THE EFFECT OF MICROCURRENT STIMULATION ON
EXERCISE-INDUCED MUSCLE SORENESS IN A HUMAN INJURY MODEL

by

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Abstract

Exercise-induced muscle soreness can present several complications including an increased intensity of pain or discomfort, edema, a decrease in range of motion, and loss of eccentric muscle strength. The purpose of this study was to determine the efficacy of prescribed microcurrent stimulation as an effective electrotherapeutic modality for improving the symptoms of muscular soft tissue injury.

Forty-eight healthy, sedentary, male subjects of university age (18 – 35 years) were randomly divided into four experimental groups: two functional groups (Group A - .3 / .7 Hertz (Hz) and Group B - .3 / 18 Hz), a control group (C), and a sham group (D). They completed 300 eccentric contractions on the Kinetic Communicator (KinCom) using the non-dominant leg to create the exercise-induced muscle injury model. The subjects were tested and data were collected at 0 hours (pre-exercise), immediately post-exercise, 6 to 8 hours post-exercise, and at 24, 48, 72, and 96 hours after the exercise bout in the mornings and afternoons. The four variables were tested and analyzed for the effectiveness of the prescribed microcurrent treatment program, to demonstrate significant differences (p < 0.05) among the exercise-induced muscle injury subject groups, and to show if there were any significant frequency intensity effects from the microcurrent stimulation devices.

Statistical analysis revealed non-significant differences (p > 0.05) for the group main effect and group interaction effect of all the tested variables. However, there was a significant
difference (p < 0.05, p = 0.0001) for all the groups as shown by an effect due to changes over time.

The results of this investigation suggest that the prescribed microcurrent treatment had no therapeutic effect on exercise-induced muscle soreness nor did it show a frequency intensity effect by either microcurrent stimulation unit.
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Chapter 1

Introduction

Injury to active muscles is a common occurrence in sports, exercise, and daily living. The most common cause of muscular injury, a mechanical stressor, is believed to induce a disruption of the contractile tissue elements especially after eccentric exercise or lengthening of muscle while under specific tension (4,5,57,77). It is postulated that the mechanical muscle stressor is followed by a biochemical stressor that seems to induce a period of perceived pain that varies in both intensity and duration (4,5,57,77). This is believed to be a result of exercise – induced muscle soreness otherwise known as delayed onset muscle soreness (DOMS) and the acute inflammatory response (4,5,57).

Eccentric muscle contractions are more apt to result in myofibrillar damage than isometric or concentric contractions because high specific forces appear to occur in the active musculature during this type of contraction (5,25,30,32,42,57,76,81). Also, eccentric contractions possibly produce greater forces per cross-sectional area of active muscle fiber because of a relatively small number of motor units being recruited compared to isometric and concentric contractions (5,25,30,32,42,57,76,81).

The musculoskeletal system, due to the high forces exerted upon it, undergoes morphological changes such as cytoskeletal or myofibrillar damage that have been shown to trigger
mobilization of some aspects of the acute inflammatory response (5,57,76). The morphological changes are suspected to create DOMS; cause a marked release of cellular infiltrates such as fluid, plasma proteins and leukocytes into the circulation due to increased microvascular permeability; and performance decrements of a decrease in the inherent maximal force capability of the muscle (4,30,57,76). These responses are indicated by the cardinal signs and symptoms of acute inflammation: redness, swelling, heat, muscular weakness, localized tenderness, decreased range of motion, and discomfort or pain (5).

Physical activity of any intensity level or duration involving eccentric muscle action or deceleration activities to which an individual is unaccustomed will result in a common phenomenon known as exercise-induced muscle soreness or delayed-onset muscle soreness (DOMS) (4,5,57,77). It is described as the sensation of discomfort or pain combined with local tenderness and stiffness which increases in intensity peaking 24 to 72 hours after the cessation of the muscular exertion (5,25,30,76,81). Delayed-onset muscle soreness is not to be confused with exertional pain (pain during exercise) or pain felt following exercise to fatigue (77). There is no evidence of long term damage or reduced muscle function because the sensation of DOMS subsides 5 to 7 days post-exercise which seems to follow the time course of the acute inflammatory stage (5,25,30,42,76,81).

Treatment to control soft tissue injuries and the eventual acute inflammation caused from physically strenuous activity include rest, ice, ultrasound, physiotherapy, and non-steroidal anti-inflammatory drugs. Hence, the goal in sports medicine is to suppress or control pain and
inflammation so that a patient's return to daily activity may be hastened. A relatively new treatment modality for exercise-induced muscle soreness is microcurrent stimulation. Microcurrent therapy is the application of electrical energy to human tissue using intensities that are below the threshold for motor or sensory perception (41); therefore, this energy is unable to evoke muscular contractions. These devices use a combination of low voltage, low frequency, and unique waveforms to maintain desired subsensory current levels for a therapeutic effect.

Proponents of microcurrent therapy believe that by reducing the current to a subsensory level the body may more efficiently accept the electrical energy into its own electrophysiological system to enhance and promote tissue repair and regeneration (67,68). It is speculated that microcurrent stimulation appears to significantly improve the body’s healing process by increasing cellular physiology and growth, and by its ability for biochemical osmosis (67,69).

Based on the literature, most of the microcurrent stimulation research has been conducted on induced wounds in animals or ischemic dermal or sub-dermal ulcers in animals and humans (67). However, researchers recently have studied the effects of microcurrent stimulation on soft tissue elements such as ligaments and tendons with positive results (55,67). The proposed study will determine the outcome of microcurrent therapy on muscular soft tissue injury.
**Purposes of the Investigation**

The purpose of this study was to determine how varying applied frequencies (.3 / .7 Hertz (Hz), .3/18 Hz, sham, control) would affect the therapeutic effects of microcurrent stimulation.

The second purpose of the study was to determine if microcurrent stimulation reduced muscle soreness perception and edema, and improved range of motion and eccentric muscle strength during recovery associated with exercise-induced muscle soreness in a human injury model.

**Significance of the Study**

Exercise – induced muscle soreness is a very common injury resulting from unaccustomed daily physical activity. The aim of this study was to determine if microcurrent stimulation is an effective treatment modality for the management of pain, edema, range of motion and force deficits associated with DOMS. In the literature, there is limited research evidence of microcurrent stimulation on muscle damage; its efficacy has yet to be significantly proven as a treatment modality.
Statement of the Problem

This study was designed to investigate the effectiveness of cumulative microcurrent stimulation as a means of significantly treating DOMS that is secondary to an eccentric muscle activity. It was also intended to test whether varying frequency outputs affect the modality’s therapeutic effect.

Delimitations

(1) Sample selection; healthy, sedentary male subjects between 18 to 35 years of age (N = 48; n = 12 per group), two groups of functional microcurrent devices (Group A = .3 / .7 Hz, Group B = .3 / 18 Hz), a control group (Group C), and a sham group (Group D).

(2) Use of the Kinetic Communicator (KinCom) dynamometer tests the subject’s muscle strength through a specific range of motion while performing isolated knee extensions.

(3) The use of eccentric exercise protocol allows the researcher to control the insult of the injury.

(4) The prescribed microcurrent treatment program is designed for application to soft muscle tissue injury.
(5) The use of the reliable visual analogue scale (VAS) is to record the patient’s perceived muscle soreness over the quadriceps musculature.

(6) The circumference measurement of the quadriceps (QCM) evaluates the girth of the thigh that includes skin, fat, muscle, bone, and possible edema, and etc. occurring within the musculature.

(7) The flexometer goniometric measurement records the active range of motion (ROM) about the injured knee.

Limitations

(1) The sample selection of men and inclusion criteria were chosen to ensure group homogeneity and high force production results. However, this limits what can be inferred about the population who are women and of varying ages.

(2) Selection of the isokinetic testing device restricts the velocity and ranges of motion about the knee as it naturally functions. The performance of isolated knee extension is a rare occurrence in sport and daily living activities.
(3) The prescribed microcurrent treatment program allows the same monitoring of the patient’s progress throughout the five day study period from start to finish. Therefore, this characteristic limits how appropriate the treatment was for each individual patient.

(4) This study may not apply directly to soft tissue injuries such as contusions, lacerations, abrasions, or injuries such as sprains or fractures.

(5) The VAS provides quantitative information regarding changes in intensity of perceived muscle soreness, however, subjective information considerably varies.

(6) The landmarks for the circumference measures may vary due to individual stature differences.

(7) The active range of motion may be influenced by one’s flexibility around the knee.

(8) The power analysis of the study is based on differences that were assumed and not directly determined through the literature. Based on sample size calculations, given the lack of ANOVA tables, the power of this study was at least 0.80 using 12 subjects per group at a significance level of $p < 0.05$ and a Cohen’s $d$ for effect size at 1.0.
Hypotheses

It was hypothesized that the prescribed microcurrent treatment program would render these results:

(1) significantly reduce perceived muscle soreness scores over the five day testing period;

(2) significantly reduce edema over the study period;

(3) significantly improve range of motion about the knee;

(4) significantly improve the eccentric muscle strength during the recovery of DOMS;

(5) the prescribed microcurrent stimulation would be significantly more effective than the control and sham groups in decreasing exercise – induced muscle soreness and edema, and improving the range of motion and eccentric muscle strength; and

(6) significantly demonstrate a frequency intensity effect from the prescribed treatment.
Eccentric Muscle Contraction

Eccentric muscle contractions, a synonymous phrase for negative work or deceleration activity, occurs when the load on the muscle is greater than the force developed by the muscle; therefore, producing a lengthening stretch of the muscle (34). The stretch that lengthens the muscle may only be experienced after the muscle produces additional tension (77). The potential for injury is greater during eccentric activities. It has been proposed that the increased probability of injury during an eccentric muscle action may be due to a greater than average force development by the activated musculature during lengthening (34).

During an eccentric contraction, the force developed is believed to be approximately two-fold greater than that produced by a maximal isometric (34). It has been suspected that injury of muscle from repeated eccentric action somehow involves the mechanical forces of stretch (77). It is suggested that the mechanism of injury is due to increased tension per individual cross-bridges causing a mechanical disruption of the ultrastructural elements within the muscle fibers (34). Their working hypothesis is that, during lengthening contractions, some sarcomeres maintain length, whereas other sarcomeres are stretched beyond overlap and are mechanically injured (34).
Although eccentric activities generate high forces in the muscle fibers, another proposed mechanism that may induce damage considers not only the high tension but also an active strain upon the musculature. This active strain may exceed the limits of the cytoskeletal framework causing injury (36). It is also reported that muscle fiber type may be a determining cause for muscle damage (36). It is suggested that specific damage can be noticed in the type 2B fast glycolytic fibers, thus hypothesizing that muscle injury may be a result of the muscle fiber oxidative capacity (36). It is believed that type 2 fibers might be more susceptible to stretch induced injury because of a less developed endomysium than type 1 fibers (77).

The pathophysiological mechanism underlying the injury is not well understood. There is some evidence that eccentric exercise creates cytoskeletal and myofibrillar damage to muscle cells (4). The sequence of events includes two mechanisms proposed to explain how eccentric exercise initiates the damage. These causative factors are primarily a mechanical injury followed by a secondary injury involving a metabolic or biochemical insult (4,30,34,36,77).

The initial mechanical injury is believed to be a result of direct myofiber damage that creates the ultrastructural damage to the muscle cells (13,34,77). These mechanical stressors include: the cross-bridge theory of a production of great tension per cross-sectional area of the active musculature (5,30,77); the possibility of fewer fibers being recruited to produce large forces (5,25,30); Z-band alterations of broadening, streaming, or total disruption (4,30,36); sarcolemmal disruptions (30,36); actin-myosin bond disruption (77); ATP dissociation (77);
increased mitochondrial volume due to edema (13,30); a swollen sarcotubular system (13,36,77); and cellular damage of intracellular protein loss (4,13,30,36). Signs of cytoskeletal and myofibrillar injury seem to begin immediately after the performance of an eccentric activity and continue for a period of days following the cessation of all activity. The delayed myofiber damage is known as the metabolic or biochemical stressor (4,30,36,77).

This secondary mechanism constitutes a specific intracellular enzyme release and process that further degrades the contractile and cytoskeletal components of the cell (36). The factors cited include: the efflux of intracellular proteins such as creatine phosphokinase (CPK) and lactate dehydrogenase (LDH) from the muscle fibers into the extracellular space (4,13); the influx of calcium causing muscle enzyme release through activation of phospholipase A2 (4,13); lysosomal disruption; connective tissue disruption; free oxygen radical production; affected ATP energy sources and high cellular temperature environment (4,13,25,30,36). It is also postulated that this secondary biochemical injury may be due to acute inflammatory cellular infiltrates like neutrophils, macrophages, and phagocytes that have been found to migrate to the injury site and begin the process of tissue repair (34,57,76,81).

Microscopic evidence of the morphological damage to the ultrastructure in the post-exercise period suggests that it is not permanent. The delayed cytoskeletal and myofibrillar injury usually peaks about 2 to 3 days after the cessation of eccentric exercise (34,36). Coinciding with this is evidence of exercise-induced muscle soreness, a decreased range of motion, and a decrease in the maximal voluntary force capability of the muscle (5,34,36).
In untrained individuals following the completion of an exhaustive eccentric exercise protocol, a decrease in the eccentric force or torque can be immediately detected (34). Initially, a measurable loss of range of motion is evident especially before soreness is experienced (77). During the first few hours, the decrease in maximal force is a function of both fatigue and injury (34). Force deficits peak approximately 50% or lower from days one to three after the exercise bout. Beyond the third day, there is a display of a steady force recovery which can reach force values of about 80% of the pre-exercise eccentric torque level (34). It is reported that maximum voluntary force of the quadriceps returned to pre-exercise levels within 24 hours whereas other studies have found pre-exercise values for eccentric torque recovery within a week of the exercise bout (57).

**Exercise Induced Muscle Soreness**

Exercise induced muscle soreness is described as the sensation of discomfort or dull aching pain combined with local tenderness and stiffness that follows a bout of unaccustomed muscular exertion (5,25,30,57,76). The debilitating soreness exacerbates over the first 24 hours peaking usually from 24 to 72 hours, while subsiding within 5 to 7 days after the cessation of exercise particularly following any eccentric activities (5,25,30,76,81).

Signs associated with DOMS become more apparent between 24 to 72 hours. Individuals experience a reduced range of motion and flexibility, and their muscles are sensitive to
movement or palpation (5,42,57,76). Sensation may vary from slight stiffness or tenderness in the muscle that disappears with routine daily activity to severe pain that interferes with mobility (5,42,57,76).

The muscle damage which has progressed in the post-exercise period is not associated with long term damage or reduced muscle function (5). Tenderness is often localized at the possible injury site; the site of perceived soreness. The soreness is believed to begin in the distal third portion of the muscle at the musculotendinous junction and eventually spreads to the center of the muscle by 48 hours (5,42,57,76). Some subjective evidence suggests that the distal muscle soreness migrates from the lateral and medial sides to the mid muscle region over the time span (77).

Newham and colleagues as reported by Armstrong (5) suggest that the spreading discomfort or pain could be due to muscle pain receptors being mostly concentrated in the regions of tendons and connective tissue of the muscle, to a localization of the damage to the distal musculotendinous region, or to a combination of such factors. One study performed on rat muscle after prolonged downhill running revealed significantly greater incidence of muscle injury to the distal third of the muscle upon detailed histological examination (5). Newham et al. cited by Armstrong (5) also suggested that localization in the distal musculotendinous region may occur because the angles of the fibers to the long axis of the muscle are greatest in
these regions, thereby, increasing the susceptibility of the muscle fibers to such a mechanical trauma.

**Exercise – Induced Muscle Soreness and Inflammation**

Exercise - induced muscle soreness is believed to be the body's response to an exercise-induced injury. A possible mechanism underlying DOMS may be inflammation especially involving the acute inflammatory stage. A review by Smith (76) suggested that similarities of observed symptoms like pain, swelling, and loss of function in DOMS and acute inflammation were nearly the same and are due to the body's response to a traumatic injury. It is also stated that since the body responds to all forms of tissue injury by activating the inflammatory response, there is no reason to believe that a separate response has evolved to deal with injury incurred during unaccustomed eccentric exercise (76).

Exercise - induced muscle soreness has the ability to cause a local inflammatory response in exercised muscle. This inflammatory response is characterized by movement of fluid, plasma proteins, and leukocytes into tissue in the response to injury, infection, or antigens (57). The purpose of the response is healing. It promotes clearance of damaged tissue, eliminates microbial invaders, and prepares the tissue for repair (42,57,76). Inflammation is considered an extremely complex defensive reaction to injury. As a result of cell damage, chemical mediators and chemotactic factors are liberated locally (26,57).
Acute inflammation, lasting no longer than a few days to a week, is the first response of the body to tissue injury. Classical signs are redness (erythema), swelling (edema), heat, tenderness and muscular weakness, a decreased range of motion, and discomfort or pain. The process of acute inflammation is associated with vascular and cellular responses (76). These responses or changes are a result of the interaction of various inflammatory mediators.

The vascular response is a local change that occurs and is associated with the classical signs of inflammation (57,76). It is phasic, involving rapid changes in blood flow. Initially, vasoconstriction lasting 5 to 10 minutes occurs followed by a period of vasodilation and increased permeability (76). The cellular response is a systemic reaction causing production of a variety of plasma proteins (57). It involves an invasion of two types of white blood cells primarily neutrophils and monocytes (5,25,30,57,76,81).

Within a few hours after tissue injury, the number of circulating neutrophils increases due to cellular chemotaxis. The neutrophils begin to aggregate at the site of the disruption reaching peak concentrations between 1 to 6 hours post-injury depending on the intensity of the injury (76,81). These cells aid in the digestion of the necrotic tissue by the release of lysosomal enzymes (76,81). As neutrophil concentrations decrease, monocytes migrate into the site via the circulation.

Monocytes mature into macrophages and become predominant at all stages of acute inflammation following the first 9 to 12 hours. They are responsible for the removal or
phagocytosis of neutrophils, and other foreign bodies (57,76,81). In the meantime, macrophage concentrations continue to increase quickly from 48 to 72 hours. While the phagocytic action is taking place, further signals for later chemotaxis of cells from the circulatory system is believed to be under way (81). Satellite cells are thought to migrate to the injury site and initiate the repair and regeneration of new muscle fibers for healing (81).

It is also noted with the time period of the injury that the discomfort or pain experienced peaks between 24 to 72 hours. The cause of the soreness that appears some time after the exercise is still undetermined (5).

**Delayed Pain Sensation**

The delayed pain sensation may be associated with DOMS and / or the acute inflammation process. The stimuli that acts to cause the delayed pain is not known, but it is possible one or a combination of chemical, mechanical, or thermal factors could conceivably be involved in producing pain in the muscles following exercise (5). The proposed action involves the cyclooxygenase and lipoxygenase systems in which specific synthetases convert or metabolize arachidonic acids to prostaglandins, leukotrienes, and oxygen-free radicals (10,26,27). Arachidonic acid is derived from the action of phospholipase on cell membrane phospholipids that are released when the cell membrane is disrupted by a certain stimuli (27).
A biochemical reason for this event was postulated to be the result of chemical substances that could accumulate in a region of damaged tissue and potentially cause the sensation of pain (5). Certain intermediates and end products of the arachidonic acid cascade, in combination with the local release of inflammatory mediators like histamine, serotonin, bradykinin as well as potassium may be able to activate the free nerve endings of nociceptors thus contributing to pain (5,26,30). Also the invasion of cellular infiltrates, namely macrophages, in and around the injury site will serve to augment the inflammatory effects of the mediators (5,76).

The sensation of pain may be caused by the synthesis of the prostaglandin E series, especially PGE2, from the stimulated macrophages (5). It is proposed that PGE2 is released in large amounts that sensitize local nociceptors within the damaged tissue over the initial 24 hours after the completion of the exercise bout (4,27,76). Furthermore, edema resulting from an increased vascular permeability of small blood vessels and exudate about the injury site may create localized pain from an increase in intramuscular pressure and/or increased pressure on local nociceptors (76).

Mechanical stimuli are elicited by an eccentric contraction that damages the muscle fibres by over-exertion on the skeletal-musculosystem. This accompanies an elevated temperature within the muscle because of the exercise intensity (4,76). These are associated with edema and the inflammatory process that could assumingly activate the nociceptors in the muscles, therefore creating a sense of pain or DOMS (5).
The sensation of pain in skeletal muscle is transmitted by myelinated group III and unmyelinated group IV afferent fibres (5). These fibres are found throughout the region of connective tissue particularly the musculotendinous junctions and the area of arterioles and capillaries (5). The larger myelinated group III fibers are believed to transmit sharp, localized pain, whereas the group IV fibers carry dull, diffuse pain (5). The group IV afferent fibers due to the free nerve endings in muscle are sensitive to the various stimuli and are capable of responding to several types of prolonged noxious stimuli (5). In addition, it is speculated that exercise-induced soreness may be modulated at the spinal level by the reticular activating system or by the sensory cortex (30).

Pain is usually experienced during attempted movement, contraction, or palpation of the injured region. Therefore, it is suspected that pain during DOMS becomes a protective mechanism to encourage immobilization during the repair and healing period (5,76).

Microcurrent Stimulation

Microcurrent stimulation refers to the introduction of an electrical stimulus that appears to be of similar amplitude to the body's bioelectrical environment (68). Microcurrent stimulation has a current amplitude designed not to excite motor and / or sensory nerves unlike other electrotherapeutic modalities (41,60,67).
When tissue becomes injured, the body's bioelectrical impulses lack the drive current to overcome the increased electrical impedance barrier caused by the traumatized tissue (67). It is believed that microcurrent therapy overcomes the electrical resistance, known as the "current of injury," by allowing a synthesized bioelectric stimulus to affect the biological homeostasis surrounding the traumatized site and hence augment the healing process (16,67). The microcurrent modality appears to significantly enhance the body's healing process by increasing the fuel for cellular metabolism (69).

Substantial evidence from research demonstrated that microcurrent stimulation increased levels of collagen formation, bettered structural remodelling and cellular organization, increased the strength of the connective tissue, and enhanced the ability to hasten recovery from injury (60). Nessler and Mass (61) microelectrically stimulated rabbit tendons of which results indicated an increased cellularity, increased epitenon proliferation and bridging of the gap, and the appearance of delicate new collagen fibrils from increased collagen synthesis. This study showed a 91% higher proline uptake than controls after 7 days, while hydroxyproline activity was increased by 255% versus controls. They concluded that tenoblastic repair was enhanced by microcurrent stimulation (61).

Litke and Dahners (55) used three different currents to determine their effect upon a rat medial collateral ligament section. After the microcurrent stimulation, the rupture or breakpoint force was monitored. The group treated with a range of 1 to 20uA (microamps) showed statistically significant improvements in maximum force and stiffness in comparison
with the other two groups. This study supports the hypothesis that early healing of ligaments can be enhanced by microcurrent stimulation. Other studies have recently reported the effects of microcurrent stimulation on soft tissue elements such as ligaments and tendons; the researchers found quantitative and qualitative effects with microcurrent stimulation (55,67).

"Current of Injury"

The "current of injury," first noted by Galvani as an electric current emanating from vertebrates when suffering soft tissue damage, flowed from the surrounding uninjured tissue into the injury site (41,69). This was believed to be the body's attempt to restore the electrical potentials that were lost due to the trauma (41,69). Researchers who have been able to substantiate the "current of injury" are Robert Becker and Bjorn Nordenstrom. They have verified that the body continuously generates low level direct currents during healing (60).

The Body Electric Theory

Becker (50) has conceptualized the existence of a direct current (DC) system controlling tissue healing. According to Becker, when the electrical balance of the body is disturbed by an injury, the resulting shift in current flow or the DC surface potentials stimulate the DC system (50). The DC system acts as a biological semiconductor that transmits the "current of injury" into the nervous system and the changes in surface potential record the transmission of the injury signals to the nervous system (50). It is further postulated that the "current of
injury" is conducted via the Schwann cell sheaths in the periphery, glial cells in the central nervous system, and the satellite cells in the dorsal root ganglion triggering repair and regeneration (50). As healing continues, the DC potential difference will approach the normal electrical balance relative to the surrounding tissues (16).

*The Bioelectric Battery*

Nordenstrom (50) theorized that the body possesses bioelectrical circuits contained within the vasculature. He has proposed that bioelectricity is conducted through five main components that may be found in any vascularized part of the body. These are: (i) insulating walls of blood vessels, (ii) conducting intravascular plasma, (iii) insulating tissue matrix (possibly including the lymph vessels), (iv) conducting interstitial fluid and (v) electrical junctions (transcapillary junctions) for redox reactions (50).

Nordenstrom (50) reportedly has measured a relatively higher electrical resistance present in the walls of the large blood vessels and a relatively lower electrical resistance in plasma and interstitial fluids, giving rise to a potential voltage gradient. The vessel walls in this bioelectrical circuit act as electrically conducting, insulating cables that carry plasma (the conducting media) and separate it from the surrounding conducting media (the interstitial fluid) except at its transcapillary junctions (the naturally occurring electrodes in the biocircuit) (50).
The capillary cell membranes act as naturally charged electrodes that allow ions to move through the cells via gates and vesicles; additional ions flow between the cells through pores (50). This local ion flow stops when excess electrons cross enzyme bridges in the capillary walls, closing the pores and gates, thereby closing the local circuit (50). This occurrence creates a long distance bioelectrical circuit in which the ions flow. The capillary cell membranes, therefore, appear to be the key component in switching from local ion flow across the capillary membranes to a long distance ion flow down the capillary walls (50).

Nordenstrom (50) further states that an accumulation of charge (excessive electrons) can be caused by soft tissue injury or even normal muscle use. The accumulation of charge may constrict arterial capillaries, switching the current on. However, venous capillaries do not constrict in an electrical field; therefore, ions and charged cells can migrate through the pores of a leaky venous capillary near the injury (50). Because the polarity of the electrical potential from an injury changes, charged cells and ions necessary for healing may ebb and flow as changes take place in the electrical insulation properties of the capillary membranes (50).

The Gate Control Theory

Proposed applications of electrotherapeutic devices not only include the promotion of healing tissue but also the modulation of pain. The gate control theory of Melzack and Wall (69) is the best explanation for use of high intensity milliampere stimulation. According to Melzack
and Wall (69), high intensity milliampere stimulation appears to block gates to the corresponding nerve impulses. Specifically, it would appear that stimulation of the beta sensory neurons, which conduct more rapidly than the smaller, pain transmitting C-fibers, inhibits the transmission of pain signals in the substantia gelatinosa in the spinal cord (69). The beta sensory input essentially floods the circuits and prevents pain signals from the C-fibers from getting through (69).

This presynaptic feedback inhibition may also be enhanced by the secretion in the central nervous system of endorphins and enkephalins, the body's naturally occurring narcotics. Electrical stimulation of the high intensity type must be sufficiently irritating to the tissues to trigger the protective response of these natural opiates (69). Such pain relief may last for several hours, but unfortunately such effects represent only a temporary masking of the pain without any long-lasting cumulative effects and without any direct effects on the body's healing process (69). However, with microcurrent stimulation, it is proposed that instead of masking the pain of injury there may be a way to initiate the body's natural healing process via the bioelectrophysiological system (69).

The benefit of microcurrent therapy claims to be the cumulative treatment effects on pain. Those using microcurrent therapy are familiar with the next-day carry over, whereby a patient may not notice any immediate analgesia but the next day reports remarkable subjective improvement corroborated by objective examination revealing reduced pain with palpation, diminished swelling, normalization of skin colouration and improved range of motion (68).
Meyer and colleagues as reported by Picker et al. (69) studied the cumulative effects of microcurrent in which there were 16 treatments over an initial eight week period. The microcurrent stimulation was compared to a placebo of microcurrent stimulation. It was found that pain significantly decreased 40% in microcurrent stimulated patients versus the placebo group. They hypothesized that temporary hyperstimulatory analgesia between treatment and placebo groups would wear off and reduce to a degree of pain that was equalized for the groups after discontinuation of the microcurrent therapy (69). It was concluded that after a second eight week period for follow-up results the pain relief gained by the microcurrent stimulation was maintained at 75% compared to 6% of placebo (69). This report however does not allude to the microamperage that was used to gain the results.

The Chemiosmotic Theory

Mitchell’s chemiosmotic theory states that the source of energy needed to phosphorylate adenosine diphosphate (ADP) and adenosine triphosphate (ATP) comes from the energy stored as a proton gradient across the mitochondrial membrane rather than from a chemical intermediate containing a high energy phosphate bond (50). The chemiosmotic theory explains why electrical energy can stimulate repair of tissues compromised by hypoxia, injury, or nutritional deficiency (50).
The delivery of a subsensory microcurrent is believed to produce a proton gradient when electrons at the cathode react with water molecules to form hydroxyl ions, and acid and protons are formed at the anode (21,50). As a result, a proton gradient and voltage gradient are established across the intervening tissues between the electrodes. The influence of the electrical field and proton concentration difference produce a proton current that moves from anode to cathode (21,50). Since the rate of proton formation at the anodic interface is equal to the rate of proton consumption at the cathodic interface, the net pH of the system, medium, and tissue remains undisturbed (21). As the migrating protons reach the mitochondrial membrane-bound H⁺-ATPase, ATP is formed (21,50). The increased ATP production stimulates amino acid transport and these two factors both contribute to increased protein synthesis (21,50).

Cheng et al. (21) studied the effects of electric currents of various intensities on ATP generation, protein synthesis, and membrane transport, all of which are critical to the healing process. Results showed that constant microcurrents from 100uA to 500uA increased ATP generation in rat skin by almost 500% and transported amino acid, critical for delivering nutrients intracellularly and extruding metabolic wastes extracellularly, increased 30% to 40% above control levels (21). More intense stimulation within the milliampere range (1000uA to 5000uA) in which 1 milliampere equals 1000 microamperes (1mA = 1000uA) caused the ATP generation to drop below baseline control levels. It was concluded that a high stimulation greater than 500uA retarded ATP generation due to reduced amino acid uptake (21). Hence, 500uA was the optimum current for amino acid production. Similar results were found with
protein synthesis. Protein synthesis was highest with 50uA to 1000uA but significantly stimulated as low as 10uA (21). Higher stimulation currents greater than 1000uA or a milliamp inhibited protein synthesis (21). The a-aminoisobutyric acid uptake was reduced by 20% to 73% and inhibited protein synthesis by as much as 50%. It was concluded that a minimum current intensity of at least 50uA is necessary to obtain a maximal stimulatory effect on protein synthesis (21).

A phenomenon that is said to occur in vitro studies is galvanotaxis or the attraction of cells to the anode (positive pole) or cathode (negative pole) (35). Studies of cell cultures have shown that electrical fields can influence the migratory, proliferative, and functional capacity of cells involved in the healing process (35). The galvanotaxic effect involves electrical fields that form around electrodes attracting specific types of ions near the different poles (12). Under the anode it is reported that there is enhanced ion transport, fibroblast migration, protein synthesis and decreased vascular congestion. Under the cathode there is an increased migration of epidermal cells, macrophages, neutrophilic leukocytes and decreased bacterial counts in a region where infection or inflammation is present (12,35). An underlying assumption of electrical stimulation for injured tissue is the application of an exogenous microcurrent the therapeutic current should facilitate the injury current, reverse the natural resting electrical field that occurs after tissue injury, and be expected to mimic the body's bioelectrical currents to enhance the tissue healing process (12,35). A study done on the healing of tenotomized rat achilles tendons using load - to – breaking measurements by low
intensity pulsed galvanic current found that after 2 weeks tendons treated with an anodal current were much stronger than those treated by a cathodal current or to the controls (66).
Sample Description

Forty-eight university aged male subjects (age = 20.29 ± 3.26 years; height = 178.04 ± 6.39 centimetres; weight 79.28 ± 13.9 kilograms) were randomly assigned to one of four experimental groups (n = 12). Subjects accepted for the study were given a consent form to read and sign which was set in accordance with the standards of the Dalhousie University Clinical Screening Committee for Research Involving Human Subjects. The study was conducted at the Physiotherapy Laboratory in the Forrest Building on the Dalhousie campus.

The investigation included four experimental groups: two treatment groups (Group A = .3 / .7 Hz, Group B = .3 / 18 Hz); a control group (Group C); and a sham group (Group D); of healthy, sedentary or untrained male subjects of university age (18 to 35 years) who did not regularly participate in physical activity more than 6 hours per week on average. The study was double-blinded for the subject and researcher to the type of microcurrent stimulation administered for treatment purposes. In this particular investigation, the non-dominant leg was used to complete the 300 eccentric contractions and act as the injury model. The patients underwent a five day exercise-induced muscle soreness treatment program in which the treatment groups were compared to the control and sham treatment groups at 0 hours (pre-exercise), immediate post-exercise, 6 to 8 hours after exercise, and twice daily at 24, 48, 72,
and 96 hours. An honorarium was given to the subjects upon successful completion of the study.

Exclusion from the study was based on certain criteria that were strictly followed. Those excluded from taking part in this study were subjects who participated in regular physical activity such as weight training, training for team or individual sports (hockey, basketball, soccer, skiing, tennis, etc.), and sprint or distance running because these activities have some eccentric component as a part of training. It appears that performance of even one bout of eccentric exercise results in an adaptation in the muscle such that the muscle is more resistant to the effects of subsequent bouts of intense exercise (32). In addition, potential participants were to be in general good health. They should not have been subject to cardiovascular complications (angina, history of heart attack, high blood pressure) or respiratory problems (emphysema, high case of exercise induced asthma). Also, those who experienced delayed-onset muscle soreness within two months prior to study, had acute or chronic knee injury, or had surgery to the non-dominant leg were excluded.

**DOMS Protocol and Measurement Techniques**

The Kinetic Communicator (KinCom) dynamometer (Chattecx Corp., Chattanooga, Tennessee) is a hydraulically powered, computer controlled exercise testing device. It was used to measure and record the isokinetic eccentric torque of the quadriceps. Also, it was the device used to create the exercise-induced muscle soreness in the patients.
The first visit for all subjects was for pre-exercise baseline measures of mean eccentric torque, pain perception, quadriceps circumference for edema, and range of motion about the knee. These pre-exercise tests were given on the first day prior to commencing the exercise protocol. The patient received instruction and three to four practice trial sets of ten repetitions consisting of submaximal contractions in order to be familiarized with the resistance that the KinCom provided.

When the patient was satisfied and familiar with the KinCom, a two minute pause was given for a rest period. The pre-exercise baseline mean torque was performed with one set of four maximal repetitions at a constant velocity of 30 degrees per second through a 60 degree range of motion to the long muscle length (95 degrees of flexion to 35 degrees of flexion) of the non-dominant leg. The patients were instructed not to resist the concentric movement on the way up but to resist the machine's eccentric force on the way down. The pre-exercise baseline mean torque value was collected from the average of the last three maximal efforts of the four repetitions.

The lever arm length of the KinCom dynamometer was set by the researcher for each subject to 75 percent of the length from the head of the fibula to the lateral malleolus with the lateral joint line of the knee in alignment with the centre of the rotational axis point of the machine. To limit undesired movement, subject stabilization while in the KinCom seat included securing of the test leg on the upper third of the quadriceps and the lower leg by the shin pad, and a
belt fitting comfortably around the waist and chest. Also, they were instructed to grab hold of the sides of the seat for additional stability.

Following this test, the subjects completed a visual analogue scale (VAS) upon completion of four deep knee bends. The VAS had a range of "no pain or discomfort" to "worst pain or discomfort" experienced. For the purpose of this study, subjects reported any feelings of localized pain or discomfort and not fatigue in the exercised leg. The VAS was used to record the subjectively perceived pain about the quadriceps of the exercised leg due to exercised-induced soreness and not pain experienced upon recollection of past injuries (See Appendix A).

After the pain test, a baseline measure for the circumference of the quadriceps (QCM) was recorded using a tape measure. This measure evaluated changes in the circumference of the quadriceps if possible edema was present. Established landmarks were 10cm and 20cm above the superior boarder of the patella.

The final experimental measure was the range of motion (ROM) for active knee flexion assessed by a flexometer (Leighton Flexometer Inc., Spokane, Wash.) goniometric device that was recorded while the subject laid prone. The flexometer was aligned with the leg's long axis using the greater trochanter, lateral epicondyle and lateral malleolus as established landmarks. The flexometer placement that was proximal to the lateral malleolus was marked on the skin with permanent marker to ensure daily consistency and accuracy when assessing
the ROM. The point at which the subjects were unable to further actively flex their knee was considered the ROM end point. This measured stiffness occurring about the knee joint.

Following the pre-exercise measures, the subjects participated in the exercise protocol. They were once again instructed to maximally resist the downward force of the lever arm through the range of motion on the KinCom. Thirty sets of ten repetitions for a total of three hundred eccentric contractions were performed. The subjects received verbal feedback from the researcher and biofeedback from the resistive force or force versus velocity curve display screen on the KinCom monitoring unit.

After completion of the exercise, there was a three minute break then a repeat of the pre-exercise baseline tests to obtain post-exercise scores. The prescribed microcurrent treatment commenced immediately after the post-exercise measures in which the subjects received the first of 14 double-blind treatments.

The Microcurrent Stimulation Modality

The non-invasive functional devices consisted of an unadjustable frequency of either .3/.7 Hertz (Hz) or .3/18 Hz, an adjustable subsensory microcurrent (uA) output because each individual differs in sensory perception, a modified waveform that is termed biphasic because it has an unique ability to switch output voltages from +37 volts to -37 volts and to change between frequencies every seven seconds, a LED display for power on, and a nine volt
alkaline battery power source. The externally applied microcurrent was delivered through electrode pads placed on the quadriceps.

The same unit, indicated by a set of serial numbers, was used by the subject at each visit until the completion of the study. To effectively control the double-blinding of the investigation, the units were randomly placed in sequential order and only an independent party knew which serial number corresponded to the type of functional or sham units distributed.

The Prescribed Microcurrent Treatment Program

In an effort to ensure subject compliance to the treatment program, the patients reported to the Physiotherapy Laboratory in the Forrest Building for the three daily treatment sessions for the duration of the study. The program was of an aggressive manner in order for the cumulative treatments to take desired effect over the short time course of the study.

All subjects in the treatment groups (Groups A and B) received the prescribed 30 minute microcurrent treatment for exercise-induced soreness secondary to the eccentric exercise bout. The patients were administered microcurrent stimulation acting within the subsensory threshold level of each individual. The control group (Group C) underwent the same injury insult but did not receive any form of microcurrent stimulation; they were to continue with regular daily activity. The sham group (Group D) had no frequency or current output but only LED power display. All groups were subjectively tested and data collected for intensity
of perceived muscle soreness at 0 hours (pre-exercise), immediate post-exercise, 6 to 8 hours after exercise, and twice daily at 24, 48, 72, and 96 hours (PEVAS). The groups also underwent the same number of post-exercise tests and data collection for improvements of eccentric muscle strength (peak torque) force deficits (PEMT) and in range of motion (PEROM), and reduction of edema (PEQCM) each day of the study.

The prescribed microcurrent treatment began immediately after the initial post-exercise measures on day one. On days 2 to 5, the morning and afternoon treatments preceded the post-exercise measures of PEQCM, PEROM, PEMT and PEVAS (See Appendix B). The subjects were instructed to do the 30 minute treatments in the morning, afternoon, and evening at relative times throughout the test week. The subjects refrained from using any traditional treatment means during the study week such as ice, non-steroidal anti-inflammatory drugs, massage, heat, and physiotherapy. They were to be well hydrated during the treatment period to prevent any remote possibility of side effects such as nausea, light-headedness, headaches, and to maintain electrolytes in the body that can act as good conductors for electrical current. Water was provided at the clinic during the initial exercise test.

In commencing the treatment phase, subjects sat in a comfortable chair and the randomly selected unit was utilized. Four electrodes were placed by the researcher on the distal one-third portion of the quadriceps. Two pads were fixed near the musculotendinous junction on the vastus medialis and vastus lateralis muscles; these were at a distance of five
centimetres (cm) above knee centre at six centimetres medially and laterally. The other two pads were placed 15 cm above knee centre at eight centimetres medially and laterally. The four electrode pad placements within the area were to allow for ample current distribution to the affected musculature. After the pads were in place, the thirty minute treatment began. The pad placements were marked with permanent marker to assure daily consistency for the treatment effect.

Parameters Tested and Rationale for Use

The KinCom dynamometer measured the mean peak eccentric torque (Newton – metres; Nm) of the quadriceps muscle about the knee. Use of the KinCom testing device provided the eccentric muscle strength raw data pertinent for results in the difference of strength gains and relationships throughout the study period among the four experimental groups. It has been reported as seen in MacIntyre et al. (58) that the reliability of concentric and eccentric torque measurements on the KinCom system have an intraclass correlation coefficient ranging form 0.93 to 0.98 for both slow (30° / sec) and fast speeds (180° / sec) in groups of healthy active subjects.

The visual analogue scale, measured in millimeters (mm), monitored subjective information regarding the patient’s perception of muscle soreness. It dealt with the pain’s intensity and duration during the study period. Price et al. (70) have reported the between – session reliability of VAS measures of experimental pain to be high (r = 0.97).
The range of motion, in degrees, measured the stiffness during active knee flexion experienced with DOMS. It has been found that the intraclass correlation coefficient of test – retest reliability to be between 0.913 and 0.996 for the Leighton flexometer measures (54). The circumference of the quadriceps, measured in centimeters (cm), evaluated the thigh girth and possible edema present about the knee that accompanies exercise-induced muscle soreness. These were quantified by taking the mean of the last three of four measures of each test.

**Statistical Analysis**

The pain data was analyzed by the means of a two factor design [(4X10) group by pain] ANOVA with repeated measures on the pain factor. The strength data was analyzed by a two factor [(4X10) group by torque] ANOVA with repeated measures on the torque factor. The range of motion data was analyzed by a two factor [(4X10) group by ROM] ANOVA with repeated measures on the ROM factor. The circumference of the quadriceps data was analyzed by the means of a two factor mixed design [(4X10) group by circumference] ANOVA with repeated measures on the circumference factor. Significance level was set at p < 0.05. A Scheffe post hoc test was done for identifying all pair-wise comparisons. The power analysis of the study based on sample size calculations was at least 0.80 using 12 subjects per group (n = 12) at a significance level of p < 0.05 and a Cohen’s d for effect size of 1.0.
Chapter 4

Results and Discussion

Results

In this study, the estimation of reliability procedures yielded an intraclass correlation coefficient of 0.99 for the mean of the last three of four data measures used for statistical analysis of the study parameters.

Anthropometric Data

No statistical differences were detected between subjects in terms of age, height, and weight (Table 1).

Perceived Muscle Pain

The observed results demonstrated non-significant differences (p > 0.05, p = 0.22, p = 0.23) of group treatment effects and group interaction effects for a reduction in perceived muscle soreness. A significant effect (p < 0.05, p = 0.0001) was noticed due to a change over time. A Scheffe post hoc test was completed and it proved these results (Table 2).

Perceived muscle soreness peaked within 24 hours post-exercise for groups A, C, and D. Group B peaked at 48 hours AM and continued to decrease for the duration of the study. It could be observed that group A began to steadily decline after the 24 hour AM period. Group
Table 1. A Comparison of Subject Characteristics

<table>
<thead>
<tr>
<th>Experimental Group</th>
<th>Age (yr)</th>
<th>Height (cm)</th>
<th>Weight (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A</td>
<td>22.00 ± 4.94</td>
<td>178.08 ± 6.01</td>
<td>79.9 ± 10.79</td>
</tr>
<tr>
<td>Group B</td>
<td>19.17 ± 1.90</td>
<td>179.25 ± 7.45</td>
<td>82.98 ± 18.67</td>
</tr>
<tr>
<td>Group C</td>
<td>19.17 ± 1.75</td>
<td>178.67 ± 6.29</td>
<td>79.42 ± 14.46</td>
</tr>
<tr>
<td>Group D</td>
<td>20.83 ± 2.86</td>
<td>176.17 ± 6.15</td>
<td>74.83 ± 10.74</td>
</tr>
</tbody>
</table>

* Values are mean ± Standard Deviation (SD)

** Group A = .3 / .7, Group B = .3 / 18, Group C = control, Group D = sham.
<table>
<thead>
<tr>
<th>Group</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>3.08 ± 1.7</td>
<td>3.8 ± 0.2</td>
<td>2.3 ± 0.2</td>
<td>2.0 ± 0.2</td>
</tr>
<tr>
<td>Median</td>
<td>2.9 ± 0.3</td>
<td>3.2 ± 0.2</td>
<td>2.0 ± 0.2</td>
<td>2.0 ± 0.2</td>
</tr>
</tbody>
</table>

Values are mean ± Standard Error (SEM) not significant (p > 0.05).

Table 2. The Table Of Means and Standard Error (SEM) for Pre-Treatment Pain at 24, 48, and 72 Hours Post-Exercise.
C was elevated for the entire 24 hour period and it was noticed that group C fluctuated in decreasing over the rest of the study. Group D experienced no placebo effect of reduced soreness. The perceived muscle soreness of the sham group peaked at 24 hours PM after exercise and remained elevated up to 72 hours AM post-exercise above the other groups. All groups at 96 hours post-exercise were below their pre-exercise levels for the subjective feeling of muscle soreness (Figure 1).

**Quadriceps Circumference**

There were no significant differences shown among the experimental groups for reduction of either quadriceps circumference measure. At 10cm above knee centre (p>0.05, p=0.71; p = 0.44) and 20cm above knee centre (p>0.05, p = 0.67; p = 0.97) results revealed no group main effect or interaction effect took place. The Scheffe post hoc tests verified these findings. Changes over time exhibited a significant effect (p<0.05, p = 0.0001) (Tables 3, 4).

Both of these measures had very similar time courses. The first 24 hours post-exercise displayed some increase of possible occurring edema. Group A peaked at 24 PM hours post-exercise for both measures. Groups B and C peaked at 24 AM hours after exercise for each measure. Group D peaked at 24 AM hours after exercise for 10 cm measure but peaked at 48 AM hours after exercise for 20 cm measure. For each of the groups from 48 hours post-exercise the possible edema began to decrease and by 96 hours post-exercise scores were almost reaching pre-exercise levels (Figures 2, 3).
<table>
<thead>
<tr>
<th>Group</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-Exercise</td>
<td>42.67 ± 3.95</td>
<td>44.48 ± 3.25</td>
<td>44.38 ± 3.21</td>
<td>44.29 ± 3.30</td>
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<tr>
<td>Post-Exercise</td>
<td>42.49 ± 3.95</td>
<td>44.48 ± 3.25</td>
<td>44.38 ± 3.21</td>
<td>44.29 ± 3.30</td>
</tr>
<tr>
<td>Post-Exercise</td>
<td>42.49 ± 3.95</td>
<td>44.48 ± 3.25</td>
<td>44.38 ± 3.21</td>
<td>44.29 ± 3.30</td>
</tr>
<tr>
<td>Post-Exercise</td>
<td>42.49 ± 3.95</td>
<td>44.48 ± 3.25</td>
<td>44.38 ± 3.21</td>
<td>44.29 ± 3.30</td>
</tr>
</tbody>
</table>

Table 3. The table of means and standard deviation (SD) for swimming reduction (Quadriceps circumference measurement in cm) at 24.38 ± 3.96 hours post-exercise.
Values are mean (cm) ± Standard Deviation (SD); *p < 0.05

<table>
<thead>
<tr>
<th>Group A</th>
<th>12 / Group B</th>
<th>18 / Group C = control, Group D = sham</th>
</tr>
</thead>
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<tr>
<td>69 ± 4</td>
<td>5.9 ± 4.4</td>
<td>4.7 ± 4.7</td>
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<td>59 ± 4</td>
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<td>49 ± 4</td>
<td>5.9 ± 4.4</td>
<td>4.7 ± 4.7</td>
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<tr>
<td>39 ± 4</td>
<td>5.9 ± 4.4</td>
<td>4.7 ± 4.7</td>
</tr>
<tr>
<td>29 ± 4</td>
<td>5.9 ± 4.4</td>
<td>4.7 ± 4.7</td>
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<table>
<thead>
<tr>
<th>Experiment Group</th>
<th>Pre-Exercise</th>
<th>Post-Exercise</th>
<th>Immediate Post-8-Hour</th>
<th>Post-8-Hour</th>
<th>48-Hour (pm)</th>
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<tbody>
<tr>
<td>Group A</td>
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<tr>
<td>Group B</td>
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<tr>
<td>Group C</td>
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<tr>
<td>Group D</td>
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</table>

Table 4: The Table of Means and Standard Deviation for Swelling Reduction (Quadriceps Circumference Measure) at 24 h after 7.28 HOURS POST-EXERCISE.
Figure 3: Average QCM 20 cm for groups A, B, C, D.
Range of Motion

Statistical analysis indicated that there were no significant differences in range of motion loss for the treatment, control, and sham groups (p>0.05, p = 0.47, p = 0.67) when comparing between - and within- groups respectively. Therefore, no significant treatment effect was shown. Further analysis by Scheffe post hoc test confirmed these results. However, it was noticed that there was a significant effect over time for the groups (p<0.05, p = 0.0001) (Table 5).

All groups had a tendency to show a slight recovery in range of motion after 6 to 8 hours post-exercise. The range of motion loss excluding immediate post-exercise was greatest at 24 AM hours post – exercise for groups A, B, and C. Group D showed a maximal decrease in ROM at 24 PM hours after exercise. An improvement in range of motion occurred around 48 hours post-exercise with greater improvements shown 72 hours post-exercise. At 96 hours post-exercise, relative pre-exercise values were reached (Figure 4).

Eccentric Strength

Peak eccentric strength analysis through peak torque measures found no significant difference for a group effect nor was there any significance of an interaction effect (p>0.05, p = 0.60; p = 0.29). There was, however, significance for peak eccentric strength across time (p<0.05, p = 0.0001) (Table 6). The Scheffe post hoc test supported the results.
### Table 5: The Table of Values and Standard Deviation for Rats of Motion Loss A + 24 + 3.96 hr Post-Exercise

<table>
<thead>
<tr>
<th>Group</th>
<th>Immediate Post-Exercise</th>
<th>Post-Exercise</th>
<th>Post-Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group A</td>
<td>1.32 ± 0.04</td>
<td>1.38 ± 0.02</td>
<td>1.34 ± 0.01</td>
</tr>
<tr>
<td>Group B</td>
<td>1.30 ± 0.06</td>
<td>1.37 ± 0.03</td>
<td>1.32 ± 0.02</td>
</tr>
<tr>
<td>Group C</td>
<td>1.28 ± 0.07</td>
<td>1.36 ± 0.04</td>
<td>1.30 ± 0.03</td>
</tr>
</tbody>
</table>

Values are mean (deflection) ± standard deviation (SI) and are significant (p < 0.05).
Figure 4: AVERAGE RANGE OF MOTION FOR GROUPS A, B, C, D.
<table>
<thead>
<tr>
<th>Group A</th>
<th>Group B</th>
<th>Group C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-Exercise</td>
<td>Post-Exercise</td>
<td>Post-Exercise</td>
</tr>
<tr>
<td>Immediate Post-8-Hour</td>
<td>Post-8-Hour</td>
<td>Post-8-Hour</td>
</tr>
</tbody>
</table>

Table 6. The Table of Means and Standard Deviation for Eccentric Strength (Peak Torque) Across 72.96 Hours Post-Exercise.
Figure 5: Average peak eccentric torque for groups A, B, C, D.
The greatest peak eccentric strength loss as expected occurred immediately post-exercise followed by a recovery period at 6 to 8 hours after exercise. Although not significant, a second decline of eccentric muscle strength was noticed at 24 hours after exercise. Group A was decreased at 24 hours AM while groups B, C, and D were decreased at 24 hours PM post-exercise. Continual improvements, with slight fluctuations, were noticed for the duration of the study to a point in which all were well above pre-exercise levels at 96 hours post-exercise (Figure 5).
Discussion

Exercise-induced soreness has been researched for years and several variables of interest have been found. Variables such as pain or discomfort, edema, range of motion, and eccentric strength are being studied and compared in order to better understand the exercise-induced or delayed onset muscle soreness (DOMS) phenomenon. The exercise-induced soreness for this investigation offered an injury model to evaluate the variables and the efficacy of a microcurrent treatment modality.

Perceived Muscle Soreness

It was hypothesized that the muscle soreness experienced would have been reduced by the prescribed microcurrent treatment. The results of this study did not support this hypothesis. Perceived muscle soreness was not significantly affected by the prescribed microcurrent treatments or by the sham treatment. The non-significant differences found with the sham group, in comparison to the other groups, may be due to a large sample of intersubject variability. Another possible reason for the non-significant results was probably not enough stimulus to induce true exercise-induced muscle soreness. The quadriceps musculature may not have been elongated enough during the eccentric exercise protocol that being 95° of flexion instead of 110° of flexion.

In this study, the soreness intensity progressively increased from 6 to 8 hours up to 24 to 48 hours after the exercise. This type of progression has been termed temporary soreness or
soreness occurring immediately following exercise and lasting a few hours possibly due to fatigue and secondly, residual soreness being felt 24 to 48 hours post-exercise due to muscle damage (79). The perceived muscle soreness peaked between 24 to 48 hours post-exercise as observed in most studies involving exercise-induced soreness. The pain returned to baseline measures within 5 days (Figure 1; Table 2). Other investigators have documented these results (37, 58, 62).

The subjects reported pain and tenderness across the distal region of the quadriceps involving the medial and lateral borders. The pain did not seem to migrate towards the muscle belly for any of the subjects. It has been reported in other studies that tenderness was primarily located at the distal, medial, and lateral parts of the quadriceps becoming more diffuse at its peak intensity (62). The distribution of tenderness sparing the muscle bellies has also been documented (38, 62). The area of the musculotendinous junction appearing to be the primary site of pain and tenderness was in agreement with those previously mentioned studies. It is believed that the muscle fibres are oriented most obliquely just proximal to the musculotendinous junction making them most vulnerable to the high tensions of eccentric exercise (30, 38). Characteristically there is no pain at rest but considerable discomfort with any movement requiring development of muscle tension (57).

The lack of pain reduction with the prescribed microcurrent treatment demonstrated in this study was a surprise because the protocol called for cumulative treatment sessions over the time course of the study. It was hypothesized that these treatments were to achieve an
analgesic carry-over affect. Pain is a very subjective variable in which researchers are dealing with a psychophysiological phenomenon involving pain threshold and tolerance levels (78). The perceived pain experienced by a patient is a product of interpretation by the mind; the intensity felt can be increased or decreased by conscious and unconscious thoughts or emotions (78). The sensory component maybe modulated by the subject's past experience where attitudes and psychological variables may influence description of the sensation (57). A person may report more or less pain than what is actually being felt and this error is usually in the direction of reporting less pain (78).

Several studies have given conflicting results for the alleviation of subjective muscle pain (9,11, 51, 52, 71, 84). These investigators studied the effects of different eletrotherapeutic devices such as high voltage pulsed current (HVPC), low-and high-frequency transcutaneous electrical neuromuscular stimulation (TENS), and microcurrent electrical neuromuscular stimulation (MENS). They have found that low-and high-frequency TENS and submotor (10% less voltage than able to evoke a muscle contraction) HVPC treatments the most effective for reducing pain; HVPC and MENS were ineffective for reducing pain. It was found that low – volt microamperage stimulation (LVMAS) had no significant effect on perceived pain but since there was significance across time they suggested that LVMAS may have had a transient analgesic response to perceived pain (29).

Other electrotherapeutic studies using acupuncture- like low frequency TENS for chronic pain sufferers have achieved immediate pain relief (31, 75). It is believed that this type of
treatment activates the endogenous pain controlling system in which an increase in the concentration of endorphins occurs (56, 75). This low frequency TENS analgesia is believed to result from increased beta-endorphin release that acts as an opiate receptor agonist resulting in centrally mediated analgesia (28).

**Edema**

Edema, following a traumatic injury, is a natural occurrence of the inflammatory process and if it were to remain untreated could often lead to increases of pain and decreases in the range of motion around a joint. Evidence of swelling has ranged from increased circumference of the exercised muscle 24 to 48 hours post-exercise to ultrastructural evidence of post-exercise edema and direct measurement of intramuscular resting pressures (33). However, measuring of edema and intramuscular pressures has yielded conflicting results (30).

The results of this study, although non-significant after statistical analysis, did show quadriceps circumference had increased in the first 24 hours following exercise and this may be due to possible edema or perhaps an increase in blood volume but this is not known for sure (Figures: 2, 3; Tables: 3, 4).

The muscle fibre swelling is believed to be caused by an increase in the intramuscular pressure as a result of increased amounts of degraded protein components and the release of protein-bound ions in damaged muscle cells (30, 33, 38). The accumulation of interstitial or
intracellular fluid can be explained as a response to myofibrillar disintegration (37). The release of intracellular proteins into the circulation likely results from a subsequent increase in the intracellular osmotic pressure (30, 33, 38). It was also hypothesized that the swelling of muscle fibres during activity leads to increased tissue pressure and disturbed microcirculation (53). When microcirculation is compromised, this leads to metabolic changes and free radical formation which may activate proteolytic enzymes (53). Another proposed explanation is increased permeability of post capillary venules to plasma proteins as the predominant mechanism of edema. As the excessive plasma proteins move into the interstitial space, fluid may be drawn out of the vascular bed because of the imbalance of proteins across the vessel wall, resulting in edema formation (80).

Various studies have found opposing results. Brown and colleagues (9) showed an no effect on edema; Kulig and colleagues (51) in one study found no effect from MENS for decreasing CPK levels and in another study Kulig et al. (52) concluded that microcurrent stimulation affected CPK levels; Rapaski and colleagues (71) revealed that microcurrent stimulation at subsensory levels decreased CPK; and Wolcot et al. (84) showed that HVPC delivered at a submotor level was most effective in reducing CPK levels. Electrical stimulation has significantly inhibited acute edema formation after a single 30 minute treatment with effects lasting 4 to 7.5 hours post treatment and it has been shown that electrical stimulation retarded edema formation after four 30 minute treatments up to 17 hours post-treatment (7, 80). Both studies used HVPC at
voltages 10% less than that needed to evoke muscle contraction. In this present study, no effect was seen with the prescribed microcurrent stimulation.

**Range of Motion**

The reduction in the range of motion could be associated with an increase in muscle stiffness. A decrease in range of motion occurred immediately after the eccentric exercise; it was found that the greatest reduction with the exception of immediate post – exercise was the 24 hours post-exercise period. There seemed to be a gradual return of ROM from 48 hours to 96 hours after exercise in which ROM values were near pre – exercise levels (Figure 4, Table 5).

Stiffness of resting muscle has been classically determined by the connective tissue elements surrounding individual fibres, bundles of fibres and the whole muscle (44). Findings indicated that the stiffness was due to connective tissue damage and possible edema within the connective tissue network; and it is suggested that a consequence of this is an increased mechanical sensitivity of muscle receptors giving rise to painful sensations when activated by stretch or pressure (47). It is believed that any injury-induced leakiness of either the surface membrane or sarcoplasmic reticulum would tend to increase the intracellular calcium (Ca$^{2+}$) creating stiffness (44).

Range of motion decrements may result from the fluid accumulation within muscle compartments or possibly chemically induced changes in the mechanical properties of
connective tissue or the cytoskeletal elements (44). Recently, it has been suggested that the structures within muscle fibres contributing to the resting stiffness include portions of the cytoskeletal network, especially connecting filaments and the low-level crossbridge interaction (44). Therefore, abnormal flexion and stiffness developed before pain further suggests that connective tissue is damaged during exercise and pain was part of a secondary response (47). Stiffness measured in intact limbs may also include components of non-muscular origin such as the joint capsule and the skin (44). There have been reports of increased circumference or girth measures occurring but no soreness was present until 24 to 48 hours post-exercise (44,79).

Studies that used microcurrent stimulation to treat DOMS or other soft tissue injuries revealed mixed results. Brown and colleagues (9) showed an increase in range of motion; Kulig and colleagues (51) found no effect from MENS for stiffness; Rapaski et al. (71) revealed that microcurrent stimulation at subsensory levels did not decrease stiffness; and Wolcot et al. (84) showed that HVPC delivered at a submotor level was most effective in reducing stiffness. There was no significant change of ROM found with the prescribed microcurrent therapy in this study. The function of prescribed microcurrent therapy on the mechanics of increasing ROM and decreasing possible edema in this present study is still undetermined.
Eccentric Strength

After a novel bout of eccentric exercise, muscle damage is precipitated and there is an immediate decrease in maximal muscle force production followed by a recovery process that can take hours or days to reach pre-exercise measures. It has been reported that maximum voluntary force of the quadriceps returned to pre-exercise levels within 24 hours but others have found it can take as long as a week or two for eccentric torque to recover (39,44,57,58,62).

The eccentric strength as measured by the mean peak eccentric torques revealed non-significant results in the relation to the prescribed microcurrent treatment. Peak eccentric strength was found to have its greatest decline immediately post-exercise. A recovery tendency was noticed 6 to 8 hours post-exercise but 24 hours after exercise displayed another decline in the mean eccentric torques. Beyond 24 hours, recovery gradually improved with slight variations to a point well above pre-exercise levels by 96 hours post-exercise (Figure 5, Table 6). True exercise-induced soreness may not have been accomplished because there was a non-significant result for the bimodal action that occurs in humans. This bimodal pattern of eccentric torque has been reported by MacIntyre et al. (58).

It is not known if the initial decline in strength is due to muscle injury, muscle fatigue or both (57). It has been proposed that eccentric exercise would damage a pool of fragile or stress-
susceptible muscle fibres; these fibres may develop through disuse associated with the lack of using a given motor recruitment pattern (2,14). The fact that greater tension per muscle fibre is generated under eccentric contraction conditions provides a situation where relatively few fibres are recruited and are producing relatively high force (62). The exercise causes considerable morphological changes that have been seen immediately post-exercise and continually progress.

The initial exercise creates microscopic damage of focal lesions at the level of individual sarcomeres that become progressively more extensive during the next four days (37,63). The sarcomeres may be over stretched and the subtle changes in sarcomere length could influence the muscle’s ability to generate force because the sarcomeres pulled apart reduce the overlap between the actin and myosin filaments (24). The injury to sarcomeres can lead to surface membrane damage with the entry of extracellular Ca\(^{2+}\) and activation of proteases and lipases (63). The myofibrillar disruption which has been observed immediately after exercise and continuing for a few days are Z-line disorganization, lysosomal and nonlysosomal cytoplasmic proteinases activation, and disturbances in calcium homeostasis (30,37,57,63). Serum muscle protein changes such as an increase in creatine phosphokinase (CPK) or creatine kinase (CK) have been used by many researchers as an indirect indicator for the morphological evidence of exercise-induced muscle damage. It has been stated that the post-exercise rise in circulating CK activity is a manifestation of skeletal muscle damage but not a direct indicator of it (24).
Weber et al. (84) included microcurrent stimulation effects on force deficits due to DOMS. Their findings were similar to this study as there was non-significance for the peak torque of group main effect and interaction effect but significance across time. Denegar et al. (29) found that LVMAS had no effect on strength recovery but there was significance over time.

Muscle fatigue has been considered as part of the cause for the loss of muscle function associated with eccentric exercise. It was observed in this study that the subjects thought this state of fatigue as being exhaustion and the decrements in eccentric mean peak torques were attributed to this "fatigue". The fatigue that really occurs has been termed low-frequency fatigue (LFF). Investigators have studied the force/frequency characteristics of eccentrically exercised muscle and found that there is a decrease in force generation at low frequencies of stimulation (57).

The development of LFF is greater when muscles work at long rather than short lengths; this is possibly a consequence of the generation of high forces at long lengths (48). Low-frequency fatigue is thought to occur at the level of the excitation-contraction coupling due to the mechanical damage of the high forces (57). The mechanical damage to the sarcoplasmic reticulum resulting in less Ca\textsuperscript{2+} release for each excitatory action potential may be a cause of LFF (62). It has been suggested that the length-dependent nature of LFF implies greater stress to the muscle at long sarcomere lengths, stress to structures in series with the contractile components and thus, possible injury to the tendinous attachments (57). This injury may result in an inflammatory response, swelling, distension and stiffness of the
connective tissue (57). Long-lasting fatigue (LLF) is common immediately after eccentric exercise and may take as long as 3 to 4 days to recover (57).

Summary of Limitations

The fluctuations of pain intensity are supposed to be dependent upon the underlying pathology, however, estimates are again subject to a certain bias due to individual differences in the ability to describe pain accurately and discriminately (17). The quality and intensity of the pain experienced is not the same among people although the study may involve identical pain producing procedures and scoring (78). Therefore, careful consideration was needed for this study’s subjective pain evaluation. It was possible that no pain relief occurred because of the frequently recorded pain scores. Subjects completed two daily visual analogue pain scales that may have influenced the data. Although blinded from previous scores, the subject’s perceptual recollection of the previous score may have been a factor.

In this investigation, scores also may have been affected because each subject anticipated that he was to experience some degree of pain immediately following the exercise protocol and perhaps after or during the PEMT test. However, a DOMS stimulus should not create any pain immediately after exercise but some fatigue may be present. Under careful instruction, it was suggested that the subjects be able to distinguish between the localized pain about the knee and quadriceps, and not the pain associated with exercise to fatigue, exertional pain, or past painful experiences (77). This was to deter the subjects from scoring their pain
equivalent to the worst pain ever experienced in their lifetime, however, some subjects did score muscle soreness more highly than others. It has been found that soreness ratings were quite variable among subjects with some subjects reporting no soreness and others reporting maximal soreness (14).

Another possible explanation of the non-significant pain results may be that the eccentric exercise protocol did not cause sufficient damage to the musculature of the given study population. Although screened to meet certain criteria, the subjects may have been participating in eccentrically biased sports or activities and in doing so gave false accounts of their habitual activity levels. This participation would have adapted the quadriceps thereby protecting the musculature from the eccentric exercise and as a consequence the subjects rating of pain when compared to the more sedentary subject would have been diminished (6, 14, 39, 47, 53). It could also be that the microcurrent stimulation frequencies used for this investigation did not make a difference.

This study did not involve the effect of microcurrent treatment on serum CPK levels and other indicators such as lactate dehydrogenase (LDH) and myoglobin. This was due to the invasiveness towards subjects, additional expense, and time constraints. Another reason for not examining the standard CPK levels was the subject CPK response relative to intersubject variability including age, body composition, and race. Subject response to CPK has been studied and concluded that after a similar bout of exercise the CPK responses vary between
subjects. It has been suggested that differences exist among no, low, and high responders (22).

Significant differences not observed with the use of the prescribed microcurrent stimulation may be partially explained by subject variability with respect to age, height, weight and habitual status. A more homogeneous sample and a larger sample size may benefit this investigation. Results may have been further affected by a learning effect or the lack of familiarization with the KinCom since the final PEMT was higher than the BLMT value. The pre-exercise value was meant to be the maximal eccentric torque value used for comparison. This learning effect may have transpired because the subjects completed 300 repetitions that could have made them more adept to the action and they were able to acquire better motor unit recruitment of the quadriceps. The subjects may have become more conditioned in performing the novel movement on the KinCom (the resisting against an external force by the lever arm while lengthening the muscle). Again as previously mentioned, an increase in the degrees of flexion should produce significant results because of this researcher's speculation that true DOMS may not have been achieved in this present study as evident from the pain and bimodal observations.

The initial BLMT could have been influenced by an apprehension in anticipation of the forthcoming events or a possible lack of coordination on the KinCom. Therefore, subjects may not have produced their maximal effort on the initial torque test. Partly at fault were time constraints, this researcher's judgement for the number of practice sessions of three or four
sets of submaximal contractions prior to the BLMT and the reduction of maximal eccentric strength due to early tiredness from the practice sessions.
Chapter 5

Summary, Conclusions, and Recommendations

Summary

The purpose of this investigation was to determine the efficacy of microcurrent treatment as an electrotherapeutic modality for muscular soft tissue injury. The second purpose was to determine if the prescribed microcurrent stimulation reduced the subjectively perceived muscle soreness and edema, and improved the range of motion and eccentric muscle strength associated with an exercise-induced muscular soft tissue injury model.

Forty-eight male subjects between the ages of 18 to 35 years were exercised on the Kinetic Communicator (Kin Com). The subjects completed 300 eccentric muscle contractions using the non-dominant leg to create the exercise-induced muscle soreness and act as the injury model. They were randomly assigned to one of four experimental groups consisting of two functional treatment groups (Group A = .3/.7 Hz, Group B = .3/18 Hz), a control group (Group C), and a sham group (Group D). The sham group was used to monitor possible placebo effects, and the control group did not undergo the prescribed microcurrent treatment program but continued habitual activities. The subjects were tested and data collected at 0 hours pre-exercise, immediate post-exercise, 6 to 8 hours after exercise and twice daily at 24, 48, 72, and 96 hours over the time course of the investigation. Raw data was recorded by using a visual analogue scale (VAS), a flexometer goniometer, quadriceps circumference measure, and the KinCom for specific range of motion testing.
Results indicated no significant difference (p>0.05) for the tested parameters of exercise-induced muscle soreness between or within the groups. Significant differences (p<0.05) were found for the tested parameters due to changes across time. The prescribed microcurrent treatment program had no effect on managing DOMS secondary to eccentric muscle activity.

Conclusions

In hindsight to the predetermined hypotheses of this research, the following assumptions were concluded:

1. the prescribed microcurrent treatment did not result in significant reduction of the subjectively perceived muscle pain over the five day study period according to VAS scores;

2. the prescribed microcurrent treatment did not result in significant reduction of edema as recorded by quadriceps circumference measures;

3. the current study suggested that there were no significant improvements for range of motion about the knee after the prescribed microcurrent treatments;

4. the prescribed microcurrent treatment had no significant effect on improving eccentric muscle strength during the recovery period of DOMS;
5. the prescribed microcurrent treatment was not significantly more effective than the sham and control groups in decreasing exercise-induced muscle soreness and edema, or for improving the range of motion and eccentric muscle strength; and

6. the current study also implied that the prescribed microcurrent stimulation did not significantly demonstrate a frequency intensity effect for the treatments.

Recommendations

Although the current investigation findings do not support the prescribed microcurrent stimulation for treatment of symptoms due to exercise-induced muscle soreness, it is deemed necessary by this researcher that further study into this topic be continued. The non-significant results appeal this request and the deficiencies of this study have to be investigated appropriately to achieve the desired goal. Some deficiencies of this study can be found in the previously mentioned summary of limitations and as follows within these recommendations.

The perceived muscle pain should be reexamined. It is a subjective variable that may require other pain scales and pain producing agents to obtain a better outcome. A pain scale such as the McGill Pain Questionnaire or the Descriptor Differential Scale might be considered (57,58). Another pain producing technique as described by Hasson et al. (42) should be used
to mechanically depress the sore region of the muscle. These changes may present a more objective means of measuring the pain felt by a subject and its intensity.

Clinicians in this type of research on exercise-induced muscle soreness should further investigate the effects of prescribed microcurrent stimulation on a cellular level by histologically examining more objective biochemical indicators such as malondialdehyde (MDA) for measuring of lipid peroxidation and the subsequent increase of oxygen free radicals on muscle cell damage, and a specific cytokine Interleukine – 6 (IL-6) which may be a more evident marker of muscle cell injury. The MDA as an index of lipid peroxidase secondary to exercise-induced soreness has been measurable over the duration of a DOMS study (85). Nuclear medicine techniques for phagocytic cellular activity by means of marking white blood cells like neutrophils, leukoctyes and monocytes, and muscle biopsies for muscle myeloperoxidase done periodically over the time course of the investigation may aid in measuring the inflammatory response to microcurrent stimulation. The use of magnetic resonance imaging (MRI) as described by Shellock et al. (74) or ultrasound could quantify the inflammatory response to the microcurrent stimulation and perhaps define a time course of the inflammatory stage. In order to differentiate between the reduction of muscle damage and the enhanced rate of body healing, examining for amino-acid production and intracellular ATP might show positive changes in collagen and/or protein synthesis at the injured site. Also, the effect of microcurrent stimulation on blood flow may be studied to detect any subtle changes that would reveal a possible exchange occurring of an influx of nutrients and an efflux of exudate in such an injury model.
The musculature used in this study may have been a factor in the results. Although the eccentric exercise protocol using the quadriceps as the injury model has been successful (58), much of the research into microcurrent stimulation has involved the elbow flexors. This could be due to the fact that the quadriceps are a lower extremity body part that do more weight bearing and daily activity, therefore, they are more adapted to extreme loads. The region may be affected by such factors as the skin, fat, muscle, bone and etc. with regards to area and volume than the upper extremities. This could have an effect on treatment times to get the desired penetration required for therapy. The 60° range of motion for the initial exercise insult and the post-exercise mean peak torques on the KinCom may have to increase from 95° flexion to 35° flexion to 110° flexion to 50° flexion also as found by this researcher, the range of motion may be limited by the size and uncomfortable positioning of the calf muscle against the bottom of the KinCom seat. The use of 1 repetition maximum (1RM) on a leg press exercise apparatus working to exhaustion as weight is decreased over time might induce a substantial injury to create a more measurable exercise – induced muscle soreness that can be tested on the KinCom for force deficits.

Further study may provide evidence that a possible bimodal pattern of the eccentric peak torque took place. The initial decline of force occurred immediately post-exercise and a second decline occurred within the 24 hour interval but it was not found to be significant. This pattern has been previously noticed and reported in humans (58). Most investigators have reported the greatest decline of force in humans immediately after exercise with recovery at 24 hours and onward (58). This study measured mean peak torques immediately after
exercise, 6 to 8 hours and 24 to 96 hours post-exercise in the morning and afternoon for its duration. It has been suggested that the bimodal response may be due to two mechanisms. The first decline in force may be a function of mechanical injury and fatigue, especially where subjects have just completed an exhaustive eccentric protocol and the second decline in force occurs in response to phagocytic activity at the site of the initial damage (58).

Other controlled research that deserves investigation for the effects of microcurrent stimulation should include chronic inflammatory injuries such as arthritis or osteoarthritis, the beta-endorphin release of endorphins or enkephalins for endogenous pain control, and low-frequency fatigue differences by electromyography (EMG) analysis.

Lastly, the study into microcurrent stimulation and exercise-induced muscle soreness warrants future research. At the moment proponents believe in its effect, however, the literature on microcurrent stimulation and human injury models appear to be limited and some of the reports are anecdotal in nature, and most of the evidence is lacking credibility because of uncontrolled clinical trials. Therefore, investigation into this topic may shed further light on the efficacy of microcurrent stimulation as an effective electrotherapeutic modality. To this point, the present investigation has shown no effect of microcurrent stimulation on exercise-induced muscle soreness.
References


APPENDICES
Appendix A: Visual Analogue Scale
### Table: Design of Pretreatment Posttreatment Treatment / Exercise - Induced Somnolence Study

<table>
<thead>
<tr>
<th>Group</th>
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<th>No Exclusion</th>
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</thead>
<tbody>
<tr>
<td>1</td>
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<td>VASI</td>
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<td>10</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
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<td>VASI</td>
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<td>0</td>
</tr>
<tr>
<td>3</td>
<td>Group B</td>
<td>VASI</td>
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<td>0</td>
</tr>
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<td>Group C</td>
<td>VASI</td>
<td>10</td>
<td>10</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

**Notes:**
- **VASI** (Visual Analog Scale)
- **PEVAS** (Post-exercise Visual Analog Scale)
- **PECOM** (Post-exercise Control)
- **PREM** (Pre-exercise)
- **POST** (Post)
- **PRE** (Pre)