt pulsed microamp stimulation; Part II. *Clinical Management.* 1989;9:28–33.
8. Denegar C, Perrin D. Effect of transcutaneous electrical nerve stimulation, cold, and a combination treatment on pain, decreased range of motion, and strength loss associated with delayed onset muscle soreness. *Athl Train, JNATA.* 1991;27:200–206.
9. Armstrong RB. Mechanisms of exercise-induced delayed onset muscular soreness: a brief review. *Med Sci Sports Exerc.* 1984;16:529–538.
10. Byrnes WC, Clarkson PM, White JS, Hsieh SN, Frykman PN, Maughan RJ. Delayed onset muscle soreness following repeated bouts of downhill running. *J Appl Physiol.* 1985;59:710–715.
11. Newham DJ, Mills KR, Quigley BM, Edwards RHT. Pain and fatigue after concentric and eccentric muscle contractions. *Clin Sci (Colch).* 1983;64:55–62.
12. Keppel G. *Design and Analysis: A Researcher's Handbook.* Englewood Cliffs, NJ: Prentice Hall; 1991:367–389.
13. Donnelly AE, Clarkson PM, Maughan RJ. Exercise-induced muscle damage: effects of light exercise on damaged muscle. *Eur J Appl Physiol.* 1992;64:350–353.
14. Hasson S, Barnes W, Hunter M, Williams J. Therapeutic effect of high speed voluntary muscle contractions on muscle soreness and muscle performance. *J Orthop Sports Phys Ther.* 1989;10:499–507.