

available at www.sciencedirect.comjournal homepage: www.elsevier.com/jbmtJournal of
Bodywork and
Movement
Therapies

MICROCURRENT ELECTROTHERAPY

The efficacy of frequency specific microcurrent therapy on delayed onset muscle soreness

Denise Curtis, MSc, NMT^{a,*}, Stephen Fallows, PhD^a, Michael Morris, MSc^a, Carolyn McMakin, MA DC^b

^a Centre for Exercise & Nutrition Science, University of Chester, Parkgate Road, Chester CH1 4BJ, England, UK

^b Fibromyalgia and Myofascial Pain Clinic of Portland, 69 SW Hampton Street, Portland, OR 97223, USA

Received 6 October 2008; received in revised form 11 January 2010; accepted 24 January 2010

KEYWORDS

Frequency specific
microcurrent therapy;
Delayed onset muscle
soreness;
Eccentric;
VAS

Summary This study compared the effects of frequency specific microcurrent (FSM) therapy versus sham therapy in delayed onset muscle soreness (DOMS) in order to determine whether specific frequencies on two channels would produce better results than single channel single frequency microcurrent therapy which has been shown to be ineffective as compared to sham treatment in DOMS. 18 male and 17 female healthy participants (mean age 32 ± 4.2 years) were recruited. Following a 15-min treadmill warm-up and 5 sub-maximal eccentric muscle contractions, participants performed 5 sets of 15 maximal voluntary eccentric muscle contractions, with a 1-min rest between sets, on a seated leg curl machine. Post-exercise, participants had one of their legs assigned to a treatment (T) regime (20 min of frequency specific microcurrent stimulation), while the participant's other leg acted as control (NT). Soreness was rated for each leg at baseline and at 24, 48 and 72 h post-exercise on a visual analogue scale (VAS), which ranged from 0 (no pain) to 10 (worst pain ever). No significant difference was noted at baseline $p = 1.00$. Post-exercise there was a significant difference at 24 h ($T = 1.3 \pm 1.0$, $NT = 5.2 \pm 1.3$, $p = 0.0005$), at 48 h ($T = 1.2 \pm 1.1$, $NT = 7.0 \pm 1.1$, $p = 0.0005$) and at 72 h ($T = 0.7 \pm 0.6$, $NT = 4.0 \pm 1.6$, $p = 0.0005$). FSM therapy provided significant protection from DOMS at all time points tested. © 2010 Elsevier Ltd. All rights reserved.

Introduction

Delayed onset of muscle soreness (DOMS) has been described as damaged muscle tissue membranes combined with

a secondary inflammatory condition (Gleeson et al., 1995; Wilmore and Costill, 2004; Connolly et al., 2003) resulting from unaccustomed eccentric contractions (Taleg, 1973; Newman et al., 1983a,b; Armstrong, 1984; Denegar and Perrin, 1992) and maximal isometric contractions (Clarkson

* Corresponding author. Tel.: +353 46 9059095.

E-mail address: denisemcurtis@yahoo.co.uk (D. Curtis).

et al., 1986). Although many variables are reported in the quantification of muscle damage, the typical symptoms associated with DOMS are loss of strength, pain, muscle tenderness, stiffness, swelling and elevated levels of the enzyme creatine kinase (McHugh et al., 1999). Symptoms can vary from mild muscle tenderness to severe debilitating pain (Cheung et al., 2003).

DOMS is a well researched phenomenon and the morphological injury to the muscle has been well described, however the mechanism underlying the injury remains poorly understood. For many years, DOMS was attributed to an accumulation of the metabolic end products of exercise resulting in elevated muscle lactate. This assumption is now understood to be unconnected to DOMS. It is now proposed that the soreness may be the result of, amongst others, mechanical (Newman et al., 1983a,b; Armstrong, 1984; Stauber et al., 1990) or biochemical (Armstrong, 1984; McIntyre et al., 1995) factors.

Research suggests that the soreness typically appears between 8 and 24 h post-exercise, peaks at 24–48 h and can last for up to 7 days (Cleak and Eston, 1992; Howell et al., 1993).

Although the precise details of muscle damage following eccentric exercise remains unknown, it appears that even a single bout of eccentric muscle contractions can offer significant protection against muscle soreness in subsequent performances of the same exercise. This phenomenon, which has been known to last for several months, was termed the "repeated bout effect" by Nosaka and Clarkson (1995).

DOMS is a universal symptom familiar to most athletes that usually occurs after an extended layoff from exercise or unfamiliar, predominantly eccentric exercise. Athletic performance is typically impaired when an athlete is sore. Research by Proske et al. (2003) implied that muscle soreness, following a bout of unaccustomed eccentric exercise, may also contribute to muscle weakness, possibly as a result of reduced excitability of the motor cortex. Thus, any practice or therapy that limits soreness and restores the maximal function of the muscles as quickly as possible would be of interest and practical value to the athlete.

Numerous treatment strategies, both prophylactic and rehabilitative, have been introduced to help relieve the severity of DOMS. Some of the proposed treatments include pre- and post-exercise static stretching (Herbert and Gabriel, 2002; Cornwell et al., 2002; Yamaguichi and Ishii, 2005), pharmacological treatments using non-steroidal anti-inflammatory drugs (NSAIDs) (Grossman et al., 1995; O'Grady et al., 2000; Sayers et al., 2001; Connolly et al., 2003; Lanier, 2003), nutritional supplements (Kaminski and Boal, 1992; Warren et al., 1992; Jakeman and Maxwell, 1993), massage therapy (Tiidus and Shoemaker, 1995; Lightfoot et al., 1997), continuous compression (Kraemer et al., 2001) and ice-water immersion (Sellwood et al., 2007). However, little scientific evidence exists to support the effectiveness of any of these therapeutic interventions.

The following study compared the effects of FSM therapy versus sham therapy on DOMS in order to determine if the use of certain specific frequencies would produce better results than simple single frequency microcurrent therapy which had been shown by Allen et al. (1999) to be ineffective when compared to sham treatment in DOMS. Allen et al. (1999) determined that 20 min of single channel,

single frequency microamperage current using 30 Hz at 200 μ A for 10 min and 0.3 Hz at 100 μ A for 10 min was not effective in reducing pain or increasing range of motion 24, 48 and 72 h after DOMS induction in the biceps muscle in a group of 18 subjects (3 males, 18 females). The sham group in the Allen paper received treatment from a unit that had been disabled by the manufacturer to provide no electrical stimulation and both the subjects and experimenter were blinded. In the present study the sham treatment was provided by a unit that was not turned on and only the subjects were blinded. To the authors' knowledge, no controlled studies to date have examined the effects of FSM therapy on DOMS.

History of frequency specific microcurrent (FSM) therapy

Microcurrent electrical neuromuscular stimulation (MENS) was developed in the 1970s as a battery operated physical therapy modality delivering current in the microampere range. An ampere (amp) is a measure of the strength of electric current and measures the rate of flow of charge in a conducting medium. One micro amp (μ A) equals 1/1000th of a milliamp (mA). By comparison, interferential, TENS, and high-volt pulsed galvanic stimulators deliver currents in the milliamp range causing muscle contraction, pulsing and tingling sensations. TENS applies an electrical force that stimulates pain suppressing A-beta afferent fibers which compete against A-delta and C fibers that transmit pain signals. Most TENS units deliver current around the 60 mA range (Kirsch and Lerner, 1998). Although microcurrent devices are approved in the category of TENS for regulatory convenience, in practical use they are in no way similar and cannot be compared to TENS in their effect.

With microcurrent the patient cannot feel the current since there is not enough current to stimulate sensory nerve fibers (Mercola and Kirsch, 1995). Traditionally, microcurrent therapy has been used to increase the rate of healing in injured athletes, to treat and manage muscle pain and dysfunction and to increase the rate of fracture repair (Rowley et al., 1974; Bertolucci and Grey, 1995; Kirsch, 1996, 1997; Lambert et al., 2002).

Current in the range of 10 up to 500 μ A was observed to increase ATP production, amino acid transport, protein synthesis, and waste product removal in rat skin whereas ATP production leveled off between 500 and 1000 μ A and decreased when the current was above 1000 μ A (Cheng, 1982). TENS devices provide up to 60 times higher current levels than that seen to decrease ATP production which may explain why TENS units have not been found to be effective in treatment of DOMS (Craig et al., 1996). Typical microcurrent applications use only low and simple one channel frequencies such as 0.3 Hz, 3 Hz, 10 Hz, 30 Hz, and 300 Hz (Manley, 1994; Allen et al. 1999).

The therapeutic use of frequencies and electrotherapy began in the early 1900s in the United States and England with thousands of medical physicians using a number of devices to treat a wide range of conditions from arthritis and tuberculosis to pneumonia (Kirsch and Lerner, 1998). The Electromedical Society and the journal *Electromedical Digest* served as a forum for physicians to share their

research and clinical findings. Copies of *Electromedical Digest* were found in the rare book room of the National College of Naturopathic Medicine in Portland containing frequencies and protocols for the above conditions and articles documenting clinical outcomes in every edition available published between 1920 and 1951. In 1934, as part of its effort to standardize medicine and medical education, the American Medical Association (AMA) decreed that pharmaceutical medications and surgery were the legitimate tools of medicine and that electromagnetic therapies, homeopathy, herbs and other treatments were "unscientific" (Berliner, 1975; Barzansky and Gevitz, 1992). The biophysics and medical research that would provide the mechanisms and science explaining electro-medicine would not be done until the 1980s (Becker and Seldon, 1985; Oschman, 2000). The use of electromagnetic therapies and frequencies declined, the research being reported in *Electromedical Digest* ceased and the last edition of the journal available was published in 1951 (*Electronic Medical Digest*, 1951). The FDA made the original devices illegal around the same time.

The frequencies used in this study were obtained in 1995 from a retired British osteopath who bought a practice in Vancouver, BC (Canada) in 1946 that came with a machine (manufacturer unknown) and a list of frequencies that were created in 1922 thought to address specific tissues and neutralize specific conditions. The list acquired from the osteopath included approximately 100 frequencies alleged to neutralize certain pathologies or conditions and over 200 frequencies thought to address certain tissues. The osteopath's method of treatment included using a frequency on one channel to "remove a pathology" combined with a frequency on the second channel to "address a specific tissue". The device used by the osteopath has long since disappeared and has never been available for inspection. While it is thought to have plugged into the wall current which may have been DC in 1922, it is not known what current level it delivered and there is no reason to suspect that it delivered microamperage current which was not introduced until the early 1980s. Frequencies found on the back page of *Electromedical Digest* in a wall chart being sold by Albert Abrams were identical to those that came with the osteopath's machine where the two lists overlapped. The use of microcurrent and frequencies for the treatment of nerve, muscle pain and injury repair was developed clinically using the osteopath's two channel, condition and tissue treatment paradigm and has been taught as Frequency Specific Microcurrent (FSM) since 1997 (McMakin, 1998; McMakin, 2004; McMakin et al., 2005).

The technique requires use of any microcurrent device that can provide a different frequency on each of two channels using a ramped square wave and alternating pulsed direct current. The devices used in this study are calibrated by the manufacturer (Precision Microcurrent, Newberg, Oregon, USA) and the company standards require that the frequencies be accurate to within 0.5 Hz on both channels. Frequencies on one channel are thought to be effective in neutralizing specific conditions such as hemorrhage, fibrosis, scar tissue, mineral deposits, histamine, and acute and chronic inflammation. These frequencies are combined with frequencies on a second channel thought to be specific for muscles, fascia, tendons, nerves and arteries and other tissues (McMakin, 2004).

The frequency specific protocols were developed clinically through trial and error by one of the authors after it was determined through clinical use on volunteers that the use of a frequency combination that did not produce improvement also did no apparent harm. The descriptions of the frequencies from the list were taken at face value and used speculatively for various chronic and acute conditions in clinical practice to determine if they would produce a change in symptoms and clinical improvement (McMakin, 1998; McMakin, 2004; McMakin et al., 2005).

For example, the frequencies described on the list as reversing "hemorrhage" in the "arteries" were used speculatively in acute injuries to reduce bruising "as if" it correctly represented the effect of the frequency. It was subsequently observed not only to prevent bruising and reduce pain but also coincidentally noted to stop bleeding for up to 12 h in patients who were menstruating at the time of treatment. No other frequency tried produced this effect. This frequency had no effect on any other condition. No formal research has been done to verify the effect of this frequency but it has been reproduced on numerous occasions by the authors and many of the 1200 clinicians using FSM worldwide including athletic trainers for the USA National Football League (NFA), surgeons and an obstetrician who use this frequency specifically to stop bleeding and bruising in medically appropriate settings.

The other frequencies used in FSM therapy were explored in the same way. 40 Hz was described on the osteopath's list and in *Electromedical Digest* as being useful to "reduce inflammation". Use of this frequency in a clinical setting suggested that it did only that and was not useful to change any other condition. Use of 40 Hz on channel A and 10 Hz on channel B was found to reduce pain in fibromyalgia patients and to reduce all of the inflammatory cytokines as measured by micro-immunochromatography (McMakin et al., 2005). One control patient treated with a protocol that did not include 40 Hz had no change in cytokines (McMakin et al., 2005).

Clinical response to the frequencies over the last 14 years suggests that the conditions being treated and the tissues being addressed are accurately represented by the frequency descriptions although decades of research will be required to confirm and clarify these effects. Until such research is done no claims can be or are made by the authors for the specific effects of frequencies on biological tissues or conditions. Clinical research, such as this paper, may report the observed and reported effects in a research setting of certain frequency combinations without making specific claims for the frequencies used. Fortunately, medicine is pragmatic and it is not uncommon for apparently effective medications, such as aspirin, to be used for many years before the mechanism is understood.

Methods

Participants

Following the posting of an advertisement on the student notice board at the National Training Centre (NTC) in Dublin, Ireland, forty-four students volunteered to participate in the

study. 18 male and 17 female students (mean age 32 ± 4.2 years) were selected from these volunteers to take part in the study. Each of the participants had one of their legs assigned to a treatment group, while the opposite leg was assigned to a control group. The nomination of the participants' leg (left or right) as treatment or control was randomized by the toss of a coin.

Participants were required to meet the following inclusion and exclusion criteria to be eligible for the study.

Inclusion

Participants were:

- 1) aged between 20 and 40 years;
- 2) healthy and recreationally active;
- 3) required complete a health screening questionnaire prior to the study;
- 4) required to give written consent.

Exclusion

Potential participants were excluded if they were:

- 1) engaged in resistance training or eccentrically biased exercises for the lower body three months prior to the study;
- 2) suffering from unstable cardiovascular or pulmonary conditions or diseases;
- 3) suffering from any pain or injury in the legs or other health problems;
- 4) pregnant.

Participants received a participant information sheet two weeks before the study commenced and were given three days to decide if they wanted to be involved in the research. The study was reviewed by the Ethics Committee of the School of Applied and Health Sciences, University of Chester, UK. A health screening questionnaire was completed by each of the participants on the day of the study to rule out any pathology that may have excluded them from taking part in the research. Participants were asked not to massage, stretch or treat the hamstring muscles in any way and to refrain from NSAIDs or supplements until the final set of data was completed. Massage, stretching, NSAIDs and supplements are common practices that exist for the treatment of DOMS and may therefore have affected the final results.

Once the students agreed to participate in the study and had given written consent a phone call was made to each participant to confirm times and dates and also reconfirm inclusion criteria. During this phone conversation participants were verbally instructed to drink at least 2 l of water in the 2 h prior to their allocated time for participation in the study. During the warm-up and training session, a 500 ml bottle of water was provided to each participant to prevent dehydration.

Design

Following a 15-min warm-up on an *ascent* Pulse Fitness® treadmill, at a speed of 6 km/h, participants were instructed to perform five sub-maximal eccentric contractions on a Pulse Fitness® seated leg curl machine to familiarize

themselves with the equipment. Participants were then instructed to perform five sets of 15 maximal eccentric contractions, with a 1-min rest between each set.

Post-exercise, one of their legs, randomly chosen, underwent a 20-min FSM programme and the other leg was not treated. The frequencies delivered in the programme were chosen from a list provided by Frequency Specific Seminars, Inc. (Vancouver, Washington, USA) and are thought to be specific for tissues and conditions. The channel A frequency values that were used in this study were chosen because they were thought to be specific to some of the main pathologies induced by DOMS, while the channel B frequency values that were used were chosen because they were thought to be specific to some of the main soft tissues that are affected by DOMS. 18 Hz on channel A was combined with 62 Hz on channel B for 4 min. 124 Hz on channel A was combined with 62 Hz, 142 Hz and 191 Hz on channel B for 1 min each. 40 Hz on channel A was combined with 116 Hz on channel B for 4 min. 40 Hz on channel A was combined with 62 Hz, 142 Hz and 191 Hz on channel B for 2 min each. 49 Hz on channel A was combined with 62 Hz, 142 Hz and 191 Hz on channel B for 1 min each. The intensity was set at 200 μ A and the waveslope was set at 10 for the entire 20-min programme.

Procedure for seated leg curl

Participants were asked to sit into a Pulse Fitness® leg curl machine and align the knee joint with the axis of the machine. The seat was then set so that their backs made full contact with the back rest and to ensure that the posterior aspect of the knee joint was positioned at the edge of the leg curl seat. Starting in full leg extension, their ankles were dorsi flexed and placed on the rollers with the feet no wider than hip distance apart. Subjects were asked to hold the side handles for support. The machine was set to allow for full range of movement (Figure 1). Subjects were instructed to curl the rollers downwards and backwards to full leg flexion (Figure 2) and then slowly return the rollers to full leg extension. Male participants began with a starting weight of 25 kg, whereas female participants started with a weight of 20 kg. Participants either performed 15 repetitions with their starting weight or continuous repetitions until they could no longer push or resist the weight. When participants could no longer push or resist the weight, the weight was reduced by 5 kg and the protocol continued either to fatigue or until the fifteen repetitions were completed. Participants were verbally encouraged to exert maximal resistance in the upward (eccentric) phase of the movement. To ensure consistency, participants were instructed to control the lifting velocity of the rollers by counting from one to five from the beginning to the end range of the eccentric action.

Procedure for FSM treatment

Post-exercise, participants were instructed to lie in the prone position on a massage table. Each of the participants' legs were attached to separate FSM machines (Precision Microcurrent, Newberg, Oregon, USA) that were placed on either side of the table in alignment with the hamstring



Figure 1 Eccentric end range on leg curl machine.

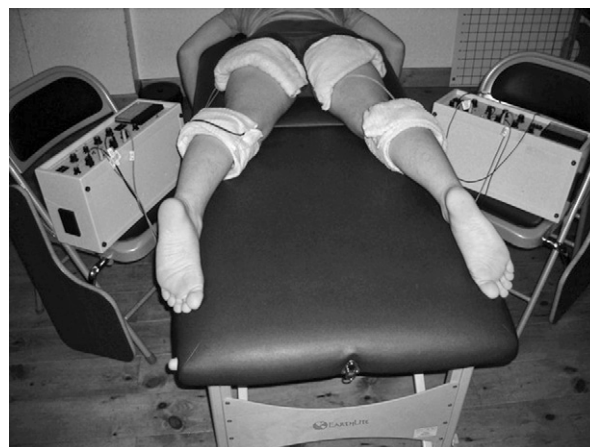


Figure 3 Subject position for FSM treatment.

muscles and positioned so that the patient could not see the front panel of the device or determine which machine was turned on (Figure 3). As DOMS was induced in only the hamstring muscle group, the current was directed only through the soft tissues in this muscle group. The positive leads from the device were attached to graphite gloves that were wrapped in wet towels and placed on the upper portion of the participants' thighs. The negative leads were attached to graphite gloves that were wrapped in wet towels and placed below the participants' knees. This allowed the current to flow between the two leads through the soft tissues of the treated leg. One of the machines was turned off providing the sham treatment and the volume on the working machine was turned down.

Rated soreness and tenderness were evaluated at baseline and 24, 48 and 72 h post-exercise using a visual analogue scale (VAS). The VAS consists of a 10 cm horizontal line with the two end points labeled 0 (no pain) to 10 (worst soreness ever) (Huskinson, 1974; Joyce et al., 1975). Participants were asked to make a vertical slash across the 10 cm line that corresponded to the level of pain intensity between the limits of no pain felt (left end of line) and worst soreness ever (right end of line). A blank scale was used each time to avoid bias from previous measurements.



Figure 2 Concentric end range on leg curl machine.

The VAS has been shown to be a valid and reliable measurement for determining the intensity of human pain, it is minimally intrusive and is easily and quickly administered (Lee and Kieckhefer, 1989; Mattacola et al., 1997).

Statistical analysis

As the VAS falls into the ratio level of measurement (Myles et al., 1999), parametric tests were conducted to investigate significant differences within and between the groups. Changes in the VAS within the groups were analysed via a One Way repeated measures ANOVA and post hoc analysis utilising multiple paired t-tests. Differences between the groups were investigated using multiple Independent t-tests, one at each time point (baseline, 24, 48 and 72 h). Normality was assessed and confirmed prior to each test via the Shapiro Wilk statistic and data are presented as mean \pm standard deviation (SD). All data were analysed using SPSS for Windows (Version 14.0) and significance was set at the 0.05 level. A post hoc sample size calculation based on the data from this study revealed an effect size 0.08. Based on a significance level of 0.05, being a two tailed test with 80% power, this provided a sample size of 26 participants in each group.

Results

Perceived muscle soreness

The baseline values for perceived muscle soreness before exercise as assessed by the VAS (Table 1) for each group revealed no significant difference ($p = 1.000$). This indicated that the groups had no prior muscle pain and understood how to use the scale correctly.

Once each group had undergone the exercise regime to induce the muscle damage the ratings on the VAS significantly increased. This was observed in both groups with the non-treatment group increasing from zero at baseline to 5.2 ± 1.3 at 24 h ($p = 0.0005$) and the treatment group increasing from zero to 1.3 ± 1.0 ($p = 0.0005$). This significant increase demonstrated that the exercise regime had worked at inducing muscle damage. It can also be seen that

Table 1 Perceived muscle soreness at baseline, 24, 48 and 72 h for each treatment.

	Baseline	24 h	48 h	72 h
Treatment	0 ± 0	1.3 ± 1.0 ^a	1.2 ± 1.1 ^a	0.7 ± 0.6
Non-treatment	0 ± 0	5.2 ± 1.3 ^a	7.0 ± 1.1 ^a	4.0 ± 1.6 ^a
<i>p</i> Value (between groups)	1.000	0.0005	0.0005	0.0005

N.B.: Results presented as mean ± SD.

^a Significant difference from baseline VAS score within each group ($p < 0.05$).

the non-treatment group reported a significantly greater ($p = 0.0005$) increase in perceived muscle soreness than the treatment group.

A similar trend existed between 24 and 48 h with both groups demonstrating significant increases in perceived muscle soreness (non-treatment $p = 0.0005$; treatment group $p = 0.001$). The perceived muscle soreness in the treatment group (1.2 ± 1.1) was significantly less ($p = 0.0005$) than in the non-treatment group (7.0 ± 1.1).

At 72 h the perceived muscle soreness in the treatment group had almost returned to baseline levels (0.7 ± 0.6) indicating an absence of any pain although the scores in the non-treatment group remained elevated (4.0 ± 1.6) and significantly higher than baseline values ($p = 0.0005$). A summary of the results is presented in Figure 4.

Discussion

The aim of this investigation was to compare the effects of FSM therapy versus sham at 24, 48 and 72 h post-exercise. Allen et al. (1999) found that 20 min of single channel microcurrent therapy that was not frequency specific compared to sham treatment was not effective in reducing pain or increasing range of motion 24, 48 and 72 h after DOMS induction in the biceps muscle. Clinical evidence suggested that dual channel microcurrent using different frequency combinations was very effective in reducing the pain associated with muscle trauma and DOMS providing the motivation for conducting this study. This study was undertaken to provide a controlled trial evaluation of

20 min of FSM therapy compared to sham treatment. It was hypothesized that FSM therapy would offer significant protection from post-exercise muscle soreness.

The participants were blinded to which leg was being treated because they could not see the machine and because the current is subsensory. This reduced the possibility of a placebo effect while the treatment was being given. However it should be acknowledged that by using the participant's opposite leg as a control, it is likely that participants could have guessed quite quickly (probably within hours of the treatment being given) which leg had been treated and which leg had not. As initial improvements in one leg may have had them guessing which leg had been treated, this would have meant that they were no longer blinded.

The use of graphite gloves wrapped in wet towels as conductors was assumed to prevent the reduction in voltage seen in Petrovsky's measurements of graphite electrodes in TENS devices (Petrofsky et al., 2006). The use of wet towels also ensures that the current will remain subsensory since graphite electrodes against dry skin may make even microamperage current sensible (Grimnes, 2008).

The method selected for inducing DOMS was deemed successful, as the data collected at 24, 48 and 72 h post-exercise differed significantly from the data collected at baseline. This pattern was similar to previous literature related to the time course and intensity of DOMS (Cleak and Eston, 1992; Howell et al., 1993; Nosaka and Clarkson, 1996) and suggests that the methodology was appropriate to create DOMS. The VAS was selected as a measurement for perceived pain because it is patient friendly, low cost, easy to administer and not too time consuming. However, although the VAS is a well established, valid and reliable measurement for determining the intensity of pain, the authors acknowledge the possibility that variations may have occurred in participant responses during the data collection period. As participants were required to independently perceive their soreness at four different time points over a 72 h period, it should be noted that how they perceived their pain may have altered over the 72 h, depending on how they were being affected by their pain at that specific moment. Also, individual tolerance for pain can vary greatly from person to person.

Acknowledgement is also given to the possibility that reciprocal facilitation may have had an effect on the overall findings. The FSM units were set up so that the treated leg had current flowing between the positive and negative leads while the untreated leg had no current delivered as the machine on this leg was turned off. Although there is no evidence in any electrical theory or practice that suggests that the current will migrate to other areas outside the area between the two leads, because of the interconnectedness of the body there is no way of knowing what effect, if any, the FSM treatment had on the control leg.

No attempt was made in the present study to control for the effects of environmental electromagnetic influences ("electronic smog") since such influences would have had equivalent effects on both the treated and untreated leg. In future studies in which there is a sham control group being treated at another time and setting than the

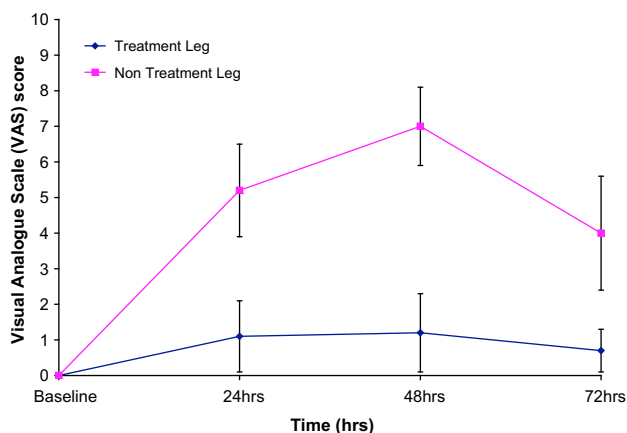


Figure 4 VAS scores for TL and NTL over a period of 72 h. N.B.: Results expressed as mean ± SD.

treatment group consideration may need to be made for the possible effects of extraneous electrical interference.

A possible limitation to the study was that only one marker, perceived muscle soreness, was used to assess muscle damage. Other markers such as maximal isometric strength, range of motion, angle of peak torque, leg circumference and plasma creatine kinase levels were not assessed.

A suggestion for future research would be to use multiple markers as an assessment for DOMS. Future studies of FSM in DOMS or any condition may include a methodology that includes separate sham and active treatment groups and will also allow for double blinding the subjects and the experimenters. Even though the patients turned in their pain scores without any further contact with the un-blinded experimenter, the possibility of some experimental error due to lack of experimenter blinding cannot be excluded. To ensure double blinding it is suggested that future studies include a sham unit that is disabled by the manufacturer so that no electrical stimulation passes through it.

Conclusion

The results of this study show that at the parameters selected for this investigation FSM therapy did provide significant protection from post-exercise muscle soreness.

Acknowledgements

The authors would like to thank Bobby Fitzsimons for his help and assistance with this research project and also the students at the NTC who took part in the study.

No funding of this study or incentive for publication was provided by any commercial interest.

References

- Allen, J.D., Mattacola, C.G., Perrin, D.H., 1999. Effect of micro-current stimulation on delayed onset muscle soreness: a double blind comparison. *Journal of Athletic Training* 34, 334–337.
- Armstrong, R.B., 1984. Mechanisms of exercise-induced delayed onset muscular soreness: a brief review. *Medicine and Science in Sports and Exercise* 16, 529–538.
- Barzansky, B.S., Gevitz, N., 1992. *Beyond Flexner: Medical Education in the Twentieth Century*. Greenwood Press, Westport, CT, pp. 195–222.
- Becker, R.O., Seldon, G., 1985. *The Body Electric: Electromagnetism and the Foundation of Life*. Quill/William Morrow, New York.
- Berliner, H.S., 1975. A larger perspective on the Flexner report. *International Journal of Health Services* 5 (4).
- Bertolucci, L.E., Grey, T., 1995. Clinical comparative study of microcurrent electrical stimulation to mid-laser and placebo treatment in degenerative joint disease of the temporomandibular joint. *Cranio: The Journal of Craniomandibular Practice* 34, 602–607.
- Cheng, N., 1982. The effect of electric currents on ATP generation, protein synthesis and membrane rat skin. *Clinical Orthopedics* 171, 264–272.
- Cheung, K., Hume, P., Maxwell, L., 2003. Delayed onset muscle soreness: treatment strategies and performance factors. *Sports Medicine* 33, 145–164.
- Clarkson, P.M., Byrnes, W.C., McCormack, K.M., Turcotte, L.P., White, J.S., 1986. Muscle soreness and serum creatine kinase activity following isometric, eccentric and concentric exercise. *International Journal of Sports Medicine* 7, 152–155.
- Cleak, M.J., Eston, R., 1992. Muscle soreness, swelling, stiffness and strength loss after intense eccentric exercise. *British Journal of Sports Medicine* 26, 267–272.
- Connolly, D.A.J., Sayers, S.P., McHugh, M.P., 2003. Treatment and prevention of delayed onset muscle soreness. *Journal of Strength and Conditioning Research* 17, 197–208.
- Cornwell, A., Nelson, A.G., Sidaway, B., 2002. Acute effects of stretching on the neuromechanical properties of the triceps surae muscle group. *European Journal of Applied Physiology* 86, 428–434.
- Craig, J.A., Cunningham, M.B., Walsh, D.M., Baxter, G.D., Allen, J.M., 1996. Lack of effect of transcutaneous electrical nerve stimulation upon experimentally induced delayed onset muscle soreness in humans. *Pain* 66, 285–289.
- Denegar, C.R., Perrin, D.H., 1992. Effect of transcutaneous electrical nerve stimulation on pain, decreased range of motion and strength loss associated with delayed onset muscle soreness. *Journal of Athletic Training* 27, 200–206.
- Electronic Medical Digest, 1951. Electronic Medical Foundation, San Francisco, CA (Paper copy in rare book room at National College of Naturopathic Medicine, Portland Oregon).
- Gleeson, M., Almey, J., Brooks, S., Cave, R., Lewis, A., Griffiths, H., 1995. Haematological and acute-phase responses associated with delayed-onset muscle soreness in humans. *European Journal of Applied Physiology* 71, 137–142.
- Grimnes, S., 2008. Electrovibration, cutaneous sensation of microampere current. *Acta Physiologica Scandinavica* 118 (1), 19–25.
- Grossman, J.M., Arnold, B.L., Perrin, D.H., Kahler, D.M., 1995. Effect of ibuprofen use on delayed onset muscle soreness of the elbow flexors. *Journal of Sport Rehabilitation* 4, 253–263.
- Herbert, R.D., Gabriel, M., 2002. Effects of stretching before and after exercising on muscle soreness and risk of injury: systematic review. *British Medical Journal* 325, 468.
- Howell, J.M., Chleboun, G.S., Conatser, R.R., 1993. Muscle stiffness, strength loss, swelling and soreness following exercise induced injury to humans. *Journal of Physiology* 464, 183–196.
- Huskinson, E.C., 1974. Measurement of pain. *Lancet* 2, 1127–1131.
- Jakeman, P., Maxwell, S., 1993. Effect of antioxidant vitamin supplementation on muscle function after eccentric exercise. *European Journal of Applied Physiology* 67, 426–430.
- Joyce, C.R.B., Zutshi, D.W., Hrubes, V., Mason, R.M., 1975. Comparison of fixed interval and visual analogue scales for rating chronic pain. *European Journal of Clinical Pharmacology* 8, 415–420.
- Kaminski, M., Boal, R., 1992. An effect of ascorbic acid on delayed-onset muscle soreness. *Pain* 50, 317–321.
- Kirsch, D.L., May–June 1996. A basis for understanding micro-current electrical therapy, part I. *The American Chiropractor*, 30–40.
- Kirsch, D.L., Sept–Oct 1997. How to achieve optimal results using microcurrent electrical therapy for pain management, part II. *The American Chiropractor*, 12–14.
- Kirsch, D.L., Lerner, F.N., 1998. Electromedicine the other side of physiology. In: Weiner, R. (Ed.) *Pain Management: a Practical Guide for Clinicians*, fifth ed., Vol. 2. CRC Press LLC, Boca Raton, Florida (Chapter 55).
- Kraemer, W.J., Bush, J.A., Wickham, R.B., Denegar, C.R., Gomez, A.L., Gotshalk, L.A., Duncan, N.D., Volek, J.S., Newton, R.U., Putukian, M., Sebastianelli, W.J., 2001. Continuous compression as an effective therapeutic intervention in treating eccentric-exercise-induced muscle soreness. *Journal of Sport Rehabilitation* 10, 11–23.

- Lambert, M.I., Marcus, P., Burgess, T., Noakes, T.D., 2002. Electromembrane microcurrent therapy reduces signs and symptoms of muscle damage. *Medicine and Science in Sports and Exercise* 34, 602–607.
- Lanier, A.B., 2003. Use of non-steroidal anti-inflammatory drugs following exercise-induced muscle soreness. *Sports Medicine* 33, 177–186.
- Lee, K.A., Kieckhefer, G.M., 1989. Measuring human responses using visual analogue scale. *Western Journal of Nursing Research* 11, 128–132.
- Lightfoot, J.T., Char, D., McDermott, J., Goya, C., 1997. Immediate post exercise massage does not attenuate delayed onset muscle soreness. *Journal of Strength and Conditioning Research* 11, 119–124.
- Manley, T., 1994. *Microcurrent Therapy Universal Treatment Techniques and Applications*. Manley and Associates, Corona, California.
- Mattacola, C.M., Perrin, D.H., Gansneder, B.M., Allan, J.D., Mickey, C.A., 1997. A comparison of visual analogue scale and graphic rating scales for pain and intensity following DOMS. *Journal of Sport Rehabilitation* 6, 38–46.
- McIntyre, D.L., Reid, W.D., McKenzie, D.C., 1995. Delayed muscle soreness: the inflammatory response to muscle injury and its clinical implications. *Sports Medicine* 20, 24–40.
- McHugh, M.P., Connolly, D.A.J., Eston, R.G., Gleim, G.W., 1999. Exercise-induced muscle damage and potential mechanisms for the repeated bout effect. *Journal of Sports Medicine* 27, 158–170.
- McMakin, C., Gregory, W., Philips, T., 2005. Cytokine changes with microcurrent treatment of fibromyalgia associated with cervical spine trauma. *Journal of Bodywork and Movement Therapies* 9, 169–176.
- McMakin, C., 2004. Microcurrent therapy: a novel treatment method for chronic low back myofascial pain. *Journal of Bodywork and Movement Therapies* 8, 143–153.
- McMakin, C., 1998. Microcurrent treatment of myofascial pain in the head, neck and face. *Topics in Clinical Chiropractic* 5, 29–35.
- Mercola, J.M., Kirsch, D., 1995. The basis for microcurrent electrical therapy in conventional medical practice. *Journal of Advancement in Medicine* 8 (2). <http://therapyproducts.net> Available on-line from: pages not numbered.
- Myles, P.S., Troedel, S., Boquest, M., Reeves, M., 1999. The pain in visual analogue scale: Is it linear or nonlinear? *Anesthesia and Analgesia* 89, 1517–1520.
- Newman, D.J., McPhail, G., Mills, K.R., Edwards, R.H., 1983a. Ultrastructural changes after concentric and eccentric contractions on human muscle. *Journal of Neurological Science* 61, 109–122.
- Newman, D.J., Mills, K.R., Quigley, B.M., Edwards, R.H.T., 1983b. Pain and fatigue after concentric and eccentric contractions. *Journal of Clinical Science* 64, 55–62.
- Nosaka, K., Clarkson, P.M., 1995. Muscle damage following repeated bouts of high force eccentric exercise. *Medicine and Science in Sports and Exercise* 27, 1263–1269.
- Nosaka, K., Clarkson, P.M., 1996. Changes in indicators of inflammation after eccentric exercise of the elbow flexors. *Medicine and Science in Sports and Exercise* 28, 953–961.
- O'Grady, M., Hackney, A.C., Schneider, K., Bossen, E., Steinberg, K., Douglas, J.M., Murray, W.J., Watkins, W.D., 2000. Diclofenac sodium (voltaren) reduced exercise-induced injury skeletal muscle. *Medicine and Science in Sports and Exercise* 32, 1191–1196.
- Oschman, J., 2000. *Energy Medicine, The Scientific Basis*. Churchill Livingstone, Edinburgh.
- Petrofsky, J., Schwab, E., Cuneo, M., George, J., Kim, J., Almalty, A., Lawson, D., Johnson, E., Remigo, W., 2006. Current distribution under electrodes in relation to stimulation current and blood flow: are modern electrodes really providing the current distribution during stimulation we believe they are? *Journal of Medical Engineering and Technology* 30, 368–381.
- Proske, U., Weerakkody, N.S., Percival, P., Morgan, D.L., Gregory, J.E., Canny, B.J., 2003. Force-matching errors after eccentric exercise attributed to muscle soreness. *Clinical and Experimental Pharmacology and Physiology* 30, 576–579.
- Rowley, B.A., McKenna, J.M., Wollcott, L.E., 1974. The use of low level electric current for the enhancement of tissue healing. *Biomedical Scientific Instrumentation* 10, 111–114.
- Sayers, S.P., Knight, C.A., Clarkson, P.M., van Wegan, E.H., Kamen, G., 2001. Effects of ketoprofen on muscle function and sEMG after eccentric exercise. *Medicine and Science in Sports and Exercise* 33, 702–710.
- Sellwood, K.L., Brukner, P., Williams, D., Nicol, A., Hinman, R., 2007. Ice-water immersion and delayed-onset muscle soreness: a randomized controlled trial. *British Journal of Sports Medicine* 41, 392–397.
- Stauber, W.T., Clarkson, P.M., Fritz, V.K., Evans, W.J., 1990. Extracellular matrix disruption and pain after eccentric muscle action. *Journal of Applied Physiology* 69, 868–874.
- Taleg, T.S., 1973. Residual muscular soreness as influenced by concentric, eccentric and static contractions. *Research Quarterly* 44, 458–469.
- Tiitus, P.M., Shoemaker, J.K., 1995. Effleurage massage, muscle blood flow and long-term post-exercise strength recovery. *International Journal of Sports Medicine* 16, 478–483.
- Warren, G.L., Jenkins, R.R., Packer, L., Witt, E.H., Armstrong, 1992. Elevated muscle vitamin E does not attenuate eccentric exercise-induced muscle injury. *Journal of Applied Physiology* 72, 2168–2175.
- Wilmore, J.H., Costill, D.C., 2004. *Physiology of Sport and Exercise*, second ed. Human Kinetics, Leeds.
- Yamaguichi, T., Ishii, K., 2005. Effects of static stretching for 30 seconds and dynamic stretching on leg extension power. *Journal of Strength and Conditioning Research* 19, 677–683.