

## The Effects of Continuous External Compression on Delayed-Onset Muscle Soreness (DOMS)

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*Delayed-onset muscle soreness (DOMS) is a sensation of soreness that develops 24–48 hr after intense unaccustomed exercise. Clinical characteristics of DOMS include local tissue edema, decreased muscle strength, and decreased range of motion. Although controversial, some research has implicated swelling as a cause or contributor to soreness. We designed this study to determine the effects of continuous external compression on swelling resulting from eccentric contraction and the accompanying clinical characteristics of DOMS. Twenty-three healthy college students (16 females, 7 males;  $\bar{X}$  age = 26.0 years) completed 70 maximal eccentric contractions of the elbow flexors to induce soreness and then received random assignment to either a control or a compression sleeve group. The compression sleeve group wore an elastic compression sleeve on the exercised arm, extending from the deltoid insertion to the wrist, throughout the study. We obtained measures of subjective soreness, range of motion (ROM), circumference, arm volume, and isokinetic peak torque for both groups immediately before exercise and 10 min and 24, 48, and 72 hr after exercise. No significant differences were present between groups at any time reference for any of the dependent variables. Soreness peaked at 48 hr and then began to decline. Circumference and volume measurements increased over time with the greatest difference occurring 72 hr postexercise. Subject's ROM and peak torque decreased immediately following exercise and continued to be lower than baseline during the next 72 hr. Continuous external compression, which prior research has shown to be an effective treatment of edema resulting from a variety of acute injuries, was not effective in reducing the edema associated with DOMS, nor was it effective in reducing soreness, strength loss, or ROM loss as a result of DOMS in elbow flexors. Therefore, the clinical use of compression garments in treating DOMS in upper extremities is questionable.*

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**KEY WORDS:** muscle soreness; swelling; compression.

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## INTRODUCTION

Delayed-onset muscle soreness (DOMS), commonly associated with an intense bout of unaccustomed exercise that involves a significant eccentric component (Armstrong, 1984; Armstrong *et al.*, 1980; Bobbert *et al.*, 1986; Fitzgerald *et al.*, 1991; Hough, 1902; Tälåg, 1973), is a sensation characterized as a dull, aching pain that occurs in the muscle within 24–48 hr after the exercise. Patients with DOMS often describe the affected muscles as “stiff” or “tender,” becoming increasingly sensitive after 2 or 3 days and then diminishing in sensitivity until the soreness dissipates at approximately 7 days (Armstrong, 1984; Tälåg, 1973). The irritation usually is primarily near the musculotendinous junction in the affected extremity (Edwards *et al.*, 1981).

DOMS clinically presents as increased stiffness, electrically silent muscle shortening, a decreased range of motion, tenderness, decline in force, and release of muscle enzymes into the blood and swelling (Armstrong, 1984; Cleak and Eston, 1992; Kuipers *et al.*, 1985; Schwane *et al.*, 1983). To date, there has been no general agreement about the pathophysiological mechanisms responsible for DOMS (Armstrong, 1984; Friden *et al.*, 1983b; Kuipers *et al.*, 1985). However, there is general agreement that elevated muscle tension resulting from intense unaccustomed eccentric exercise does produce myofiber damage as well as alterations to the extracellular matrix (Friden *et al.*, 1983a; Newham, 1988; Newham *et al.*, 1983). Myofiber disruption and/or extracellular matrix discontinuity may release protein-associated ions, resulting in increased osmotic pressure and muscle edema (Friden *et al.*, 1986; Stauber *et al.*, 1990). The resultant edema would cause a reduction in resting length of the muscle as the swollen tissue pushes against the fascia and shortens the muscle passively. Progression of muscle edema and increased intramuscular pressure may relate to the delayed onset response of muscle soreness perception. Friden *et al.* (1986) demonstrated individual muscle fiber swelling and an inflammatory response following eccentric exercise. Friden asserted that fiber disruption leads to formation of degraded protein components, release of protein-bound ions, and a subsequent increase in osmotic pressure and fluid buildup. Fluid pressure may then activate group IV sensory neurons that terminate in free nerve endings distributed primarily in the muscle connective tissue between fibers, thereby producing the sensation of dull diffuse pain associated with DOMS.

Therapy for DOMS has included high speed voluntary muscle contractions, topical application of thermal agents, ultrasound, orally administered analgesics, and antiinflammatories. These therapies have produced questionable and mixed results (Donnelly *et al.*, 1988, 1990; Francis and Hoobler, 1987, 1988; Hasson *et al.*, 1989, 1993; Yackzan *et al.*, 1984). The only generally recognized prevention of DOMS is prior training in the particular exercise (Blanave and Thompson, 1993).

External compression, a widely recognized standard care for acute musculoskeletal injury, has not undergone investigation as a treatment for DOMS (Wilkinson, 1985). This treatment technique reduces edema by producing external pressure to oppose the internal pressure created from fluid and the osmotically active particles contained in the edematous fluid (Sarin *et al.*, 1992). Applying external compression inhibits seepage of fluid into underlying tissue spaces and dis-

perses excess fluid (Sarin *et al.*, 1992; Wilkerson, 1985). Thus, if compression inhibits seepage, as measured by limb volume and circumference, then resultant scores on measures of soreness, strength, and range of motion should change minimally over time compared to uncompressed exercised arms. Therefore, the purpose of this study was to evaluate the effect of applying continuous external compression commonly used in other types of musculoskeletal injuries on DOMS and the accompanying manifestations of soreness, swelling, range of motion, and isokinetic strength for a period of 4 days following induction of DOMS. The first research hypothesis was that exercised arms receiving external compression postexercise would show significantly less swelling over time compared to exercised arms not receiving compression. The second hypothesis was that this same pattern would obtain for measures of pain, strength, and range of motion.

## METHODS

### Subjects

We obtained informed consent prior to testing in accordance with the policy statement regarding the use of human subjects of Medicine and Science in Sports and Exercise and the University of Alabama at Birmingham internal review board. Subjects received a written form of instructions to follow during the three days of study. Included in the list of written guidelines were instructions not (a) to take any aspirin or antiinflammatory medications; (b) to perform upper-extremity exercises of the involved muscles during the 72 hr of investigation; (c) to use any analgesics, topical ointments, ice, or any other type of pain relief treatment for the 72 hr of the study; or (d) to remove the compression sleeve except during testing and measurement at the laboratory.

### Sample Size

The estimate for the size of the study's sample derives from the effect sizes obtained by two related published studies. Hasson *et al.* (1989) found a significant difference in quadriceps soreness 48 hr post DOMS induction between the control group and the group that received a regimen of high-speed muscle contractions following DOMS induction. The effect size obtained was approximately 1.0. In a study of the effects of elastic compression on venous stasis by Lewis *et al.* (1976), full leg-length elastic compression significantly reduced the time for venous clearance compared to noncompressed legs for the entire leg and the thigh, popliteal, and calf sections. Effect sizes were between .82 and 1.25. Because these studies differed in intervention, site, and dependent measures, we selected a conservative effect size of .7. For the proposed ANOVA design, in which the comparison of interest is the control versus the treatment groups, we determined that the number of subjects needed for  $\alpha = .05$  and  $\beta = .20$  was 20 (Cohen, 1977).

We recruited 23 healthy 23- to 42-year-old college students (16 females, 7 males) from the University of Alabama at Birmingham, Division of Physical Therapy. The mean age for the group was  $26 \pm 4$  years, the mean weight was  $65.3 \pm 11.5$  kg, and the mean height was  $170.2 \pm 7.5$  cm. None of the participants in the study reported involvement in weight-lifting programs, had soreness in their arms, or had any musculoskeletal problems involving the upper extremity within 6 months prior to the study.

Because the literature contains references to the use of both upper and lower compartments in the study of DOMS (Armstrong, 1984; Bobbert *et al.*, 1986; Francis and Hoobler, 1988; Friden *et al.*, 1986; Talag, 1973) and because of this laboratory's successful experience with using the upper extremities, we selected the elbow flexors for DOMS inducement. We obtained measurements of subjective soreness, range of motion, proximal, middle, and distal circumference, limb volume, and isokinetic mean peak flexor torque before exercise and 10 min and 24, 48, and 72 hr after exercise for both the treatment and the control groups. Subjects assigned randomly to the treatment group wore an elastic compression sleeve throughout the entire study, except 10–15 min each day during which we obtained measurements in the laboratory. Control subjects wore no sleeve during the 72 hr of the study. Of the seven male subjects, four were in the control group and three were in the compression sleeve group.

### Instrumentation

We induced DOMS by eccentric exercise performed in resisting the LIDO 2 isokinetic dynamometer (Loredan Biomedical Inc., West Sacramento, CA). Each participant performed 70 eccentric contractions of the nondominant elbow flexor muscles, opposing the motion of the dynamometer moving at  $120^\circ/\text{sec}$  through  $120^\circ$  of motion beginning at an angle of  $120^\circ$  (Stauber *et al.*, 1990). Subjects used the seated testing position, as described by the manufacturer, with the nondominant arm resting at the head of a padded incline table and the dominant hand grasping onto the side of the table. Restraining straps were in place around the waist, across the chest, and loosely over the upper arm of the involved limb. Each movement was a maximal effort to resist the ability of the dynamometer to extend the elbow. A 1.5-sec rest between each exercise movement allowed the dynamometer to return passively to the starting position. Thus, the participant performed only eccentric muscle activity (i.e., resisted muscle lengthening).

To ensure that subjects were performing vigorous contractions, they received verbal instructions to work harder when visual torque readings from the computer were more than 5 ft lb less than the average of the initial five contractions. Following the eccentric exercise to induce DOMS, subjects received random assignment either to be controls or to wear a compression sleeve. The compression sleeve worn by the sleeve group consisted of two thickness of 7.2-cm-diameter elastic sleeve (Brecon Inc., Talladega, AL) extending from a transverse line bisecting the deltoid insertion to the wrist.

We determined the pressure produced by the elastic compression sleeve from a standard pressure curve obtained by measuring the pressure of the sleeve on cylinders of known diameters, using a mercury sphygmomanometer. We obtained pressure readings by first placing the blood pressure cuff around a fixed cylinder of known diameter. We then inserted the cuff and cylinder into the compression sleeve, and the cuff inflated to 60 mm Hg. We subsequently removed the sleeve and recorded the pressure. The difference between the final reading and 60 mm Hg was the amount of pressure produced by the sleeve. We repeated this process for several known diameters and compiled a pressure–diameter linear regression. Average sleeve pressure over the area corresponding to diameter measurements for the average arm circumference was 17 mm Hg.

We obtained subjective soreness ratings using Henry's scale for assessing muscle soreness patterned after Tälåg (1973). The scale ranges from 0, representing no soreness, to 6, representing unbearable soreness. The scale also incorporates a visual scale using character drawings of facial expressions representing various levels of discomfort corresponding to the numerical scale.

We measured elbow extensor range of motion with a 360° circle plastic goniometer with 1° increments and obtained angle measurements in the relaxed supine position with the arm passively resting at the side. The investigators made no effort to alter the angle. A pad was present under the upper arm to allow unrestricted gravitational motion of the elbow (full extension = 0°). We took measurements in less than 1 min after placing the elbow on the pad, using the ulnar styloid and the midline of the proximal humerus as proximal and distal markers.

We measured edema using both circumference and volumetric measurements. We determined volumetric measurements by measuring the amount of water displaced by immersing the exercised limb in a water plethysmograph. We duplicated the area immersed by using a line drawn transversely across the upper limb through the insertion of the deltoid into the humerus. We drew lines at the deltoid insertion of the arm onto the limbs with indelible ink markers so that repeated measurements were reproducible over the 4 day period.

While lying prone on a standard examination plinth with the measured arm hanging over the side, subjects lowered their arms into the plethysmograph slowly, receiving verbal cues concerning limb position until the area below the drawn line on the arm submerged. The subject was able to support the limb by allowing the webspace between the third and the fourth digits to rest on a dowel crossing the plethysmograph transversely at its bottom. We measured the limbs repeatedly each session until we obtained three consecutive measures with less than a 20-ml difference.

We obtained circumference measures using three measurement tapes placed at designated sites. Designated measurement sites included 4 cm below and 4 and 8 cm above the cubital crease. Measurement involved three steps, with each step completed for all three sites before moving on to the next step, in distal to proximal order. The three steps included in the order performed were determining proper tape placement measuring from the cubital crease, applying correct tension, and recording values.

We measured maximal concentric elbow flexor force using the Lido 2 isokinetic dynamometer at the same angular velocity of 120°/sec used to induce soreness. We utilized concentric exercise for strength measurements at the different time periods to limit any additional injury that might arise from muscle contraction. Concentric muscle contractions relate to lower intramuscular pressure and muscle edema more than eccentric contractions do (Friden *et al.*, 1986; Staff 1988). We positioned subjects for testing in the same sitting position used for the exercise protocol. The test consisted of three to four practice repetitions followed by five recorded maximal repetitions. For analysis, we used the average of the five peak torque values recorded.

To ensure reliability and consistency of measurement, only one investigator obtained the same repeated measures on each of the dependent measures. Intrarater reliability for each of the dependent measures obtained in a pilot study was greater than .98.

### Analysis

We used a one within, one between analysis of variance with repeated measures to delineate effects of treatment and chronology for each dependent variable. This statistical design permits simultaneous tests for differences between the compressed and the uncompressed limbs (the Group effect), differences over time independent of between-subjects effects (the Time effect), and differences between groups at different data points (the Interaction effect). Post hoc analysis of a significant interaction effect will take precedence over significant time and/or group effects using a Newman-Keuls test ( $p < 0.05$ ). Prior to this analysis, we analyzed descriptive data for each variable, assessing for assumption violations for completing the ANOVA.

To assess the relationship between measures of volume and circumference over time, we used a canonical correlation procedure. This descriptive analysis permits assessment of the strength of relationship between two sets of variables. In this study, the five volume measures were the one set of variables that we used to correlate with three different sets of the circumference measures representing the different locations on the biceps. This analysis will determine the degree to which volume and circumference measures were redundant, as we expected.

## RESULTS

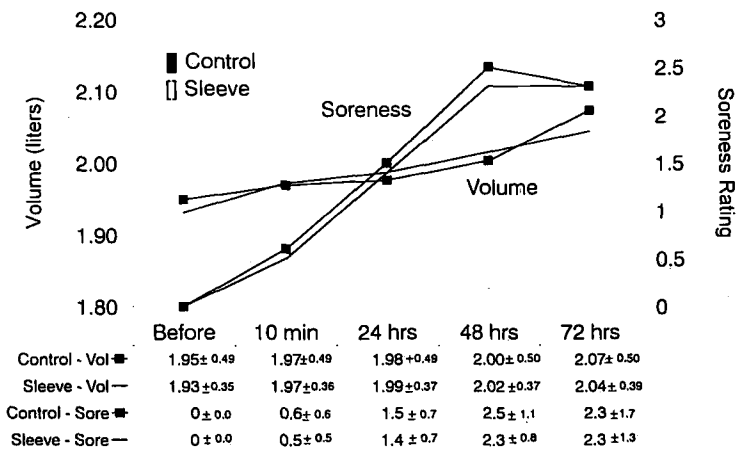
Figures 1-4 show the time graphs for the swelling measures, volume, and three arm circumference measures. Results of the ANOVA for volume revealed no significant group effect ( $F < .00$ ,  $p = .9$ , power = .04) and no significant group  $\times$  time interaction effect ( $F = .89$ ,  $p = .4$ , power = .27), but a significant time effect ( $F = 20.3$ ,  $p < .001$ , power  $> 1.0$ ). Obtained power for each of the tests mirrored their significance levels. Volume significantly increased by 28 ml by 24 hr in the control group and 57 ml in the experimental group (Fig. 1). The volume continued

to increase at nearly the same rate of 30 ml/day throughout the time frame of study. The volume by 72 hr had increased approximately 6%, to 2073 ml. This same pattern of findings held for the three circumference measures shown in Figs. 2-4. For the distal circumference variable, the group and interaction effects were not significant ( $F = .01, p = .9, \text{power} = .04$ , and  $F = 2.09, p = .09, \text{power} = .59$ , respectively) and the time effect was significant ( $F = 5.34, p = .001, \text{power} = .96$ ). For the middle circumference variable, the group and interaction effects were not significant ( $F = .45, p = .5, \text{power} = .10$ , and  $F = .65, p = .6, \text{power} = .21$ ) while the time effect was significant ( $F = 12.5, p < .001, \text{power} > 1.0$ ). Finally, group and interaction effects for the distal circumference variable were not significant ( $F = .07, p = .79, \text{power} = .05$  and  $F = .91, p = .4, \text{power} = .276$ ), but the time effect again was significant ( $F = 18.9, p < .001, \text{power} > 1.0$ ). The redundancy of volume and circumference measures is evident in Table I, which reveals that volume measures over time closely mirror each of the circumference measures over time.

Results of the analysis of soreness (Fig. 1), range of motion, and strength measures (Fig. 5) followed the same pattern as the swelling variables. For the soreness

**Table I.** Canonical Correlations Between Volume and Circumference Measures

	Distal biceps	Midbiceps	Proximal biceps
Volume	0.97	0.95	0.95



**Fig. 1.** Soreness ratings and volume (liters ± SD) of the forearm flexor muscles of the control group and the experimental group (sleeve) who wore the elastic compression stocking. Soreness ratings were based on a scale of 1 = normal to 6 = unbearable soreness. Measures were taken before eccentric exercise of the forearm flexors (before) and 10 min and 24, 48, and 72 hr after eccentric exercise.

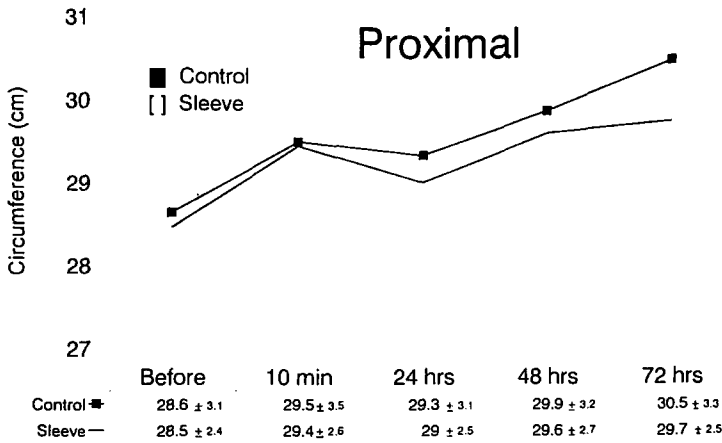


Fig. 2. Changes in proximal circumference (cm ± SD) of the elbow flexors of the control and experimental group before and 10 min and 24, 48, and 72 hr after eccentric exercise.

variable, the group and interaction effects were not significant ( $F = .12, p = .7$ , power = .05 and  $F = .09, p = .9$ , power = .06, respectively), while the effect due to time was significant ( $F = 39.9, p < .001$ , power > 1.0). By 24 hr, both groups' soreness measures had increased at least 140% over baseline, and by 72 hr, the increase was identically 230% over baseline.

The analysis of the range-of-motion variable revealed that the group and interaction factors were not significant ( $F = .04, p = .8$ , power = .05 and  $F = .61, p = .6$ , power = .2, respectively). The effect due to time was significant, however ( $F = 23.2, p < .001$ , power > 1.0). Range of motion in both groups decreased over 350% (from -4.9 to -18.7 for the control group and from -4.6 to -22.0 for

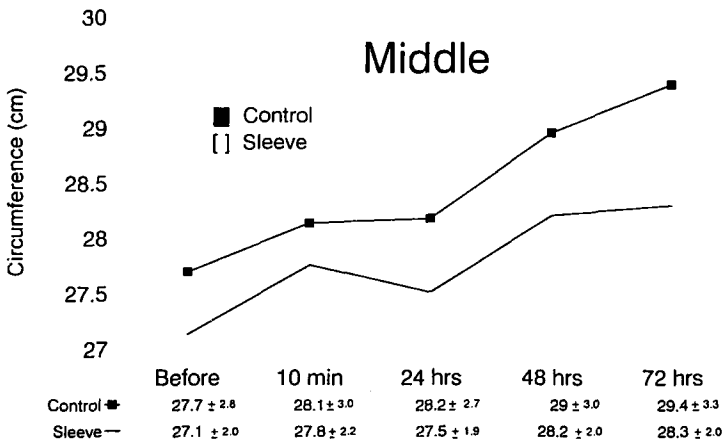


Fig. 3. Changes in middle circumference (cm ± SD) of the elbow flexors of the control and experimental group before and 10 min and 24, 48, and 72 hr after eccentric exercise.



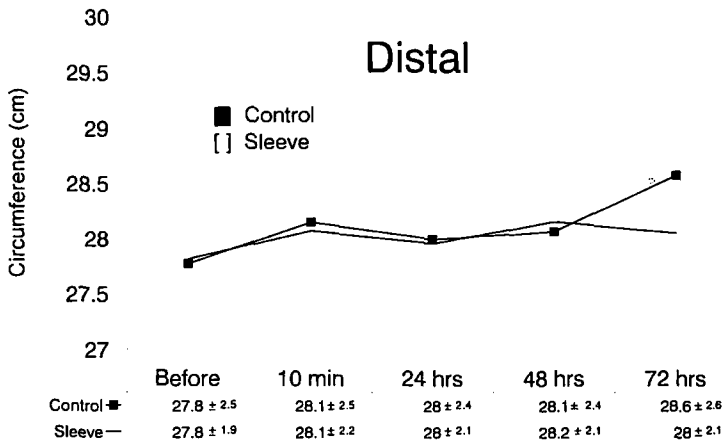


Fig. 4. Changes in distal circumference (cm ± SD) of the elbow flexors of the control and experimental group before and 10 min and 24, 48, and 72 hr after eccentric exercise.

the sleeve group) immediately following exercise and continuing throughout the 72-hr post exercise period (Fig. 5). By 72 hr, the control group had decreased 522% and the experimental group had decreased 480%.

Figure 5 also shows that the strength changes paralleled the changes in range of motion. Effects due to group and effects due to the interaction of group and time both were not significant ( $F = .22, p = .6, \text{power} = .06$ , and  $F = .69, p = .6, \text{power} = .2$ , respectively), while the time effect was again significant ( $F = 7.72, p < .001, \text{power} = .99$ ).

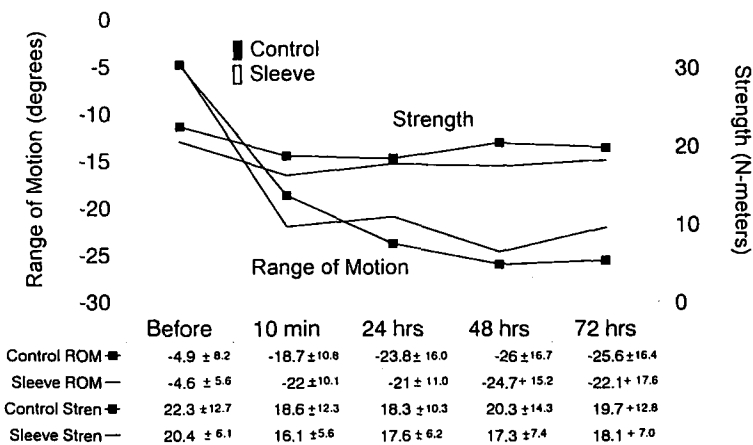


Fig. 5. Changes in peak concentric torque of the forearm flexors (expressed as Newton-meters) and relaxed angle (ROM) (± SD) of the control and experimental group before and 10 min and 24, 48, and 72 hr after eccentric exercise. ROM was determined as the relaxed angle in degrees (elbow angle when arm is resting at side).

## DISCUSSION

Research studies have suggested different mechanisms associated with the soreness perception of DOMS (Armstrong, 1984; Bobbert *et al.*, 1986; Hasson *et al.*, 1993). Because active inflammatory cells, such as white blood cells and neutrophils, are not always present with signs and symptoms of DOMS (Schwane *et al.*, 1983), other factors likely contribute to the soreness perception (Friden *et al.*, 1986; Hasson *et al.*, 1993; Newham, 1988; Stauber *et al.*, 1990).

Friden *et al.* (1988) suggested that increased tissue pressure from tissue swelling may be one of the critical factors associated with the development of DOMS. Armstrong (1984) proposed that sensory neurons that terminate in the muscle connective tissue between myofibers are sensitive to increased osmotic pressure and may carry the sensation of pain associated with swelling. For example, there exists an association between raised intramuscular pressures and pain in extreme conditions, such as "anterior compartment syndrome," in which the intramuscular pressures are sufficiently high enough to occlude circulation and cause ischemic pain (Mubarak and Hargens, 1983). If there is indeed an association between tissue swelling and soreness, then one could hypothesize that the application of a procedure, such as compression, that is effective as a deterrent to swelling in other acute care musculoskeletal injuries, would be effective in reducing swelling, soreness, and losses in strength and range of motion observed with DOMS (Matsen and Krugmire, 1974; Wilkerson, 1985). However, the results of this study consistently fail to support this hypothesis. The sequential course of the clinical manifestations of DOMS appears to follow the previously established pattern (Cleak and Eston, 1992; Friden *et al.*, 1986; Hoheisel and Mense, 1989) despite the application of external compression by an elastic stocking.

For all variables, the significant effect due to time indicates that each variable increased or decreased significantly from baseline independent of any effects due to group membership (i.e., compression versus no compression). That no significant differences were present for either the group or the interaction effects indicates that the observed change over time for all variables was independent of whether or not we used compression. The low obtained power for the group effect for each variable suggests that the study sample was not sufficiently large to detect a difference between groups. Thus, a reasonable question is if the experimental manipulation produced an effect sufficiently large to be of substantive interest despite the nonsignificant findings. As an indicator of effect size, we calculated eta squared statistics. [ $\eta^2$  squared is essentially a correlation ratio contrasting the variability of, in this case, the effect due to the experimental manipulation to the total experimental variability (Tabachnick and Fidell, 1989). Thus,  $\eta^2$  gives the percentage of experimental variance accounted for by the effect of interest.] For all variables,  $\eta^2$  for the group effect was no larger than .02 (2% of the total experimental variance), this effect involving the middle circumference variable, and was as small as .3% for the proximal circumference variable. The only other variable for which the experimental manipulation had an effect larger than 1% was the strength variable. Clearly, a study that would demonstrate significant differences between compressed and uncompressed arms following exercise would have to have such an enormous

number of subjects that it would be prohibitively expensive and of little clinical utility.

We can now ask the question, "Why was the compression ineffective?" Were there methodological problems, was the applied pressure not great enough to prevent the edema, or is it feasible that edema induced pressure does not relate to, but rather coexists with, DOMS?

One possible explanation is that the person taking the measurements was not blind to the subject's treatment condition. Although blinding the person taking the measurement would have minimized any conscience or unconscience bias in the measurements, blinding was not feasible due to the possibility of additional edema forming upon removal of the sleeve. To minimize that possibility, we removed sleeves immediately before and reapplied them immediately after measurement. If such a bias existed, it would most likely favor the sleeve group. However, the data analysis did not support this possibility. Thus, any bias, if it existed, had to be minimal.

Optimal compression pressure for elastic stockings reported to produce the greatest increase in venous flow velocity consistent with safety and practicality of hospital use averaged between 8 and 18 mm Hg (Sigel *et al.*, 1975). Husni *et al.* (1968) noted that inflation of air splints beyond 20 mm Hg compressed deep veins and elevated resting venous pressure. Ashton (1962) reported that air splints inflated to 30 mm Hg reduced blood flow by more than 50%. This occurred in a pilot study, conducted prior to the present study, to determine the optimal number of thickness of compression sleeve to use. Three thicknesses (producing pressures in excess of 33 mm Hg) consistently resulted in finger edema and were unsafe to wear for an extended period of time. The average 17 mm Hg pressure applied by the compression sleeve used in the present study was within the optimal range and should have been of sufficient pressure to reduce swelling without collapsing the deep veins (Ashton, 1962; Husni *et al.*, 1968; Matsen and Krugmire, 1974). However, because swelling increased in both the treated and the control arms without difference, the level of compression pressure was insufficient to prevent edema. Higher pressures may reduce edema and pain but risk decreased blood flow and additional tissue damage.

Although appreciable damage can result from the mechanical strain of severe eccentric contraction, the time course between damage and pain is controversial. For example, the release of intracellular components from the damaged tissue into the interstitium, including prostaglandins, attracts neutrophils and monocytes, initiating an inflammatory response. The inflammatory response begins as rapidly as a few hours after tissue injury, before muscles became painful (Armstrong *et al.*, 1980). Clarkson and Tremblay (1988) noted that pain sensation is maximal 48 hr after a bout of eccentric exercise but the resting muscle length decreases immediately postexercise. Friden *et al.* (1986) also identified an increase in pressure and volume in the anterior compartment of the lower limb immediately following eccentric contractions before muscles were painful. During the painful period, the pressures remained elevated, but not more than in the pain-free period immediately after exercise. In a similar study using elbow flexors, Newham and Jones (1985) were unable to detect any differences in intramuscular pressure of the elbow flexors between control and eccentric exercised muscles. Therefore, the results of these studies and the present study indicate that eccentric contractions may cause raised intramuscular pressures but the time

courses of the resultant pressures are not similar to those of the pain and thus suggest that pressure is not the major algescic stimulus in DOMS.

## REFERENCES

- Armstrong, R. B. (1984). Mechanisms of exercise-induced delayed onset muscular soreness: A brief review. *Med. Sci. Sports Exerc.* 16: 529-538.
- Armstrong, R. B., Garshnek, V., and Schwane, J. (1980). Muscle inflammation: Response to eccentric exercise. *Med. Sci. Sports* 12: 94-102.
- Ashton, H. (1962). Critical closing pressure in human peripheral vascular beds. *Clin. Sci.* 22: 79-83.
- Blanave, C., and Thompson, M. (1993). Effect of training on eccentric exercise-induced muscle damage. *J Appl Physiol.* 75: 1545-1551.
- Bobbert, M. F., Hollander, A. P., and Huijing, P. A. (1986). Factors in delayed onset soreness of man. *Med. Sci. Sports Exerc.* 18: 75-78.
- Clarkson, P. M., and Tremblay, I. (1988). Exercise-induced muscle damage, repair, and adaptation in humans. *J. Appl. Physiol.* 65: 1-6.
- Cleak, M. J., and Eston, R.G. (1992). Muscle soreness, swelling, stiffness and strength loss after intense eccentric exercise. *Br. J. Sports Med.* 26: 267-272.
- Cohen, J. (1977). *Statistical Power Analysis for the Behavioral Sciences*, Academic Press, New York, pp. 14-15.
- Donnelly, A. E., McCormick, K., and Maughan, R. J. (1988). Effects of a non-steroidal anti-inflammatory drug on delayed-onset muscle soreness and indices of damage. *Br. J. Sports Med.* 22: 35-38.
- Donnelly, A. E., Maughan, R. J., and Whiting, P. H. (1990). Effects of ibuprofen on exercise-induced muscle soreness and indices of muscle damage. *Br. J. Sports Med.* 24: 191-195.
- Edwards, R. H. T., Mills, K. R., and Newham, D. J. (1981). Measurement of severity and distribution of experimental muscle tenderness. *J. Physiol.* 317: 1P-2P.
- Fitzgerald, G. K., Rothstein, J. M., and Mayhew, T. P. (1991). Exercise-induced muscle soreness after concentric and eccentric isokinetic contractions. *Phys. Ther.* 71: 505-513.
- Francis, K. T., and Hoobler, T. (1987). Effects of aspirin on delayed muscle soreness. *J. Sports Med.* 27: 333-337.
- Francis, K. T., and Hoobler, T. (1988). Delayed onset muscle soreness and decreased isokinetic strength. *J. Appl. Sport Sci. Res.* 2: 20-23.
- Friden, J., Kjorell, U., and Thornell, L. E. (1983a). Delayed muscle soreness and cytoskeletal alterations: An immunocytochemical study in man. *Int. J. Sports Med.* 4: 177-184.
- Friden, J., Sjostrom, M., and Ekblom, B. (1983b). Myofibrillar damage following intense eccentric exercise in man. *Int. J. Sports Med.* 4: 170-176.
- Friden, J., Sfakianos, P. N., and Hargens, A. R. (1986). Muscle soreness and intramuscular fluid pressure: Comparison between eccentric and concentric load. *J. Appl. Physiol.* 61: 2175-2179.
- Friden, J., Sfakianos, P. N., Hargens, A.R., and Akeson, W. (1988). Residual muscular swelling after repetitive eccentric contractions. *J. Orthop. Res.* 6: 493-498.
- Hasson, S. M., Barnes, W., Hunter, M., and Williams, J. (1989). Therapeutic effect of high speed voluntary muscle contractions on muscle soreness and muscle performance. *J Orthop Sports Phys Ther.* 10: 499-507.
- Hasson, S. M., Daniels, J. C., and Divine, J. G. (1993). Effect of ibuprofen use on muscle soreness, damage, and performance: A preliminary investigation. *Med. Sci. Sports Exerc.* 25: 9-17.
- Hoheisel, U., and Mense, S. (1989). Long-term changes in discharge behavior of cat dorsal horn neurons following noxious stimulation of deep tissues. *Pain.* 36: 239-247.
- Hough, T. (1902). Ergographic studies in muscular soreness. *Am. J. Phys.* 7: 76-92.
- Husni, E., Simenes, J., and Hamilton, F. (1968). Pressure bandaging of the lower extremity: Use and abuse. *JAMA* 206: 2715-2719.
- Kuipers, H., Keizer, H., and Verstappen, F. T. J. (1985). Influence of a prostaglandin inhibiting drug on muscle soreness after eccentric work. *Int. J. Sports Med.* 6: 336-339.
- Lewis, C. E., Antione, J., Mueller C., Talbot, W., Swaroop, R., and Edwards, W. (1976). Elastic compression in the prevention of venous stasis. *Am. J Surg.* 132: 739-743.
- Matsen, F. A., and Krugmire, R. B. (1974). The effect of externally applied pressure on post-fracture swelling. *J. Bone Int. Surg.* 56-A: 1586-1591.
- Mubarak, S. J., and Hargens, A. R. (1983). Acute compartment syndromes. *Surg. Clin. N. Am.* 63: 539-565.

- Newham D. J. (1988). The consequences of eccentric contractions and their relationship to delayed onset muscle pain. *Eur. J. Appl. Physiol. Occup Physiol.* 57: 353-359.
- Newham, D. J., and Jones D. A. (1985). Intra-muscular pressure in the painful human biceps. *Clin. Sci.* 69: 27p.
- Newham, D. J., McPhail, G., and Mills, K. R. (1983). Ultrastructural changes after concentric and eccentric contractions of human muscle. *J. Neurol. Sci.* 61: 109-122.
- Sarin, S., Scurr, J. H., and Coleridge-Smith, P. D. (1992). Mechanism of action of external compression on venous function. *Br. J. Surg.* 79: 499-502.
- Schwane, J. A., Johnson, S. R., Vandenakker, C. B., and Armstrong, R. B. (1983). Delayed-onset muscular soreness and plasma CPK and LDH activities after downhill running. *Med. Sci. Sports Exerc.* 15: 51-56.
- Sigel, B., Edelstein, A., Savitch, L., Hasty, J., and Felix, R. (1975) Type of compression for reducing venous stasis. *Arch. Surg.* 110: 171-175.
- Staff, P. (1988). Clinical consideration in referred muscle pain and tenderness: Connective tissue reactions. *Eur. J. Appl. Physiol.* 57: 369-372.
- Stauber, W. T., Clarkson, P. M., and Fritz, V. K. (1990). Extracellular matrix disruption and pain after eccentric muscle action. *J. Appl. Physiol.* 69: 868-874.
- Tabachnick, B., and Fidell, L. (1989). *Using Multivariate Statistics*, 2nd edition., Harper Collins, New York.
- Talag, T. S. (1973). Residual muscle soreness as influenced by concentric, eccentric, and static contractions. *Res. Q.* 44: 459-469.
- Wilkerson, G. B. (1985). External compression for controlling traumatic edema. *Phys. Sportsmed.* 13(6): 97-106.
- Yackzan, L., Adams, C., and Francis, K. T. (1984). The effects of ice massage on delayed muscle soreness. *Am. J. Sports Med.* 12: 159-165.