# Electro-membrane microcurrent therapy reduces signs and symptoms of muscle damage

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**ABSTRACT**

LAMBERT, M. I., P. MARCUS, T. BURGESS, and T. D. NOAKES. Electro-membrane microcurrent therapy reduces signs and symptoms of muscle damage. *Med. Sci. Sports Exerc.*, Vol. 34, No. 4, pp. 602–607, 2002. **Purpose:** Delayed onset muscle soreness (DOMS) occurs after unaccustomed physical activity or competitive sport, resulting in stiff, painful muscles with impaired function. Acustat® electro-membrane microcurrent therapy has been used to treat postoperative pain and soft tissue injury; however, its efficacy in reducing symptoms of muscle damage is not known. **Methods:** Thirty healthy men were recruited for a double-blind, placebo-controlled trial. The muscles of their nondominant arms were damaged using an eccentric-exercise protocol. Subjects were then randomly assigned to treatment with either Acustat or a matching placebo membrane for 96 h and monitored for a total of 168 h. **Results:** Subjects in both groups experienced severe pain and swelling of the elbow flexors after the eccentric exercise. After 24 h, the elbow joint angle of the placebo group had increased significantly more than those in the Acustat group (13.7 ± 8.9° vs 7.5 ± 5.5°; placebo vs Acustat, $P < 0.05$), possibly as a consequence of the elbow flexor muscles shortening. For the first 48 h after exercise, maximum voluntary contraction of the elbow flexor muscles was significantly impaired in the placebo group by up to 25% ($P < 0.05$), whereas muscle function was unchanged in the Acustat group. Peak plasma creatine kinase activity was also lower in the Acustat group (peak = 777 ± 1438 $U \cdot L^{-1}$) versus the placebo group (peak = 1918 ± 2067 $U \cdot L^{-1}$; ($P < 0.05$). The membranes were well tolerated by the subjects in both groups without any adverse effects. **Conclusion:** These data show that treatment of muscle damage with Acustat electro-membrane microcurrent therapy reduces the severity of the symptoms. The mechanisms of action are unknown but are likely related to maintenance of intracellular $Ca^{2+}$ homeostasis after muscle damaging exercise. **Key Words:** SKELETAL MUSCLE, PAIN, MICROCURRENT THERAPY, MAXIMUM VOLUNTARY CONTRACTION, CREATINE KINASE ACTIVITY

Delayed onset muscle soreness (DOMS) frequently occurs after recreational physical activity or competitive sport (8), causing stiff, painful muscles with reduced contractile function (20,21). Many treatment modalities of DOMS, including stretching (6), massage (26), ice therapy (27), oral nonsteroidal anti-inflammatory agents (4,11) and transdermal nonsteroidal anti-inflammatory agents (22), have been studied. None has proved to be consistently effective in reducing the severity of DOMS (9).

Recently, the evidence supporting electrical stimulation as a treatment modality for wound healing and soft tissue injury has been evaluated (13). Although the authors concluded that electrical stimulation can effect wound healing, the mechanism is unclear. Over the years, the principle of electrical stimulation for analgesia and tissue and wound healing has been adapted into therapies such as electroacupuncture, transcutaneous electrical nerve stimulation therapy (10), and, more recently, microcurrent therapy. The last approach involves positioning an electrostatically charged membrane over the skin of the damaged area. There has been little systematic evaluation of microcurrent therapy, although there is anecdotal evidence of efficacy and safety. Acustat® (TC Corporation, Tustin, CA) is a commercially available membrane that provides microcurrent stimulation. It comprises a nonwoven, nonallergenic, self-adhesive, microporous polymer membrane measuring approximately 8.5 cm by 15 cm. The polymer is an electret that stores a strong negative electrostatic charge, produced during manufacture. In contact with the skin, it discharges over a 48-h period, inducing a flow of electrons into the skin and subcutaneous tissues. The total current flow over this period has been measured at 20 microamps. The patient feels no sensation during treatment. Electret membranes have been shown to reduce posttraumatic tissue inflammation (24).

A randomized clinical trial of the use of microcurrent therapy after muscle damage has not been undertaken. Accordingly, the aim of this study was to measure the effectiveness of treatment with Acustat in reducing the severity of pain, inflammation, and loss of function arising from muscle damage.

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## MATERIALS AND METHODS

**Study design.** The study had a double-blind, placebo-controlled design and used healthy male volunteers. Elbow

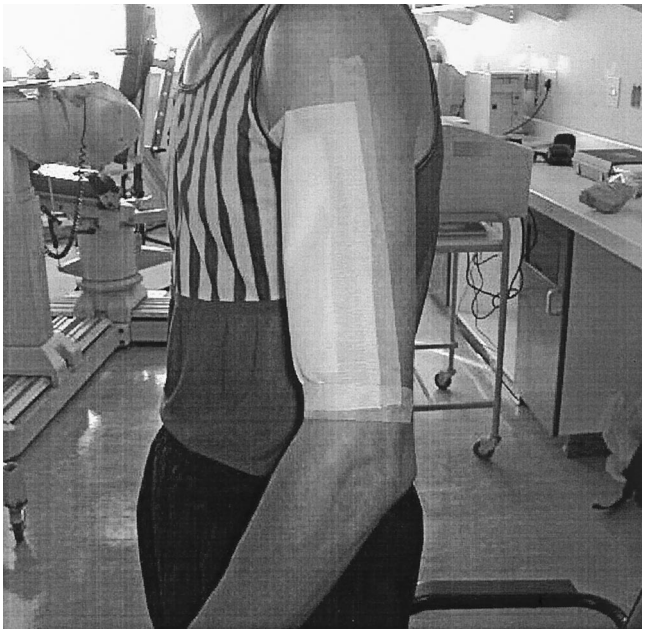

**FIGURE 1—The placement of the Acustat membrane.**

flexor muscle function of both arms was measured before the subjects' nondominant arms were exposed to an eccentric-exercise protocol that caused muscle damage. Thereafter, painful muscles were treated with either Acustat or an indistinguishable placebo for 96 h. The treatment or placebo membrane was applied only to the exercised arm. The dominant nonexercised arm acted as a control. To standardize the placement of the membranes, the subjects stood erect with their arms hanging by their sides. The self-adhesive Acustat membrane was placed using the upper most lateral head of the radius as a bony landmark. The end of the membrane was positioned at that point and the length of the membrane was positioned proximally over the belly of the biceps muscle (Fig. 1). The membrane was applied with slight pressure to ensure close contact with the skin, after which it was held in place with a bandage over the biceps muscle to ensure that contact was maintained during any joint movement. The membrane was left in position for 48 h. It was then replaced with another new membrane as described above for another 48 h. The Acustat membrane was kept dry. It was removed when necessary for bathing and then reapplied.

The damaged and nondamaged elbow flexor muscles in both groups were studied intermittently for up to 168 h after exercise for muscle pain, function, inflammation, and passive shortening. Serum creatine kinase activity was measured in the blood of the subjects during this period. This research model was designed to recreate the clinical circumstances of an acute soft tissue injury leading to inflammation. A schematic diagram of the research design is shown in Figure 2.

The study was granted ethical clearance by the Ethics and Research Committee of the Faculty of Health Sciences, University of Cape Town Medical School. Subjects gave written consent after being informed about the demands of the study.

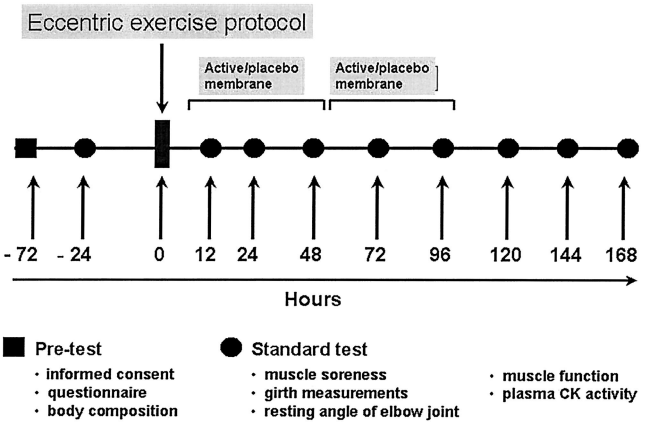


**FIGURE 2—Study design. Active/placebo membrane represents the duration of the first (48 h) and second (48 h) Acustat or placebo membrane.**

**Active and placebo Acustat.** The active and placebo Acustat membranes were indistinguishable. The active membrane was electrostatically charged during manufacture. The placebo membrane designed specifically for the study had no electrical charge and therefore did not provide any microcurrent therapy. As the study was double blinded, the active and placebo Acustat membranes were coded. An independent auditor decoded the randomization on completion of the study, after the data had been analyzed.

**Subjects.** Thirty men, without known elbow joint disease, were recruited for the study. The subjects completed a questionnaire to determine their age, training history, medical and surgical history, and any past or present injuries to the upper limbs. Subjects gave their informed consent to participate in the study after the nature, purpose, and testing procedures were thoroughly explained. The subjects had not received eccentric training of the arms for the 12 wk before the study started. All subjects had their body composition assessed according to procedures previously described (23).

The subjects were assigned to either the placebo or experimental groups. The first subject was randomly allocated (by flipping a coin). The second subject in that pair was assigned to the other group. This process was repeated for the 15 pairs of subjects.

The subjects were requested to avoid any other form of medication, including anti-inflammatory agents, as well as alcohol for 2 d before testing and for the duration of the study. In addition, subjects were asked to refrain from any strenuous physical activity for the duration of the study. Subjects were questioned on any adverse events associated with the treatment each day when they visited the laboratory.

**Eccentric exercise to induce muscle damage and pain.** Muscle damage was induced in the nondominant elbow flexor muscles by a series of eccentric contractions on a Kin-Com isokinetic dynamometer (Chattanooga Group, Inc., Chattanooga, TN). The protocol consisted of 25 repetitions of 5 sets of eccentric contractions at 80% of the subject's maximum eccentric force. The membrane (either Acustat or placebo) was applied immediately after the exercise protocol.

**Muscle soreness.** Muscle tenderness was measured at nine fixed sites on the biceps by measuring the force required to cause pain. A custom-designed, round-ended probe (2-cm diameter) was pressed into the muscle (25). The probe was calibrated so that a depression of 1 cm was equivalent to a force of 4 N. The pain score classification was the following: 0 cm depression causing pain = score of 4; 1 cm depression causing pain = score of 3; 2 cm depression causing pain = score of 2; 3 cm depression causing pain = score of 1; and 4 cm depression causing pain = score of 0.

The sum of scores of the nine sites in each subject represented the objective pain measure for that day. Muscle soreness was also measured subjectively according to a "rating of general perceived pain" on a scale of 0 to 10. Muscle soreness was assessed immediately before each test of muscle function.

**Biceps girth measurements.** The resting girth of the biceps muscle of each arm was measured 24 h before the initial exercise bout and again on each occasion when muscle function was remeasured. The girth was measured exactly mid-way between the acromion and radiale bony landmarks, with the arm relaxed and hanging by the side.

**Resting angle of elbow joint.** The resting angle of the elbow joint (humerus/arm) was measured with a goniometer 24 h before the initial exercise bout and again on each occasion when muscle function was remeasured. It was assumed that the increased flexion in the resting angle of the elbow joint was proportional to the shortening of the biceps muscle.

**Muscle function.** Muscle function of the damaged and nondamaged elbow flexor muscles was assessed using the Kin-Com isokinetic dynamometer. The maximum voluntary contraction was defined as the maximum force generated by the muscle while contracting at $60°\cdot s^{-1}$. Muscle function was measured 24 h before the initial exercise bout and remeasured at 12, 24, 48, 72, 96, 120, 144, and 168 h later.

**Blood sampling and analysis.** Blood samples (5 mL) were collected from an antecubital vein before each test of muscle function. Blood was allowed to clot and then the samples was kept on ice until centrifugation at 2000 × *g* for 10 min at 4°C. Samples were stored at −20°C until the analysis of serum creatine kinase (CK) activity. CK activity was measured by spectrophotometric (Beckman DU-62, Beckman Instruments, Fullerton, CA) enzymatic assays (CK-NAC-activated, Boehringer Mannheim Automated Analysis for BM/Hitachi Systems 704, Meylan, France).

**Statistical analyses.** All data are presented as the mean ± standard deviation. Statistical analyses were performed using Statistica software (StatSoft, Inc, Tulsa, OK). Statistical significance was assessed by an analysis of variance (ANOVA) with repeated measures. Scheffe's *post hoc* comparisons were performed where necessary. Statistical significance was accepted as $P < 0.05$.

## RESULTS

**Subjects.** The general characteristics of the subjects are shown in Table 1. There were no significant differences between groups for any of these variables.

TABLE 1. General characteristics of subjects in Acustat® ($N = 15$) and placebo ($N = 15$) groups (mean ± SD).

| Variables | Acustat | Placebo |
|---|---|---|
| Age (yr) | 29.5 ± 4.5 | 29.4 ± 3.9 |
| Mass (kg) | 76.6 ± 14.0 | 77.6 ± 10.5 |
| Stature (cm) | 178.4 ± 4.8 | 176.5 ± 7.5 |
| Sum of 7 skinfolds[a] (mm) | 86.1 ± 38.2 | 90.1 ± 27.7 |
| Body fat (%) | 18.4 ± 4.4 | 19.3 ± 3.5 |

[a] Triceps, biceps, subscapular, supra-iliac, abdominal, thigh, and calf.

**Tolerability.** No adverse events related to the membranes were reported for either group during the experiment.

**Muscle soreness.** The differences in general pain scores between the exercised and nonexercised arms are shown in Figure 3a. There was a significant difference in general pain over time ($F = 58.02$; $P < 0.0000001$), but the response was not different between groups, and there was no significant interaction of group versus time. The differences in pain scores measured with the pressure probe between the exercised and nonexercised arm are shown in Figure 3b. There was a significant difference over time ($F = 79.2$; $P < 0.0000001$), but the response was not different between

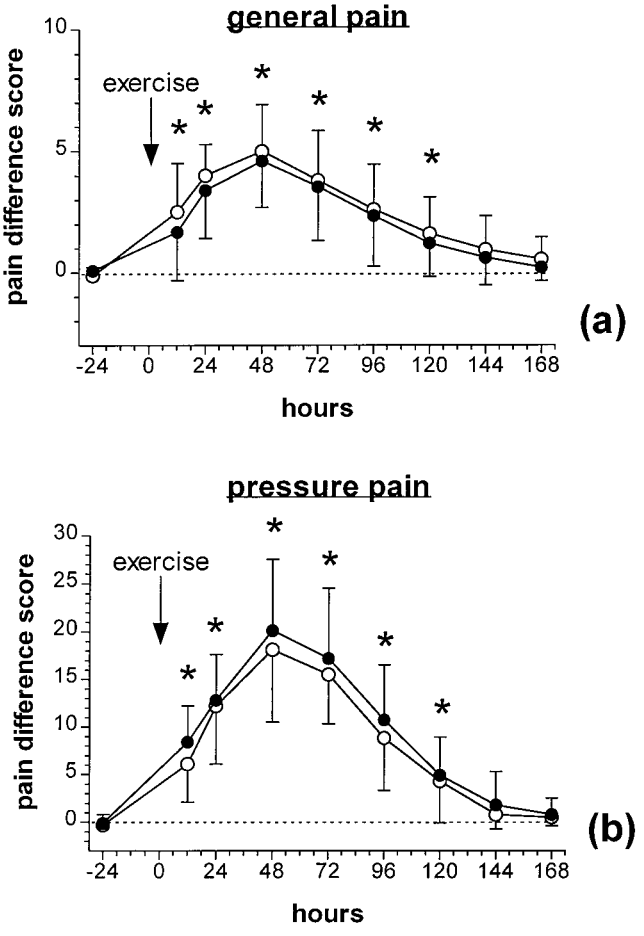


**FIGURE 3—a, Differences in general pain scores (subjective units) between the exercised and nonexercised arms of subjects in Acustat® (●) ($N = 15$) and placebo (○) ($N = 15$) groups (mean ± SD). * Pre vs 12, 24, 48, 72, and 96 h ($P < 0.00001$); * pre vs 120 h ($P < 0.0001$). b, Differences in pain scores measured with the pressure probe between the exercised and nonexercised arm of subjects in Acustat (●) ($N = 15$) and placebo (○) ($N = 15$) groups (mean ± SD). * Pre vs 12, 24, 48, 72, and 96 h ($P < 0.0001$); * pre vs 120 h ($P < 0.05$).**

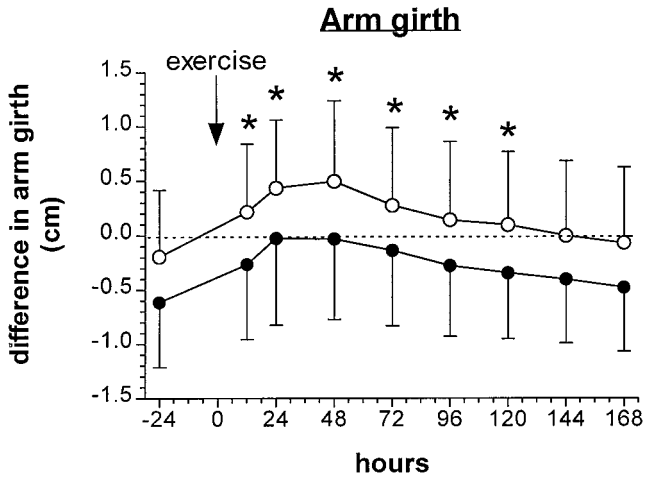


**FIGURE 4—Differences in arm girth (cm) between the exercised and nonexercised arms of subjects in Acustat (●) ($N = 15$) and placebo (○) ($N = 15$) groups (mean ± SD). Both groups: * pre vs 12, 24, 48, and 72 ($P < 0.00001$); * pre vs 96 h ($P < 0.0001$); * pre vs 120 h ($P < 0.01$).**

groups, and there was no significant interaction of group versus time.

**Muscle girth.** There was a significant increase in the girth measurements of the exercised arms from 12 h after the exercise protocol until 120 h later ($F = 17.75$; $P < 0.0000001$; Fig. 4). There was no difference between groups and there was no significant interaction of group versus time.

**Resting elbow joint angle (muscle length).** The differences between the joint angles, and by implication the muscle lengths of the damaged and undamaged elbow flexor muscles, are shown in Figure 5. There was a significant difference in the measurement over time ($F = 47.40$; $P < 0.0000001$). There was also a significant interaction of group versus time ($F = 2.063$; $P < 0.05$). The *post hoc* analysis showed that 24 h after the initial exercise bout, the joint angle of the damaged muscles of the placebo group was significantly more than that of the Acustat group ($P < 0.05$).

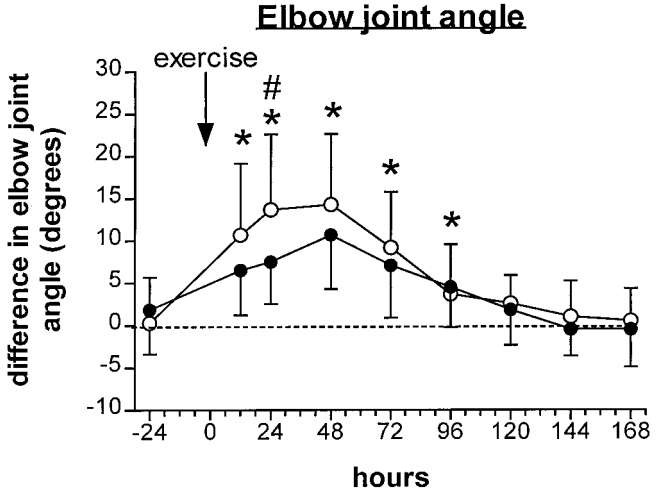


**FIGURE 5—Differences in elbow joint angle between the exercised and nonexercised arms of subjects in Acustat (●) ($N = 15$) and placebo (○) ($N = 15$) groups (mean ± SD). * Pre vs 12, 24, 48, and 72 h ($P < 0.00001$); * pre vs 96 h ($P < 0.0001$); # Acustat vs placebo at 24 h ($P < 0.05$).**

**Muscle function.** The maximum voluntary contraction of the exercised and nonexercised arms for the Acustat and placebo groups are shown in Table 2. Maximum voluntary contraction decreased at 12, 24, and 48 h in the placebo group ($F = 6.3$; $P < 0.01$). The data were normalized by adjusting the initial maximum voluntary contraction of the exercise and nonexercised arms to 100 and then all the subsequent values were adjusted accordingly (Fig. 6). Similarly, with this analysis muscle function was not different in exercised and nonexercised arms in the Acustat group for the duration of the trial (Fig. 6, top panel). In contrast, the maximum voluntary contraction was significantly lower in the exercised arm in the placebo group at 12 and 48 h after the initial exercise bout (Fig. 6, bottom panel).

**Serum creatine kinase activity.** There was a significant interaction between groups over time for serum creatine kinase activity ($F = 3.0$: $P < 0.01$ (Fig. 7). The serum creatine kinase activity was significantly lower in the Acustat group at 96, 120, 144, and 168 h (all different at $P < 0.05$).

## DISCUSSION

The eccentric exercise protocol used in this study induced changes in the elbow flexor muscles in the exercised arms consistent with DOMS, such as skeletal muscle microtrauma, inflammation, and loss of function (1,8,9,20,21). Treatment with Acustat reduced some of the clinical manifestations of damage, including reduced muscle shortening, as inferred by a reduction in elbow joint angle (Fig. 5), maintenance of maximum force production of the elbow flexor muscles, and reduced CK activity in the blood. It did not, however, alter pain perception (Fig. 3) or arm swelling (Fig. 4). As the study was adequately controlled and blinded, these results cannot be attributed to a placebo effect. The way in which Acustat caused these findings is not known, but a possible mechanism can be suggested on the basis of the known pathological changes in eccentric muscle damage and recovery.

The muscle shortening after damage in this experiment occurred at least 24 h before the appearance of CK in the blood (Fig. 5), and this is consistent with other studies (17,19). Muscles that shorten after damage are electrically silent (16,17), suggesting that the shortening results either from changes in the noncontractile elements of the muscle (21), from edema within the connective tissue network (16) or from cross-bridge formation arising from the presence of calcium in the cell. A transient reduction in force production (Fig. 6) is another consequence of muscle damage (8,9,17,20). This effect was prevented by Acustat even though postexercise pain was unaltered.

Perhaps the most striking action of Acustat was to reduce the creatine kinase activity in the blood (Fig. 7). Creatine kinase is released into the blood when the cell membrane is damaged or when there is a change in membrane permeability (1).

TABLE 2. The peak concentric force (Nm) of the nonexercised and exercised arm of subjects in in AcuSTAT® (n = 12) and placebo (n = 12) groups; all data are expressed as the mean ± SD.

| | Acustat | | Placebo | |
|---|---|---|---|---|
| Time | Nonexercised | Exercised | Nonexercised | Exercised |
| Pre | 82.3 ± 16.5 | 71.6 ± 15.6 | 76.6 ± 18.1 | 74.8 ± 12.8* |
| 12 h | 79.9 ± 21.3 | 63.0 ± 22.2 | 69.7 ± 18.4 | 52.7 ± 13.5* |
| 24 h | 79.5 ± 25.7 | 68.8 ± 24.1 | 70.6 ± 15.2 | 57.2 ± 14.4* |
| 48 h | 79.4 ± 25.2 | 66.8 ± 23.8 | 74.2 ± 16.5 | 56.1 ± 17.3* |
| 72 h | 79.7 ± 24.2 | 71.0 ± 25.1 | 76.0 ± 19.3 | 61.5 ± 16.1 |
| 96 h | 79.5 ± 22.8 | 70.4 ± 22.6 | 70.5 ± 18.6 | 63.9 ± 16.9 |
| 120 h | 83.5 ± 20.7 | 74.9 ± 23.0 | 73.4 ± 19.4 | 63.8 ± 14.5 |
| 144 h | 79.3 ± 12.9 | 73.3 ± 25.1 | 70.8 ± 17.3 | 65.4 ± 15.8 |
| 168 h | 82.2 ± 17.0 | 75.8 ± 23.4 | 66.5 ± 17.1 | 61.7 ± 17.5 |

Placebo group: pre vs 12, 24, 48 h in exercised arm ($P < 0.01$).
* Data from three subjects in each group were lost in the software storage process.

Increases in intracellular calcium concentration may influence these changes in membrane integrity (2,17) through an increase in calcium influx through stretch-activated channels in the sarcolemma. Calcium then moves into the cell down the concentration gradient (3,14). Alternatively calcium influx can occur through ruptures or lesions in the sarcolemma (12).

Although transient changes in calcium concentration are essential for muscle excitation-contraction coupling, sustained increases may result in the activation of calcium sensitive proteases and phospholipases (5). This is deleterious to cell membrane and sarcoplasmic reticulum integrity, causing a change in membrane permeability (1,18). Another consequence of a sustained elevation of intracellular $Ca^{2+}$ concentration is the activation of nonlysosomal cysteine proteases, such as calpain (5). Calpain cleaves a variety of protein substrates, including cytoskeletal and myofibrillar proteins. Calpain-mediated degradation of these proteins is thought to contribute to changes in muscle structure (15). These morphological changes in the contractile machinery of the muscle may underlie the reduced muscle function of DOMS.

Interference with mechanisms of calcium homeostasis might explain the beneficial effects of Acustat in this study. The failure of Acustat to influence the extent of the development of pain and arm swelling suggests that other pathophysiological processes influence these changes. Several other studies have shown a dissociation between serum CK activity, pain, and the decrement in muscle function in experimental DOMS (7,21,22). It may also be that pain pathways are fully recruited at a moderate level of muscle inflammation and that the charged membrane's protective effect did not reduce the nociceptive stimuli to a level at which less pain would be felt. These theories are speculative and will have to be confirmed with further experiments.

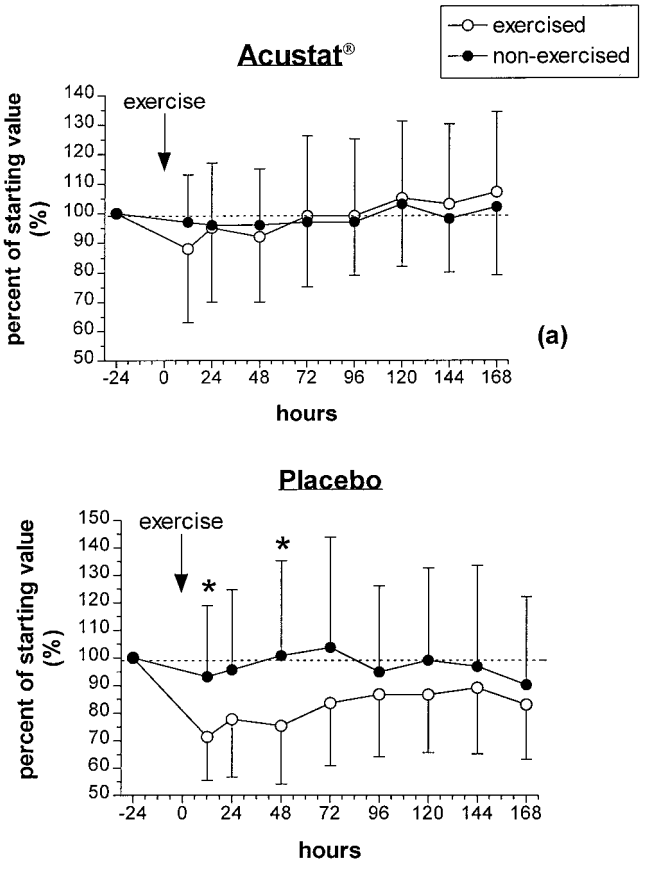


**FIGURE 6—The maximum voluntary contraction of the exercised and nonexercised elbow flexor muscles in the Acustat and placebo groups. The data are normalized by expressing the starting value as 100 and then adjusting all the subsequent values accordingly, and are expressed as mean ± SD. * Pre vs 12 h placebo ($P < 0.01$); * pre vs 48 h placebo ($P < 0.05$); # data from 3 subjects in each group were lost in the software storage process.**

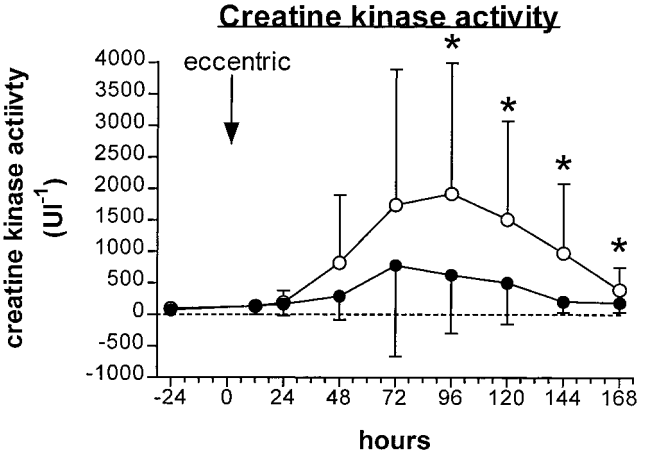


**FIGURE 7—Serum creatine kinase activity (U·L$^{-1}$) in Acustat (●) ($N = 15$) and placebo (○) ($N = 15$) groups before, and for 168 h after the eccentric exercise protocol. * 96 h Acustat vs placebo ($P < 0.05$); * 120 h Acustat vs placebo ($P < 0.05$); * 144 h Acustat vs placebo ($P < 0.05$); * 168 h Acustat vs placebo ($P < 0.05$).**

In summary, these data show that Acustat electro-membrane microcurrent therapy reduces some of the clinical features of delayed onset muscle soreness. The mechanisms of action are not known but may be related to a reduction in the disturbance of the intracellular $Ca^{2+}$ homeostasis. Further studies need to examine the physiological significance of these clinical features.

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The results of the study do not constitute endorsement of the product by the authors or the American College of Sports Medicine.

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