The Effects of Athletic Massage on Delayed Onset Muscle Soreness, Creatine Kinase, and Neutrophil Count: A Preliminary Report

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Delayed onset muscle soreness (DOMS) occurs in both trained and untrained individuals in response to unaccustomed exercise. The pain and stiffness associated with DOMS generally appears between 8 and 24 hours after exercise, peaks at around 48 hours, and dissipates over the course of a few days (2,15). This discomfort may vary from a slight stiffness to extreme debilitating pain and may occur in any exercised muscle (1). The location of the soreness is generally most prominent at the myotendinous junction where connective tissue is most abundant (18).

However, in severe cases, pain may be felt throughout the entire muscle belly (15,19).

A strong association exists between eccentric muscle action, which involves the lengthening of the muscle under tension, and the subsequent development of DOMS (2,16). In human and animal studies, it has been repeatedly verified that eccentric exercise causes disruption of contractile and/or connective tissue (18,34,40). Generally, acute inflammation occurs whenever muscle and associated connective tissue are injured (41). It has been proposed that DOMS is a form of acute inflamma-
tion activated in response to tissue injury and that the soreness sensations represent inflammatory pain (16,34,39).

Moderate to severe DOMS may interfere with athletic performance (22), most likely due to associated swelling (19), reduction in the range of motion of the involved joint (15,40) and reduction in force output (9,15). Delayed onset muscle soreness also interferes with daily living activities, impedes adherence to exercise programs, and may interfere with an individual's willingness to perform therapeutic exercise during rehabilitation (17). Consequently, a number of strategies have been employed in an attempt to reduce DOMS. Stretching before (25) and after exercise (3), as well as the application of ice (44), have not proven successful in reducing DOMS. However, the application of a topical ointment (26) and dexamethasone iontophoresis (24) appear to significantly reduce DOMS. A number of studies have examined the effectiveness of nonsteroidal, anti-inflammatory drugs (NSAIDS); Some report a significant reduction in DOMS (17,23) while others report no effect (12,13,31).

Sports massage is routinely recommended as an aid for speeding recovery from vigorous exercise (4,5,7,32); however, there is a limited amount of research to substantiate these claims. Several studies that have investigated the impact of massage on the reduction of DOMS have concluded that it is ineffective (14,36,43,44). In these studies, massage was administered either immediately after exercise or at 24 or 48 hours after exercise (36,43). In contrast, Soviet sports therapists have suggested that to enhance athletic performance, "restorative" massage should be administered between 1 and 3 hours after the termination of strenuous exercise (4,30,37). Although no scientific rationale has been provided to support this recommendation, it does suggest that for massage to be effective, the time period after exercise at which massage is rendered might be critical. We hypothesized that the reason for the effectiveness of massage at this time might be related to interference with the initiation of the acute inflammatory response.

An initial critical event in the inflammatory process that occurs within a few hours after an injury is the accumulation of the first wave of white blood cells, the neutrophils, at the injured site. The accumulation of the first wave of white blood cells, the neutrophils, at the injured site (33). The accumulation of tissue neutrophils is preceded by an elevation in circulating neutrophils due to accelerated release from the bone marrow (41). This is followed by a decline in circulating neutrophils as these cells marginate to the vessel walls in the area of injury and subsequently emigrate to the traumatized tissue (41). Subsequent expression of the inflammatory response, such as the accumulation of macrophages at the site of injury, is largely dependent on this initial accumulation of neutrophils (10,11). After eccentric exercise, in a similar fashion, increases and declines in circulating neutrophils have been reported at comparable times to what typically occurs during classic, acute inflammation (38). In addition, neutrophils have been seen at the site of muscle trauma (16,39).

Therefore, if DOMS is the result of the inflammatory process, migration and emigration of neutrophils must be considered a critical event. If massage given a few hours after eccentric exercise is effective, as is claimed by Krilov et al (30) and Paliok (37), it seems reasonable to hypothesize that this process disrupts margination and subsequent emigration of neutrophils into the area of injury. This would most likely manifest itself in a prolonged elevation of circulating neutrophils. Theoretically, the reduced emigration of neutrophils would reduce the intensity of the inflammatory event and reduce pain and discomfort associated with DOMS.

Therefore, the objectives of this study were to determine if massage rendered 2 hours after the termination of eccentric exercise would significantly reduce DOMS sensations as well as serum creatine kinase (CK) levels, an indirect marker of muscle tissue damage. Furthermore, if such an attenuation occurred, would this be associated with different levels of circulating neutrophils? In addition, we were interested in determining whether massage would result in increases in serum cortisol levels, since cortisol has potent anti-inflammatory properties (8).

**METHODS**

**Subjects**

Fourteen healthy, active, untrained Caucasian males volunteered for this study. Subjects completed an extensive medical history and signed an informed consent that had been approved by the university's Policy and Review Committee for Human Research. A computer-generated program was used to randomly assign...
Eccentric Exercise Protocol

All exercise was performed between 7 am and 9 am. Subjects exercised using the Kin-Com Muscle Testing System (500H, Chattanooga Corporation, Chattanooga, TN). The exercise consisted of eccentric, isokinetic contractions of the forearm flexors (biceps) and extensors (triceps) of the nondominant arm. Subjects warmed up by performing 20 submaximal concentric contractions at 120°/sec, through a range of approximately 90°, followed by five maximal concentric contractions; the highest value was recorded as maximum. Markers were placed on the screen at a level equivalent to 90% of the concentric maximum value. This setting represented about 75% of each subject’s maximal eccentric strength. Subjects then performed four to five sets of 35 eccentric muscle actions, attempting to maintain this force output by watching the display screen while performing the exercise. A 2-minute rest was allowed between sets. The number of sets performed depended on whether the subject was able to maintain the preset intensity for five complete sets.

Massage Therapy

Massage/placebo treatments were administered between 9:30 am and 11:30 am. At 2 hours following the exercise bout, a 30-minute sports massage was performed by a licensed physical therapist on the exercised arm of the massage subjects (N = 7). Subjects remained supine on a padded table for the duration of the massage. Each massage was replicated through recorded verbal cues on an audio cassette to control for the amount of time spent per stroke as well as to control for the depth of the massage. Lotion (Nivea cream, Beiersdorf, Inc., Norwalk, CT) was used to reduce friction between the therapist’s hands and the subject’s skin. The massage protocol was as follows: effleurage (stroking), hand to shoulder (2 minutes); shaking of arm (10 seconds); pettrissage (kneading) and effleurage of hand, with elbow supported on table (2 minutes); effleurage, wrist to elbow (1 minute); thumb petissage, wrist to elbow (3 minutes); wringing of forearm (2 minutes); cross-fiber massage to forearm (3 minutes); easy petissage of forearm (1 minute); effleurage, wrist to elbow (1 minute); shaking of arm (10–15 seconds); effleurage, elbow to shoulder (1 minute); thumb petissage, elbow to shoulder (3 minutes); wringing of upper arm (1 minute); cross-fiber massage to biceps and triceps (5 minutes each); easy petissage of elbow to shoulder (1 minute); effleurage, elbow to shoulder (1 minute); and shaking of arm (15 seconds).

The placebo treatment consisted of the application of lotion (Nivea) onto the involved arm at 2 hours following the exercise bout. Subjects were told that a comparison was being made between the effectiveness of a topical preparation and massage. The control subjects (N = 7) remained supine with the exercised arm immobile for one half-hour.

Muscle Soreness Ratings

Muscle soreness was assessed before exercise and at 8, 24, 48, 72, 96, and 120 hours following the bout of eccentric exercise. Subjects were shown a soreness scale (Clarkson Scale: 1 = no soreness, 10 = unbearable soreness), and written instructions were read to them. Briefly, they were requested to bend, extend, and palpate their exercised arm, and assign a number between 1 and 10 that best represented their overall rating of soreness.

Blood Analyses

On the day of exercise, an intravenous catheter (22 gauge, 1 in, Johnson and Johnson, Tampa, FL) was placed in an antecubital vein of the nonexercised arm. Heparin was used to maintain patency. The subject remained seated quietly for 20 minutes prior to drawing the preexercise sample. Additional samples were drawn immediately after exercise and then every 30 minutes for 8 hours (18 samples). A venipuncture was performed on follow-up visits at 24, 48, 72, 96, and 120 hours.

To assess CK, blood samples were collected in nontreated vacutainers before exercise and at 24, 48, 72, 96, and 120 hours after exercise. The blood (3 ml) was allowed to clot at room temperature for 10 minutes and then centrifuged for 15 minutes. Serum was separated and frozen at −20°C for subsequent analysis. Total CK was determined spectrophotometrically (in duplicate for each sample) at 25°C using a commercially available kit (CK 10, Sigma Diagnostics, St. Louis, MO). If duplicate values were not within 5% of each other, a third sample was run and the two closest values were recorded.

To determine the neutrophil count before exercise and during the 8 hours following exercise, blood was collected in a 3-ml vacutainer treated with 4.5 mg ethylenediaminetetraacetic acid (EDTA) and
gently inverted 11 times. The total and differential white blood cell counts were assessed by electronic impulse using preferential cellular staining on a Technicon H6000 (Technicon, Tarrytown, NY); duplicate slides were made as an aid to assess the differential count. As a reliability check, one “blind” duplicate sample per subject was evaluated; all duplicates were within 100 cells/mm³ for neutrophils.

Serum samples from six massage subjects and three randomly selected control subjects were used for analysis of cortisol. All samples had been frozen at −80°C for no longer than 60 days. The following samples were analyzed: before exercise (0 hours), before massage/placebo treatment (2.0 hours after exercise), immediately after massage (2.5 hours), and then at hours 3.0, 4.0, 5.0, 6.0, 7.0, and 8.0 after exercise. A solid phase radioimmunoassay kit (Clinical Assays/Instar Corp., Stillwater, MN) was used. All samples and standards were run in duplicate.

**Statistical Analysis**

The dependent variables—muscle soreness, CK, neutrophils, and cortisol—were analyzed using a standard, split-unit, repeated-measures analysis of variance (ANOVA) with a trend analysis (27). A trend analysis was used because we were interested in whether the response pattern, the profile shapes, differed significantly for the massage and control groups over time. This analysis provides informative insights that are not accessible through a traditional, repeated-measures design (27). The level of significance was set at \( p < 0.05 \). All values reported are means and standard errors.

**RESULTS**

To determine whether there were any significant preexercise differences between the massage and control groups, \( t \)-tests were run. No preexercise significant differences were found between any of the dependent variables.

**Muscle Soreness Ratings**

When the mean soreness ratings for both groups were collapsed across time, there was a significant increase in soreness (\( p = 0.0001 \)) demonstrating that the exercise protocol was sufficiently strenuous to induce DOMS. The trend analysis revealed a significant interaction of treatment with the quadratic effect of time (\( p = 0.0477 \)). This was due to the control group having consistently higher DOMS ratings compared with the massage group from 24 through 96 hours. The control subjects reported a peak DOMS value of 5.9 ± 0.5 at 48 hours while the massage subjects reported a peak soreness value of 4.6 ± 0.7 at 24 hours (Figure 1).

**Creatine Kinase**

There was a significant time effect (\( p = 0.001 \)) for CK when the means for both groups were collapsed across time; this verified the presence of muscle trauma. A trend analysis revealed a significant interaction between treatment and the linear time trend exhibited by CK (\( p = 0.0430 \)). Although both groups showed a linear increase in CK across time, the control group showed an earlier and sharper upward slope. Creatine kinase for control subjects before exercise was 83.8 ± 9.0 U/L at 24 and 48 hours it increased to 240.4 ± 152.8 U/L and 648.4 ± 494.8 U/L, respectively; and it peaked at 96 hours at 1,917 ± 1,023 U/L. The preexercise value for massage subjects was 104.7 ± 20.5 U/L. No increases were seen in CK until 72 hours, and then levels steadily increased, peaking at 1,083 ± 476 U/L at 120 hours. Whether this was the true peak is not clear since blood samples were only drawn up to this time (Figure 2).

**Neutrophil Count**

Because of procedural difficulties, data from 12 (six subjects per group) instead of 14 subjects were used for the total and differential white blood cell count. Only the results of the neutrophil count will be reported since this was of primary interest. A trend analysis for treatment by time revealed a significant difference in linear trends between groups (\( p = 0.0324 \)). The control group displayed a negative slope compared with the slightly positive trend displayed by the massage group (Figure 3). Neutrophil values for the control group before exercise and at 8 and 24 hours after exercise were 4,066 ± 626, 4,250 ± 351, and 4,267 ± 649 cells/mm³; thus, at 8 and 24 hours neutrophil count was...
There were several reasons for selecting the 2-hour postexercise time to administer massage. Krilov et al. (30) reported that for better relaxation and faster restoration of the athlete’s body, massage should be given after 2 to 3 hours of rest following exercise. Paiko (37) suggested that restorative massage should take place 1–2 hours after exercise. In addition, the assistant executive director of the United States Cycling Federation reported that competitive cyclists typically receive massage therapy approximately 2 hours after an exercise bout (Mark Hodges, personal communication, 1990).

In the present study, additional rationale for the 2-hour time delay before administering massage was related to neutrophil activity associated with the onset of acute inflammation. The initial accumulation of neutrophils occurs within a few hours after the onset of tissue injury and appears to be crucial to the subsequent manifestation of inflammatory events (10,11,33).

It was hypothesized that sports massage might have an impact on neutrophil accumulation in the following manner: During acute inflammation, blood flow slows as vessels dilate in an area of injury (21). When this occurs, the white blood cells, including neutrophils, are displaced from the central, axial zone of blood flow to the peripheral, plasmatic zone and subsequently marginate along the vessel walls. Since vigorous massage appears to increase blood flow through the vascular bed (5,29), we theorized that this increased flow rate in the area of microtrauma could prevent the typical outward displacement of neutrophils. In addition, we speculated that the mechanical action of vigorous massage (5) could shear marginated cells from vessel walls (21) and thus hinder emigration of cells from the circulation into tissue spaces. We further assumed that interference with this typical neutrophil sequence...
might be reflected in an additional and/or prolonged increase in circulating neutrophils (20). A significant difference in linear trends of the neutrophil response was seen between the massage and the control groups. Over the 8-hour period following exercise, control subjects showed a linear decrease in circulating cells while massage subjects showed a slight increase. Furthermore, at 8 and 24 hours after exercise, neutrophils for massage subjects were approximately 15% above baseline while neutrophils for control subjects at these same times were approximately 4% above baseline. This appears to support the proposed hypothesis that if neutrophils are unable to migrate from the circulation into the tissue spaces, there would be an elevation in the blood count.

An increase of CK in the circulation after unaccustomed eccentric exercise has become acceptable, indirect evidence of muscle injury (2,15, 28). The significant increase in serum CK seen in this study for both groups after exercise is similar to previous reports (2,15,28). Of greater interest is the significant difference seen in the pattern of change between the two groups over the course of 5 days, with the control group exhibiting a more rapid and steeper increase in CK values than the massage group (Figure 2). These differences in CK could be related to differences in neutrophil activity at the site of injury. Cannon et al (6) have suggested that mobilization and activation of neutrophils at the site of injury may contribute to increased CK efflux after eccentric exercise. A major function of the neutrophil is phagocytosis (cleaning up of "debris" caused by injury). This is achieved through the release of a vast array of catabolic enzymes that may cause further damage to the muscle before healing is initiated (42). In the present study, if sports massage did interfere with accumulation of neutrophils, then Cannon et al's theory may help account for the reduced serum CK levels seen in the massage group.

Serum cortisol was measured in a further attempt to clarify how massage might have reduced DOMS. Glucocorticoids have profound anti-inflammatory and analgesic properties (8). One anti-inflammatory mechanism of glucocorticoids is that they inhibit neutrophil adherence to vessel walls at the site of injury and thus interfere with the emigration of neutrophils to the injured area (35). Generally, cortisol displays a dramatic diurnal variation, with peak levels seen in the early morning and a gradual decrease throughout the day, with lowest values seen in the evening. In the present study, during the 8-hour, postexercise period, the massage group showed less reduction in cortisol levels when compared with the control group (Figure 4). The reason for higher cortisol levels in the massage group compared with the control group could have been due to the vigorous sports massage being "interpreted" by the body as a form of stress. This could have activated the hypothalamic-pituitary-adrenal axis and resulted in increased release of cortisol into the circulation (8).

**SUMMARY**

It is proposed that vigorous sports massage rendered 2 hours after termination of unaccustomed eccentric exercise reduces the intensity of delayed onset muscle soreness and reduces serum creatine kinase levels. This may be due to interference with neutrophil activity ascribed to the mechanical action of vigorous massage and/or higher levels of serum cortisol. It is recommended that this area of research be further pursued using a larger sample size, a larger muscle mass, and experimenting with different postexercise times for administering massage.

**REFERENCES**


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