Various Treatment Techniques on Signs and Symptoms of Delayed Onset Muscle Soreness

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ABSTRACT: Eccentric activities are an important component of physical conditioning and everyday activities. Delayed onset muscle soreness (DOMS) can result from strenuous eccentric tasks and can be a limiting factor in motor performance for several days after exercise. An efficacious method of treatment for DOMS would enhance athletic performance and hasten the return to activities of daily living. The purpose of this study was to identify a treatment method which could assist in the recovery of DOMS. In the selection of treatment methods, emphasis was directed toward treatments that could be rendered independently by an individual, therefore making the treatment valuable to an athletic trainer in team settings. DOMS was induced in 70 untrained volunteers via 15 sets of 15 eccentric contractions of the forearm extensor muscles on a Lido isokinetic dynamometer. All subjects performed a pilot exercise bout for a minimum of 9 weeks before data collection to assure that DOMS would be produced. Data were collected on 15 dependent variables: active and passive wrist flexion and extension, forearm girth, limb volume, visual analogue pain scale, muscle soreness index, isometric strength, concentric and eccentric wrist total work, concentric and eccentric wrist average peak torque, and concentric and eccentric angle of peak torque. Data were collected on six occasions: pre- and post-induced DOMS, 20 minutes after treatment, and 24, 48, and 72 hours after treatment. Subjects were randomly assigned to 1 of 7 groups (6 treatment and 1 control). Treatments included a nonsteroidal anti-inflammatory drug, high velocity concentric muscle contractions on an upper extremity ergometer, ice massage, 10-minute static stretching, topical *Arnica montana* ointment, and sublingual *A. montana* pellets. A 7 × 6 ANOVA with repeated measures on time was performed on the delta values of each of the 15 dependent variables. Significant main effects ($p < .05$) were found for all of the dependent variables on time only. There were no significant differences between treatments. Therefore, we conclude that none of the treatments were effective in abating the signs and symptoms of DOMS. In fact, the NSAID and *A. montana* treatments appeared to impede recovery of muscle function.

Muscle soreness is a common occurrence following unaccustomed physical activity. Muscle soreness has been differentiated into "acute"[27,50] and "delayed onset"[1,3,50] corresponding to the time in which soreness occurs. Delayed onset muscle soreness (DOMS) presents as tenderness to palpation and/or movement[35] and decreases in flexibility and maximal voluntary force production.[5,50] DOMS is believed to result from eccentric muscle activity[5,8,27,43,50] and intense isometric exercise.[11] By impairing function 24 to 48 hours posteccentric muscle activity, DOMS may limit the ability to perform activities of daily living, therapeutic exercise, or sports participation.[24]

No intervention strategies currently exist for preventing DOMS. The only alternative is to treat the signs and symptoms after they occur. Numerous investigators have attempted to identify treatments for DOMS,[7,16,17,24,29–37,55] but none of the studies reviewed addressed all signs and symptoms; the majority only assessed muscle soreness.[1,29,33,35,37,55] Only eight studies involved the assessment of muscle function.[16,24,29–32,35,36] The purpose of this study was to determine the effect of the nonsteroidal anti-inflammatory drug (NSAID) Daypro (Searle Pharmaceutical, Skokie, IL), high velocity concentric muscle activity, ice massage, static stretching, topical *Arnica montana* ointment (Boiron, Norwood, PA), sublingual *A. montana* pellets (Boiron), and a placebo on the signs and symptoms of DOMS indicated by active and passive range of motion (ROM), forearm girth, limb volume, muscle soreness, and muscle function.

METHODS

Thirty-five males and 38 females, aged 21 to 40 years, volunteered for this study. Three subjects were lost through attrition. The protocol was approved by the Temple University Institutional Review Board. All subjects gave written informed consent and completed a health questionnaire to screen for high blood pressure, heart disease, diabetes, upper extremity pathology, and medication. We excluded pregnant and nursing women and individuals with a history of liver and kidney dysfunction, peptic ulcer disease, and asthma. All subjects were familiarized with the experimental procedure via one practice session, informed of the possible risks, and instructed to abstain from all vigorous physical activities and from all

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medications 1 week before and during the data collection phase of the study.

Pilot Study

All volunteers completed a pilot session of 15 sets of 15 repetitions of eccentric wrist extension at a velocity of 30°/s with the nonwriting upper extremity. They rested 1 minute between sets. The exercise was performed on a calibrated Lido isokinetic dynamometer (Loredan Biomedical, West Sacramento, CA) in the eccentric mode. Each subject sat in the manufacturer's recommended position with the shoulder in an anatomically neutral position, the elbow flexed to 90°, and the forearm fully pronated. The subject's limb was passively returned to an extended position after each eccentric contraction to ensure that only eccentric extension was performed. Wrist ROM was limited to 50° flexion and 50° extension. We calculated and recorded total eccentric work. This pilot session was important to confirm the production of muscle soreness. Subjects completed a pain analogue rating 24 and 48 hours postexercise to determine the extent of muscle soreness elicited. If the score on the pain analogue scale was ≥3 on a scale of 0 to 10, the subject proceeded to the next phase. If soreness was reported to be <3, the subject was not permitted to proceed.

Measurement Procedures

Data collection began a minimum of 9 weeks after the pilot session to minimize or eliminate the "repeated bout effect."[8] Immediately before data collection, all female subjects took a pregnancy test (EPT; Parke-Davis, Morris Plains, NJ). Those who tested positive were not permitted to proceed. The same investigator (DTG) took all measurements throughout the study. Baseline data, identified as "Assessment Time 1," were obtained for all subjects. This included active and passive ROM of the wrist, forearm girth and volume, muscle soreness via a visual analogue pain scale and modified punctate test, and muscle function via isometric force production and isokinetic testing. We obtained two measurements for all variables except the modified Newman punctate test, with the means recorded. The reliability and precision of the measurements were quantified through the calculation of the intraclass correlation (ICC) and standard error of measurement (SEM).[15]

ROM. We obtained active and passive wrist ROM via standard goniometry[6,41,46,48,49] with active ROM assessed before passive ROM and extension measured before flexion. All measurements were taken in the seated position with the elbow stabilized in full extension and the forearm pronated. ICC(2,1) = .95 and SEM = .31 for active and passive wrist flexion and extension.

Edema. We took forearm circumferential measurements with a Gullick anthropometric measuring tape. This tape measure is equipped with a strain gauge to ensure consistent tension on the tape for reliable measurements. The epicondyles of the humerus were identified with a permanent marker. We placed additional marks on each subject's forearm at 2.5, 5, 7.5, 10, and 12.5 cm distal to each epicondyle. Measurements: ICC(2,1) = .95; SEM = .02 cm.

Upper extremity edema was evaluated with a volumeter (Volumeters Unlimited, Redland, CA), which is an open-water filled plethysmograph. Accuracy of the volumeter has been reported to be within 1% for repeated measures (<25 mL) with a high correlation (r = .97) between volume displacement and circumferential measurements.[45] The subject slowly submerged his/her extremity in the 33°C water bath via a standardized method.[51] The water displaced from the volumeter was carefully collected in a container and measured in a graduated cylinder. Measurements: ICC(2,1) = .95; SEM = 6.27 mL.

Muscle soreness. We assessed muscle soreness two ways. The first method was a visual analogue pain scale.[40,47] The visual analogue pain scale consisted of a 10-cm line with descriptors at each end. At the left end there was the number zero with the descriptor *no soreness at all*, and at the right end there was the number ten with the descriptor *soreness as bad as it could be*. The visual analogue pain scale has been used as a valid and reliable measurement for determining the intensity of human pain.[40,47] Only when pain is measured on a ratio scale can a meaningful statement be made about a given percentage of pain reduction.[47] Each subject placed an "x" along a 10-cm line to describe the amount of muscle soreness he/she was presently experiencing with active wrist ROM. The investigator then measured from the no soreness at all end to the "x" (to the nearest 0.1 cm).

The second method of assessing muscle soreness was with a modified Newham punctate technique.[29,43] We invaginated a polyurethane sheet shaped to fit the forearm with 10 punctate sites that spanned the length and width of the wrist extensor musculature. With the elbow flexed to 90°, the forearm pronated, and the wrist placed in the neutral position, we secured the polyurethane sheet to the forearm with VELCRO® straps. The measurement device was a Model 75 force gauge probe manufactured and tested by Technical Products Company (Caldwell, NJ). This device consisted of a blunt 2-mm probe attached to a force gauge with a capacity of 14 lb and a sensitivity range of 4-oz increments. A gradually increasing force was applied to each site. Each subject verbally indicated when the force reached a level of muscle discomfort. We calculated the muscle soreness index by taking the inverse of the amount of force applied to each site and summing the 10 sites. Although an increase in muscle soreness was demonstrated by a decrease in the amount of pressure tolerated by the probe, by inverting the sum of the probe sites, the muscle soreness index would also increase. This inversion of the data eased statistical analysis. The force gauge probe is comparable to weights traceable to the National Bureau of Standards (r = .99; ICC(2,1) = .98 and SEM = .03 lb) [unpublished data (1987 to 1991) of Donald C. Meserlian].

Muscle Function

The MicroFET dynamometer (Hoggin Health Industries, Draper, UT) was used to measure isometric wrist force. The MicroFET dynamometer is a microprocessor-controlled, handheld transducer that measures force in a perpendicular direction. The three strain gauges measure force vectors from three

directions to provide accurate results (r>.95).[2] We measured isometric force for wrist extension with the forearm fully pronated and the elbow stabilized at end-range extension. Resistance was applied on the dorsum of the hand via a "break test"[14] and the maximum force generated was measured in pounds. Measurements: ICC(2,1) = .95; SEM = .58 lb.

Using a Lido isokinetic dynamometer in the concentric and eccentric mode at 30°/s, we evaluated isokinetic wrist extension. As per Loredan's recommendations (Personal communication with J Capobianco, September 27, 1993), subjects performed three submaximal repetitions before four maximal contractions with data collected on the second, third, and fourth repetitions. Total work, average peak torque, and angle of peak force generation was obtained for each mode of contraction. The Lido isokinetic dynamometer is both valid and reliable for the torque (r = .98) and velocity (r = 1.00) studied.[44]

Each subject then repeated the exercise bout described in the pilot session. We calculated and compared total eccentric work with that of the pilot session. Additional work was performed via supplemental set(s) if the total work of the data collection phase was less than that of the pilot session. Immediately following the exercise bout, active and passive wrist ROM, girth, volumetry, soreness assessments, and muscle function measurements were repeated in the identical order of Assessment Time 1. We recorded each series of assessments on a new data sheet to avoid biasing the investigator with previous measurement data. These measurements taken immediately after the exercise session were identified as Assessment Time 2.

Treatment

We randomly assigned subjects to 1 of 7 groups. To avoid bias they received no information regarding prior research or anticipated treatment effects. Treatment began immediately after Assessment Time 2. The subjects in Group A were given a 1,200-mg dose of the anti-inflammatory medication, oxaprozin (Daypro; Searle Pharmaceutical, Skokie, IL) immediately after the eccentric exercise. A supplemental "loading dose" of 600 mg was taken 12 hours later. Instructions were to take two tablets (600 mg each) every 24 hours for 3 days with precise times written on the tablet containers that the subjects took home. The subjects then rested for 20 minutes.

Subjects in Group B used an upper extremity ergometer at a velocity of 360°/s without resistance for 10 minutes. Verbal feedback assisted subjects in maintaining the desired velocity. Subjects then rested for 10 minutes after this activity.

Group C received an ice massage over the wrist extensors for 20 minutes. During the ice massage the subject was seated, the elbow and wrist in extension, and the forearm pronated. The ice cup was moved in circular motions along the length of the posterior forearm.

Group D executed a static stretch of the wrist extensors in the seated position. The subjects were passively placed in a position of full elbow extension with the wrist pronated and flexed to end range for 10 minutes. The subjects then rested for an additional 10 minutes.

Group E was treated with a homeopathic remedy known as *Arnica montana*. The researcher applied a thin layer (approximately 0.5 g) of 4% topical *A. montana* ointment to the posterior forearm. We instructed subjects to gently smooth a 0.5-g dose of the ointment into the skin every 8 hours, and specific times to apply the ointment were written on the unlabeled *A. montana* tube. The subjects then rested for 20 minutes. Group F was given a sublingual form (6C) of *A. montana*. Subjects took three pellets (50 g each). They received instructions to take three pellets sublingually every 8 hours for the next 3 days with specific times written on the pellet container that they took home. Subjects then rested for 20 minutes.

Group G functioned as the control group and was given a placebo provided by Searle Pharmaceutical. The placebo was identical in appearance to that of the anti-inflammatory tablets (Daypro). Subjects in this group were given two tablets initially and one additional tablet to be taken 12 hours later. Instructions given to the subjects in Group G were identical to that of the subjects in Group A. We reminded all subjects of the potential side effects outlined on the consent form and instructed them to contact the investigator immediately with any problems.

Follow-Up

We maintained consistency across groups by having each group wait the 20 minutes for reassessment to equate treatment times. All dependent variables were reassessed in the same order with careful adherence to previously stated conditions. This reassessment was identified as Assessment Time 3. Each subject reported back to the clinic at 24 (Assessment Time 4), 48 (Assessment Time 5), and 72 (Assessment Time 6) hours posteccentric activity. The containers of subjects in Groups A, E, F, and G were checked for compliance. When necessary, markings for girth measurements were darkened with the permanent marker for future identification. We reminded subjects to refrain from using any modalities or medications and to minimize their physical activity during data collection.

Data collection concluded when all subjects signed a statement of compliance for the respective intervention. We collected and inspected all containers and weighed and recorded tubes of *A. montana* ointment. Subjects who failed to adhere to the methodology were eliminated from the study and replaced to maintain equal group size of 10 subjects per group.

Statistical Analysis

ICCs and SEMs were calculated for all dependent variables.[15] Data for each dependent measure were analyzed with 7 × 6 ANOVA with repeated measures over time.[19] Using Newman-Keuls post hoc tests, we determined where significant differences occurred between and within groups ($p < .05$). One-factor ANOVAs were also performed on the differences between Assessment Times 3 and 4, 4 and 5, and 5 and 6 for all dependent variables ($p < .05$).

RESULTS

All subjects rated their muscle soreness level as 3 or greater (0 to 10 scale) in the pilot study and progressed to the data collection phase. There were no significant differences be-

tween treatment methods for any of the variables assessed. There were significant differences between active wrist flexion ($F = 10.66$, $p < .001$), passive wrist flexion ($F = 9.65$, $p < .001$), passive wrist extension ($F = 13.10$, $p < .001$), muscle soreness index ($F = 10.89$, $p < .001$), and peak torque angle for concentric ($F = 3.65$, $p < .001$) and eccentric ($F = 7.93$, $p < .001$) contractions, indicating that the presence of DOMS impacted these measures. There were no interactions between treatments and assessment times indicating that there were no differences between the effects. In an attempt to analyze all possible differences, one-factor ANOVAs were also performed on the differences between Assessment Times 3 and 4, 4 and 5, and 5 and 6 for all dependent variables ($p < .05$). However, no significant differences were found.

Pearson product moment correlation coefficients for forearm girth and muscle soreness and limb volume and muscle soreness were −.98 and −.95, respectively. The Pearson product moment correlation coefficients for active wrist flexion and muscle soreness and active wrist extension and muscle soreness were −.98 and −.99, respectively. In an attempt to simplify the plethora of data, Figures 1 through 4 demonstrate the grand means (ie, general trends) of the treatment groups for the four areas of interest: ROM (Fig 1), edema (Fig 2), muscle soreness (Fig 3), and muscle function (Fig 4).

DISCUSSION

Because the precise pathology of DOMS is unknown, determining an appropriate course of treatment is difficult. Researchers[7,10,16,17,20,21,24,29–37,39,46,55] have attempted to prevent and treat the various symptoms of DOMS. Based on previous studies and basic physiology, the six treatment techniques were selected and compared to a placebo (control) to investigate their influence on the symptoms of DOMS.

The method selected for inducing DOMS was deemed successful, because the data for all of the dependent variables differed from Assessment Times 1 to 2. Nine of the 15 dependent variables were significantly different. Once DOMS was induced, none of the treatments significantly influenced ROM, edema, muscle soreness, or muscle function. However, there were many interesting responses to the treatment techniques. The general trends of the dependent variables will be presented first, and the specific responses of each of the treatment groups presented second.

Active wrist flexion and extension followed a similar recovery pattern, but none of the ROM measurements returned to baseline values by Assessment Time 6 (Fig 1). Likewise, the measurements of forearm girth and limb volume data revealed a similar course of recovery after the eccentric muscle activity (Fig 2). The significant increase in girth and volume observed immediately after the exercise was probably a result of increased blood flow to the exercising muscles, a local metabolic response. Increased pressure during the eccentric muscle activity can result in an increased movement of intravascular fluid into the interstitial spaces within the exercised muscle fibers.[7] Neither forearm girth or limb volume significantly increased, however, from Assessment Times 3 to 6. Previous theories[26,27,29] associate the pain of DOMS to the edema within the exercised muscle fibers; however, Buroker and Schwane[7] argued against the hypothesis that pain neurons were physically distorted by edema. They observed that girth measurements of eccentrically exercised limbs did not increase at

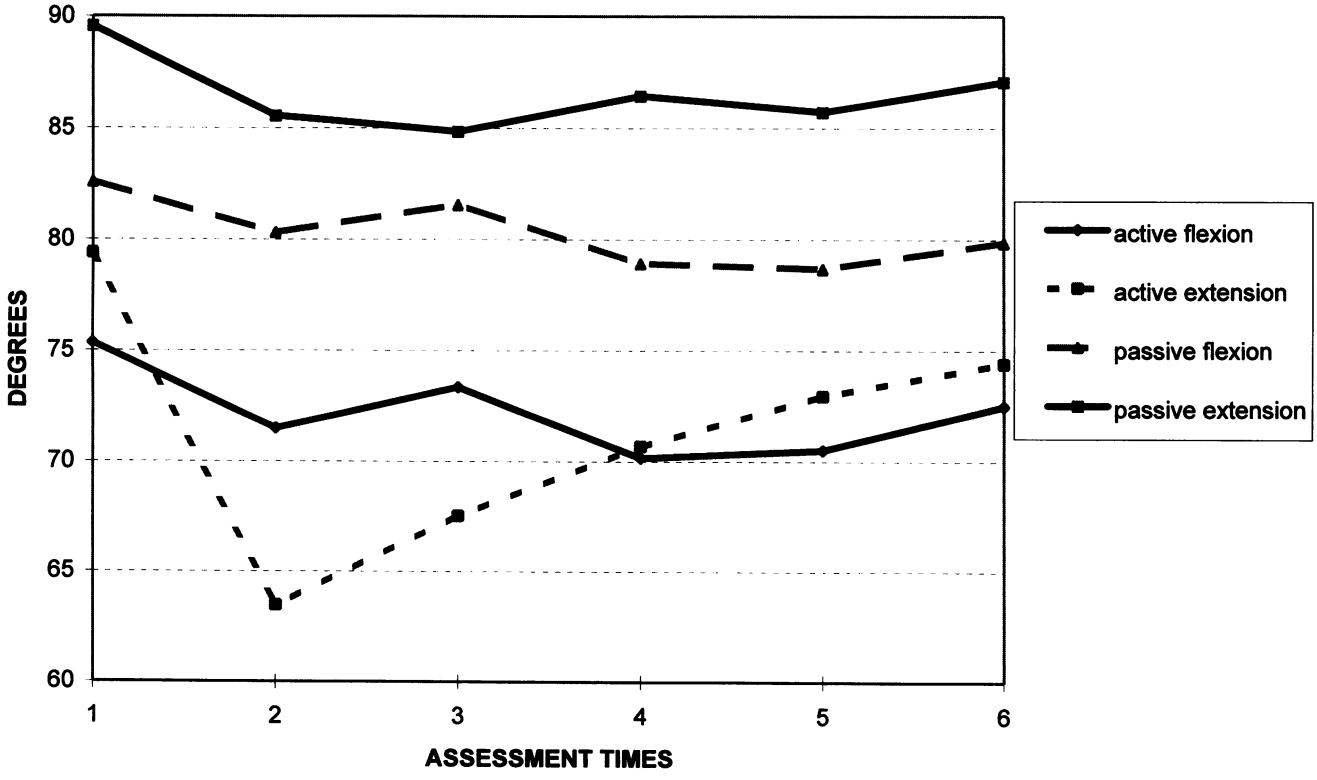

Fig 1. Wrist range of motion grand means.

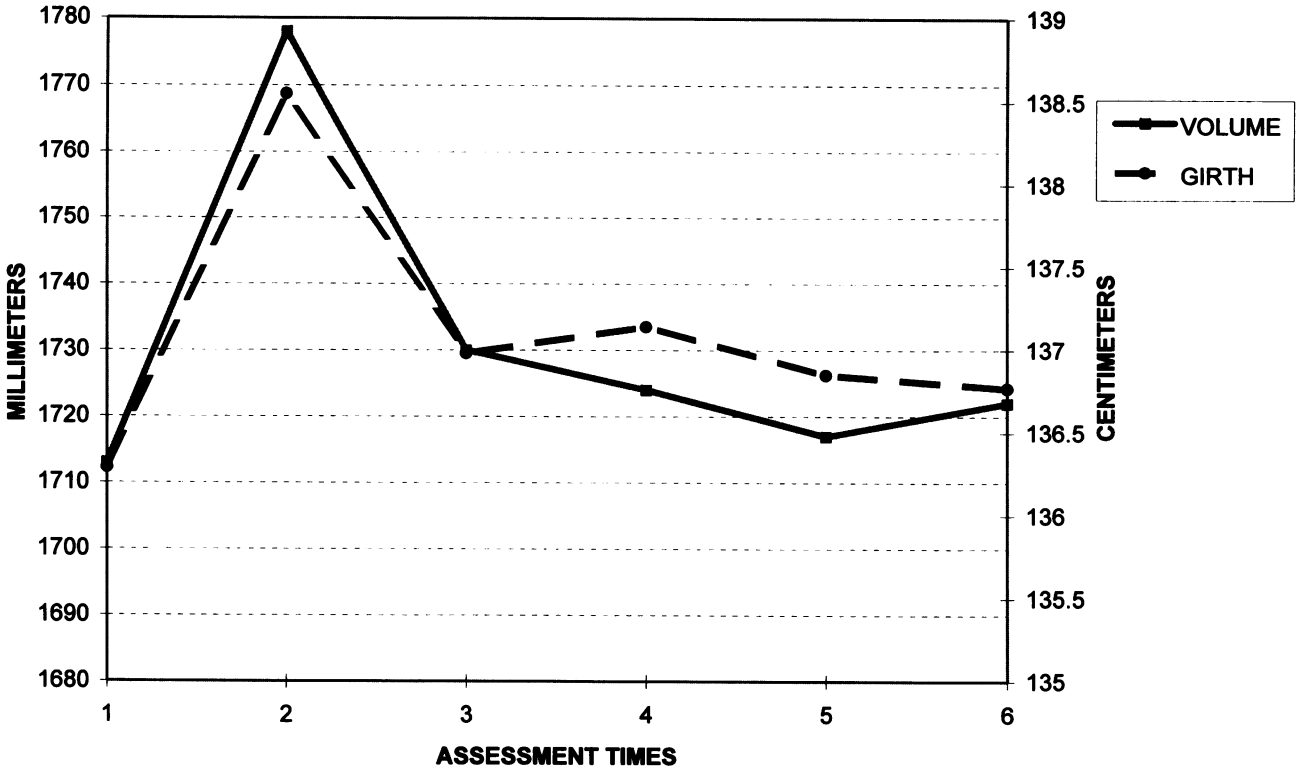

Fig 2. Forearm edema grand means.

any postexercise assessment time. The inverse relationship (ie, negative correlation) of our data supports this conclusion.[7,35]

The visual analogue pain scale data were significantly greater in Assessment Time 2 than Assessment Time 1 and a significant decrease occurred in Assessment Time 3 for all groups (Fig 3). We observed a negative correlation between muscle soreness and active ROM which is consistent with a previous report.[55]

Isometric wrist extension force data for all treatment groups followed a similar recovery pattern toward baseline measurements after DOMS was induced (Fig 4). The decreased force development in all of the groups immediately after the exercise bout (ie, from Assessment Times 1 to 2) was consistent with acute muscle soreness.[24,29,31,32] Force development increased in all groups from Assessment Times 3 to 4 and then was followed by a decrease in force generation from Assessment Times 4 to 6. These results demonstrate a recovery from acute muscle soreness by 24 hours (Assessment Time 4) and the onset of the delayed soreness by 48 hours (Assessment Time 5).

The concentric and eccentric total work and peak torque produced by the wrist extensors decreased significantly for all groups after the eccentric muscle activity. The most notable was the eccentric total work, which fell to less than 54% of the baseline values. The recovery of the concentric and eccentric muscle function appeared to parallel one another with all groups returning to a level of 79% to 90% of baseline by Assessment Time 6. This long recovery time (72 hours) suggests that the repair of the contractile elements, sarcoplasmic reticulum, and/or connective tissue is a slow process.[12]

The angle of peak concentric torque development was extremely variable between treatment groups and across time. The primary emphasis of this study was on eccentric muscle activity. The pilot phase, the exercise protocol, and all assessments involved eccentric activity. There were only three maximal concentric contractions per assessment period. This was not an adequate number of repetitions to produce a learning effect. Therefore, specificity of training may have played a role in the variability of the performance of the concentric muscle activity. The angle of peak eccentric torque development was not quite as variable. Eccentric peak torque decreased at Assessment Time 2, but the angle of peak torque generation increased. This indicates that the peak torque was generated earlier in the ROM at Assessment Time 2 and could be attributed to a learning effect. Each subject had completed over 225 eccentric muscle contractions at that point and could then "catch" the isokinetic machine earlier in the eccentric phase. By Assessment Time 3, the peak eccentric torque was occurring later in the ROM and could be related to fatigue. In the attempt to generate a maximal contraction, the muscles will increase the number of fibers recruited to maintain or increase force development. The increased fiber recruitment is time-consuming and could have delayed peak torque generation.

NSAID

The inflammatory process begins within several hours after a tissue-damaging event.[31] The signs and symptoms of DOMS do not begin until 24 to 48 hours postexercise, however. Waiting to treat the anticipated inflammatory process until the time the signs and symptoms appear has been shown to be ineffective.[20,21,30,32,33,37,39,46] Early intervention with a prophylactic NSAID has been successful in

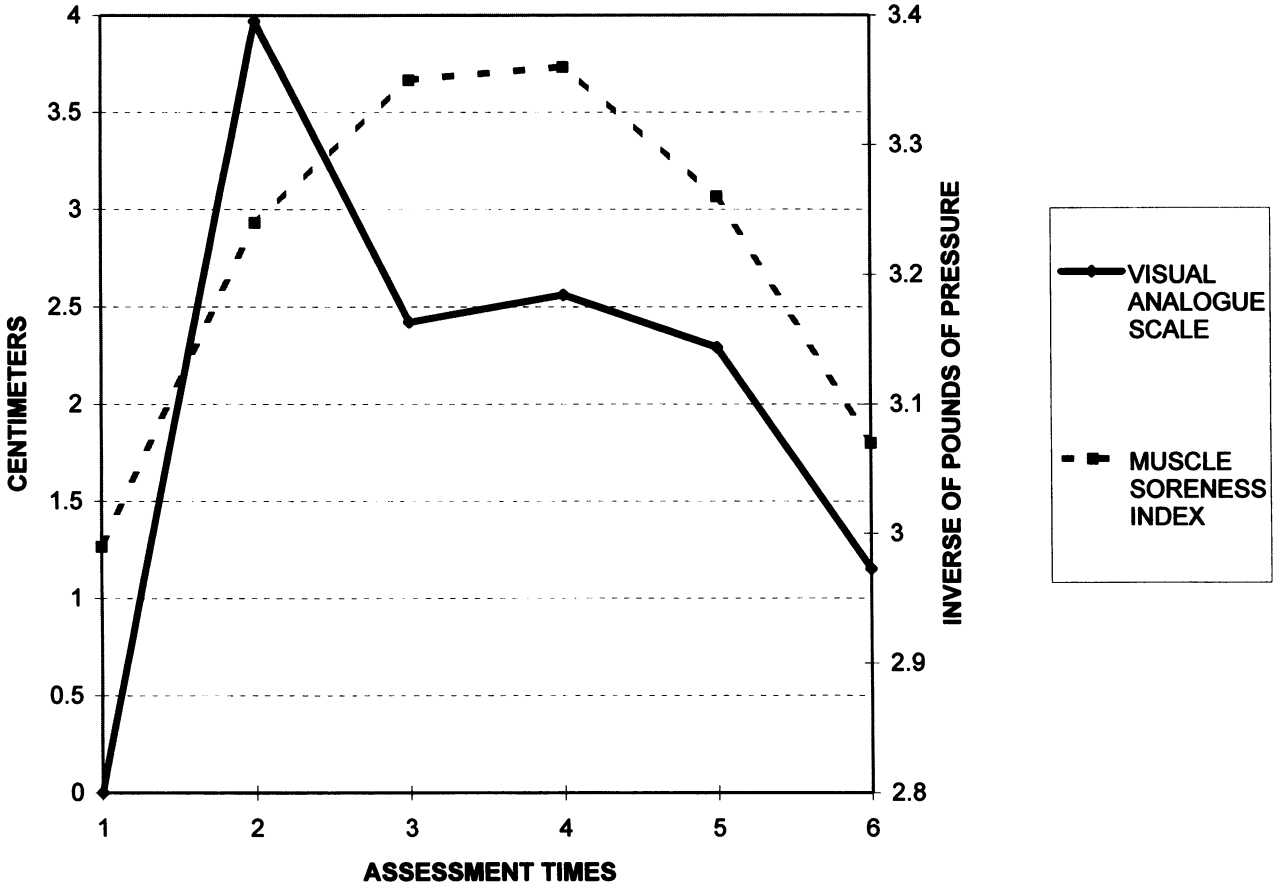

Fig 3. Muscle soreness grand means.

reducing the symptoms of DOMS.[31] Studies with varying dosages of ibuprofen[20,30] revealed contradictory results. Hasson et al[31] administered 400 mg of ibuprofen 3 times per day, whereas this investigator examined the effect of a load dose of 1,800 mg and daily doses of 1,200 mg of oxaprozin. Perhaps the inflammatory process is a necessary component of the healing process, and interference in this process impairs the recovery of muscle function. NSAIDs administered in greater dosage may in fact impede the production of myofibrillar protein.[32]

Upper Extremity Ergometer

Submaximal concentric muscle activity does not cause tissue damage and produces much lower intramuscular pressures than eccentric activity.[25] The popular belief that muscle soreness can be alleviated by "working it out" has been implemented for many years.[29] Hasson et al[29] studied the effects of high velocity concentric exercise on DOMS and found a significant reduction in muscle soreness and an improvement in muscle performance. However, neither volumetric nor circumferential limb measurements were performed to demonstrate the relationship between edema and DOMS. In this study, changes in forearm girth and limb volume did not coincide with muscle soreness. The velocity selected for treatment was similar to that of Hasson et al,[29] but there were several parameters in the present study that differed. In the present study, 10 minutes of upper extremity ergometry was performed for a total of 600 submaximal contractions, with the upper extremities immediately after the eccentric muscle activity. Hasson et al[29] used 120 maximal contractions with the lower extremities, 24 hours after the eccentric bout. Any or all of these parameters could have influenced the results of the two studies.

Ice Massage

In all groups active wrist flexion increased from Assessment Times 2 to 3; however, ROM decreased in the ice group. Lehmann and DeLateur[40] attributed an increase in collagen stiffness to the application of cold. However, after Assessment Time 5, the ROM measurements progressed toward pre-exercise levels, and the ice group was the only treatment group to return to baseline by Assessment Time 6.

The path in which the muscle soreness index data returned to baseline was similar from Assessment Times 2 to 3 for all groups except the ice treatment group. The muscle soreness index for the ice group decreased immediately following treatment. This was attributed to the numbing effect of the 20-minute ice massage. The application of a cold modality depresses the excitability of the free nerve endings and peripheral nerves, which increases the pain threshold and decreases pain.[28] The muscle soreness index data for the ice massage group notably increased from Assessment Times 3 to

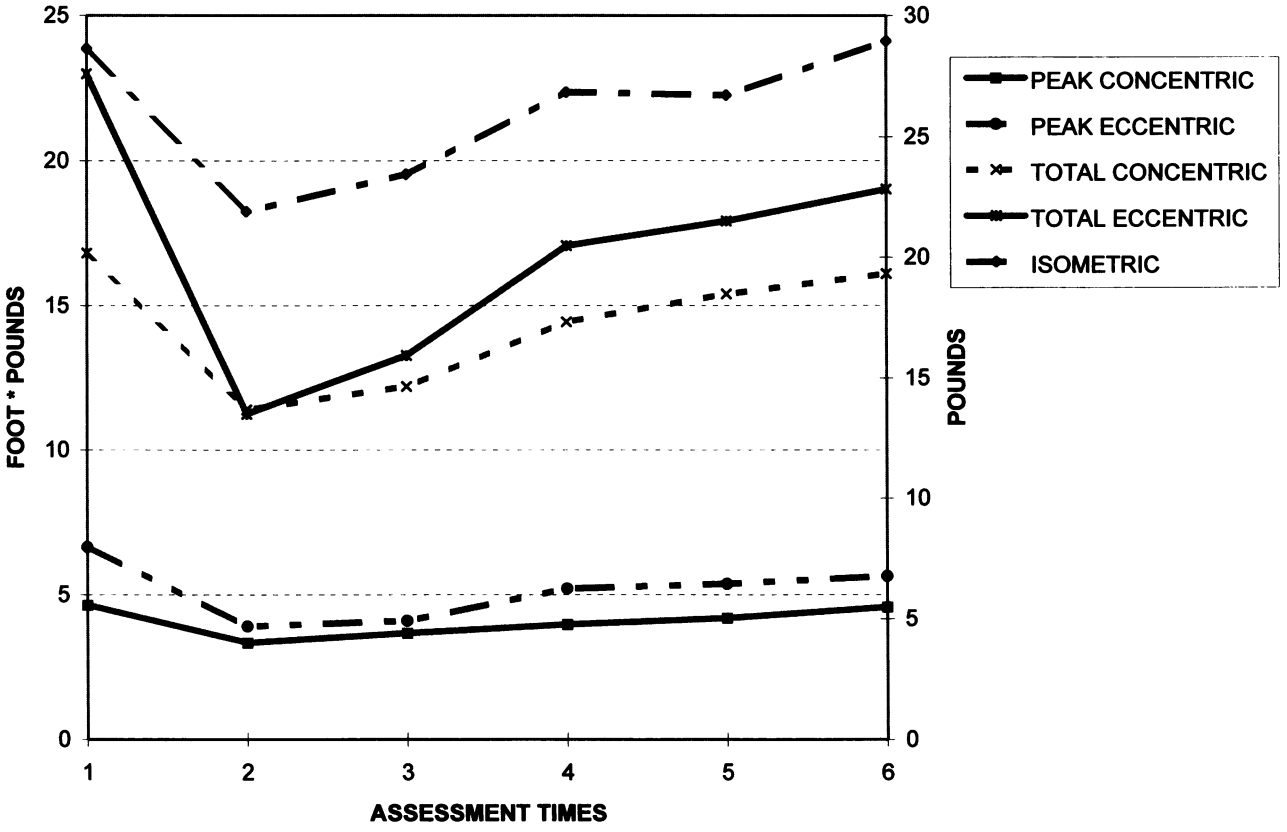

Fig 4. Wrist extension muscle function grand means.

4 and may be due to a compensatory response of the earlier numbing effect. This indicates that the ice massage provided relief from acute muscle soreness[38,41,42] but was not effective in abating DOMS. These results correspond with previous results.[54]

The ice group generated less isometric force after treatment, while other groups increased force generation. These findings are in agreement with Fox[23] who revealed a transient decrease in strength with cold application.

Static Stretching

Researchers have investigated the effects of warm-up, preexercise,[34] and postexercise[1,7,18] stretching on DOMS. The results were as varied as the stretching techniques. In this study, the greatest decrease in active wrist flexion occurred in Assessment Time 4 for all groups except the group treated with static stretching. The prolonged stretch to the forearm extensor muscles was apparently effective in maintaining active wrist flexion for at least 72 hours. This was the only parameter that was notably different in the response to the treatment of prolonged, static stretching.

Arnica montana

The use of *Arnica montana* L as a medicinal plant dates back to the 16th century.[9,13,52–54] The components of the *A. montana* compound are believed to be analgesic, antibiotic, and anti-inflammatory in nature.[9,22] Tveiten et al[52] administered five pellets of *A. montana* twice daily to marathon runners. They found a significant reduction in stiffness ratings on a visual analogue pain scale for the group treated prophylactically with *A. montana* but reported no change in muscle function. Armstrong et al[4] used animals to demonstrate that the tissue damage induced by eccentric exercise may not follow the normal inflammatory sequence. If the eccentrically induced inflammatory process is also altered in humans, the lack of improvement in ROM and muscle function with both the NSAID and *A. montana* treatments could be explained. Conversely, the preparations of *A. montana* in this study may not have been effective, because they were too diluted. Older studies with higher concentrations of this medication and European research still recommend its use for DOMS.[51] No recent double-blind studies of the effects of higher concentrations of *A. montana* on DOMS appears in the literature reviewed.

Numerous other modalities are yet to be explored. Nonetheless, the results of this study did not reveal a treatment method that was significantly better than that of a placebo for the symptoms of DOMS.

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REFERENCES

1. Abraham WM. Factors in delayed muscle soreness. *Med Sci Sports.* 1977;1:11–20.
2. Allsop KG, VanWagoner E. Application of a device for the quantitative testing of muscle strength. Presented at the RESNA 10th Annual Conference; 1987; San Jose, CA.
3. Armstrong RB. Mechanisms of exercise-induced delayed onset muscular soreness: brief review. *Med Sci Sports Exerc.* 1984;16:529–538.
4. Armstrong RB, Ogilvie RW, Schwane JA. Eccentric exercise-induced injury of rat skeletal muscle. *J Appl Physiol.* 1983;54:80–93.
5. Asmussen E. Observations on experimental muscular soreness. *Acta Rheum Scand.* 1956;2:109–116.
6. Boone DC, Azen SP, Lin CM. Reliability of goniometric measurements. *Phys Ther.* 1978;58:1355–1360.
7. Buroker KC, Schwane JA. Does post-exercise static stretching alleviate delayed muscle soreness? *Phys Sportsmed.* June 1989;6:65–83.
8. Byrnes WC, Clarkson PM, White JS, Hsieh SS, Frykman PN, Maughan RJ. Delayed onset muscle soreness following repeated bouts of downhill running. *J Appl Physiol.* 1985;59:710–715.
9. Castro M. *The Complete Homeopathy Handbook.* New York, NY: St. Martin's Press; 1991:231–232.
10. Ciccone CD, Leggin BG, Callamaro JJ. Effects of ultrasound and trolamine salicylate phonophoresis on delayed onset muscle soreness. *Phys Ther.* 1991;71:666–678.
11. Clarkson PM, Brynes WC, McCormick KM, Turcotte LP, White JS. Muscle soreness and serum creatine kinase activity following isometric, eccentric, and concentric exercise. *Int J Sports Med.* 1986;7:152–155.
12. Clarkson PM, Tremblay I. Exercise induced muscle damage, repair, and adaptation in humans. *J Appl Physiol.* 1988;1:1–6.
13. Czygan FC, Kemper FH, Reuter DR, Weiss E. *Arnica montana* bei venenleiden [*Arnica montana* for chronic venous insufficiency]. *Z Phytotherap.* 1991;5:141–145.
14. Daniels L, Worthingham C. *Muscle Testing Techniques of Manual Examination.* Philadelphia, PA: WB Saunders; 1989:126–129.
15. Denegar CR, Ball DW. Assessing reliability and precision of measurement: an introduction to intraclass correlation and standard error of measurement. *J Sport Rehabil.* 1993;2:35–42.
16. Denegar CR, Perrin DH. Effect of transcutaneous electrical nerve stimulation on pain, decreased range of motion, and strength loss associated with delayed onset muscle soreness. *J Athl Train.* 1992;27:200–206.
17. Denegar CR, Perrin DH, Rogol AD, Rutt R. Influence of transcutaneous electrical nerve stimulation on pain, range of motion, & serum cortisol concentration in females experiencing delayed onset muscle soreness. *J Orthop Sports Med.* Sep 1989;11:100–103.
18. deVries HA. Prevention of muscular distress after exercise. *Res Q.* 1961;32:177–185.
19. Dixon WJ. *BMDP Statistical Software.* Berkeley, CA: University of California Press; 1990.
20. Donnelly AE, Maughan RJ, Whiting PH. Effects of ibuprofen on exercise-induced muscle soreness and indices of muscle damage. *Br J Sports Med.* Sep 1990;3:191–195.
21. Donnelly AE, McCormick K, Maughan RJ, Whiting PH, Clarkson PM. Effects of a non-steroidal anti-inflammatory drug on delayed onset muscle soreness and indices of damage. *Br J Sports Med.* Mar 1988;1:35–38.
22. Duke J. *Handbook of Medicinal Herbs.* New York, NY: CRC Press; 1985:64.
23. Fox RH. Local cooling in muscle. *Br Med Bull.* 1961;17:14–18.
24. Francis KT, Hoobler T. Effects of aspirin on delayed muscle soreness. *J Sports Med Phys Fitness.* 1987;27:333–337.
25. Friden J. Muscle soreness after exercise: implications of morphological changes. *Int J Sports Med.* 1984;5:57–66.
26. Friden J, Sfakianos PN, Hargens AR. Muscle soreness and intramuscular fluid pressure: comparison between eccentric and concentric load. *J Appl Physiol.* 1986;61:2175–2179.
27. Friden J, Sjostrom M, Ekblom BA. A morphological study of delayed muscle soreness. *Experientia.* 1981;37:506–507.
28. Haines J. A survey of recent developments in cold therapy. *Physiotherapy.* 1967;53:222.
29. Hasson S, Barnes W, Hunter M, Williams J. Therapeutic effects of high speed voluntary muscle contractions on muscle soreness and muscle performance. *J Orthop Sports Phys Ther.* Jun 1989;10:499–507.
30. Hasson S, Daniels J, Divine J, et al. Effect of ibuprofen use on muscle soreness, damage, and performance: preliminary investigation. *Med Sci Sports Exerc.* 1993;1:9–17.
31. Hasson S, Mundorf R, Barnes W, Williams J, Fujii M. Effects of pulsed ultrasound versus placebo on muscle soreness perception and muscular performance. *Scand J Rehabil Med.* 1990;22:199–205.
32. Hasson S, Wible CL, Reich M, Barnes WS, Williams JH. Dexamethasone iontophoresis: effect on delayed muscle soreness and muscle function. *Can J Sport Sci.* 1992;1:8–13.
33. Headley SAE, Newham DJ, Jones DA. The effects of prednisolone on exercise induced muscle pain and damage. *Clin Sci.* 1986;70:85.
34. High DM, Howley ET, Franks BD. The effects of static stretching and warm-up on prevention of delayed onset muscle soreness. *Res Q Exerc Sport.* 1989;60:357–361.
35. Hill DW, Richardson JD. Effectiveness of 10% trolamine salicylate cream on muscular soreness induced by a reproducible program of weight training. *J Orthop Sports Phys Ther.* Jul 1989;1:19–23.
36. Isabell WK, Durrant E, Myrer W, Anderson S. The effects of ice massage, ice massage with exercise, and exercise on the prevention and treatment of delayed onset muscle soreness. *J Athl Train.* 1992;27:208–217.
37. Janssen E, Kuipers H, Verstappen F, Costill D. Influence of an anti-inflammatory drug on muscle soreness. *Med Sci Sports Exerc.* 1983;15:165.
38. Knight K. The effects of hypothermia on inflammation and swelling. *Athl Train, JNATA.* 1976;11:7–10.
39. Kuipers H, Keizer HA, Verstappen J, Costill DL. Influence of a prostaglandin-inhibiting drug on muscle soreness after eccentric work. *Int J Sports Med.* 1985;6:336–339.
40. Lee KA, Kieckhefer GM. Measuring human responses using visual analogue scales. *West J Nurs Res.* 1989;11:128–132.
41. Lehmann JF, DeLateur BJ. Cryotherapy. In: Lehman JF, DeLateur BJ, eds. *Therapeutic Heat and Cold.* Baltimore, MD: Williams & Wilkins; 1990:590–632.
42. Lehmann JF, Warren CG, Scham SM. Therapeutic heat and cold. *Clin Orthop.* 1974;99:207–237.
43. Newham DJ, Mills KR, Quigley BM, Edwards RHT. Pain and fatigue after concentric and eccentric contractions. *Clin Sci.* 1983;64:55–62.
44. Patterson LA, Spivey WE. Validity and reliability of the LIDO active isokinetic system. *J Orthop Sports Phys Ther.* 1992;1:32–36.
45. Persson NH, Takolander R, Bergqvist D. Lower limb oedema after arterial reconstructive surgery. *Acta Chir Scand.* 1989;155:259–266.
46. Politino V, Smith SL, Waggoner WC. A clinical study of topical trolamine salicylate for relief of delayed onset exercise-induced arthralgia/myalgia. *Curr Ther Res.* 1984;38:321–327.
47. Price DD, McGrath PA, Rafii A, Buckingham B. The validation of visual analogue scales as ratio scale measures for chronic and experimental pain. *Pain.* 1983;17:45–56.
48. Rothstein JM, Miller PJ, Roettger RJ. Goniometric reliability in a clinical setting: elbow and knee measurements. *Phys Ther.* 1983;63:1611–1615.
49. Stratford P, Agostino V, Brazeau C, Gowitzke BA. Reliability of joint measurements: a discussion of methodology issues. *Physiother Can.* 1984;36:5–9.
50. Talag TS. Residual muscular soreness as influenced by concentric, eccentric and static contractions. *Res Q.* 1973;44:458–469.
51. Thulesius O, Norgren L, Gjores JE. Foot volumetry, a new method for objective assessment of edema and venous function. *Vasa.* 1973;4:325–329.
52. Tveiten D, Bruseth S, Borchgrevink CF, Lohne K. Effect of Arnica D30 during hard physical exertion: double-blind randomized trial during the Olso marathon 1990. *J Norske Inst Natl Med.* 1991;30:3630–3631.
53. Willuhn G. *Arnica montana* L.—Portrat einer Arzneipflanze [*Arnica montana* L.—Portrait of a medical plant]. *Pharmacon.* 1991;13:9–26.
54. Willuhn G. Untersuchunger uber die inhaltsstoffe von *Arnica*-arten [Understanding the contents of the *Arnica* herb]. *Planta Med.* 1972;3:221–245.
55. Yackzan L, Adams C, Francis KT. The effects of ice massage on delayed muscle soreness. *Am J Sports Med.* 1984;12:159–165.