A Comparison of the Effectiveness of Two Modalities in the Treatment of Delayed Onset Muscle Soreness

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A COMPARISON OF THE EFFECTIVENESS OF TWO MODALITIES IN THE TREATMENT OF DELAYED ONSET MUSCLE SORENESS

By

Kenneth Newland

THESIS

Submitted to the Department of Physical Therapy at Grand Valley State University Allendale, Michigan in partial fulfillment of the requirements for the degree of

MASTER OF SCIENCE IN PHYSICAL THERAPY

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A COMPARISON OF THE EFFECTIVENESS OF TWO MODALITIES IN THE TREATMENT OF DELAYED ONSET MUSCLE SORENESS

ABSTRACT

The purpose of this study was to compare the effects of massage and light exercise in alleviating the signs and symptoms of DOMS. Fifteen male and female subjects were recruited. The subjects were divided into two groups. To obtain a DOMS response, each group performed eccentric exercise on a Biodex Isokinetic Dynamometer to both arms. As treatment, one group (N=7) received massage to one arm and light exercise in the form of upper body ergometry to the other arm. The other group (N=8) received massage to one arm and light exercise in the form of supine elbow flexion/extension to the other arm. The subjects recorded their perceived pain before DOMS exercise, and again at 8, 24, and 48 hours after treatment. Mean pain differences were calculated for each group at each time interval. Statistical analysis indicated that there were no significant differences between treatments.
ACKNOWLEDGMENTS

To my thesis committee, Arthur Schwarcz, Gordon Alderink and William Bell, thank you. Without your guidance through the research maze I would not have learned as much as I have from this project.

To Jane Toot, whose patience, understanding and caring allowed me to finish this project with my sanity intact, thank you just does not seem to be enough. I wish you nothing but the best that life has to offer.

To my wife, Karen, and my boys, Marcus, Tyler and Matthew, who endured the mood swings and remembered who I was when I managed to see them, thank you and I love you. Now we can get on with the life we have envisioned.
PREFACE

Definition of Terms

**Acute onset muscle soreness.** The moderate pain and fatigue felt at the end of strenuous exercise. This pain is brief and requires only a short rest after exercise before it subsides.

**Concentric contraction.** An active shortening of the muscle fibers.

**Creatine Kinase.** Enzyme that regulates anaerobic ATP production.

**Delayed onset muscle soreness.** The dull, aching pain which typically begins within the first 24 hours after unaccustomed, strenuous exercise. This pain may last up to seven days depending on the intensity of exercise.

**Eccentric contraction.** A muscle contraction where the muscle fibers are lengthening.

**Effleurage.** A technique used in massage that accustoms the subject to the physical contact of the massage therapist. It is composed of light gliding movement with no attempt to manipulate the tissue.

**Isokinetic exercise.** Exercise where the speed of movement remains constant but the resistance is varied so that the same force is generated throughout the movement.

**Isometric contraction.** A muscle contraction where the muscle fibers shorten enough to “take up the slack” in the muscle but no bony movement occurs, such as pushing against a wall.
**Massage.** A mechanical manipulation of body tissues with rhythmical pressure and stroking for the purpose of promoting health and well being.

**Petrissage.** A kneading of the tissue that is accomplished through pressing and rolling action using repeated grasping, pressure and lifting or rolling creating a “milking” action.

**Reliability.** The degree of consistency with which an instrument or rater measures a variable.

**Validity.** The degree to which an instrument measures what it is intended to measure.
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CHAPTER I
INTRODUCTION

Whether a conditioned athlete or "couch potato," unaccustomed physical activity can lead to muscular soreness. This pain can be categorized into two types: (1) acute onset muscle soreness (AOMS) or (2) delayed onset muscle soreness (DOMS).

AOMS is the "burning" and fatigue felt at the end of an exercise bout. This type of muscle pain requires only a brief rest period before pain subsides. DOMS is a dull, aching pain which begins within 24 hours and may last five to seven days. Accompanying DOMS is joint and soft tissue swelling and stiffness (Cleak & Eston, 1992), and decreased strength and function. Biomechanical alterations, such as decreased range of motion (ROM) (Rodenburg, Bar, & DeBoar, 1993) and altered joint kinematics (Hamill, Freedson, Clarkson & Braun, 1991) have also been described.

Concomitant with the physical symptoms of pain, decreased strength and altered functions is a psychological component, that, for example, could have developed from participating in an activity that results in DOMS. Thus, making even the heartiest souls reluctant to engage in that type of activity again. This can prove problematic for those who work in the athletic and rehabilitative professions.

Despite the many studies conducted on DOMS since the initial article by Hough in 1902, it is only recently that the underlying mechanisms are being discovered. Now, a preponderance of evidence supports the structural injury theory as the initiating event.
(Francis & Hoobler, 1988). This theory suggests that due to the increased stress placed on individual muscle fibers during eccentric contractions, susceptible or weakened fibers are disrupted at a subcellular level "kicking off" a cascade of events which include an acute inflammatory response and extracellular calcium influx that eventually causes DOMS (Armstrong, 1990; Newham, McPhail, Mills & Edwards, 1986).

A multitude of treatment methods have been studied in an effort to decrease the severity or eliminate the symptoms of DOMS. Among these are the use of drugs such as ibuprofen (Donnelly, Maughan, & Whiting, 1990), dexamethasone via iontophoresis application (Hasson, Wible, Barnes, & Williams, 1992), static stretching (Buroker & Schwane, 1989), and ice massage (Yackzan, Adams, & Francis, 1984). Each of these protocols have met with varying degrees of success. Also among these methods are low resistance exercise and athletic massage.

Hasson, Barnes, Hunter & Williams (1989) reported that high speed concentric isokinetic exercise 24 hours after inducing DOMS significantly reduced soreness while Donnelly, Clarkson, and Maughan (1992), using much the same protocol, reported no success in reducing DOMS signs and symptoms. Isabell, Durrant, Myrer, and Anderson (1992) administered light exercise treatments 0, 2, 4, 6, 24, 48, 72 and 96 hours post-DOMS and again noted no significant reduction in soreness. No studies were found in the literature which utilized a one-time bout of light exercise immediately after inducing DOMS through eccentric exercise.

As with post-DOMS exercise regimens, massage therapy has also met with varying degrees of treatment success. Smith, et al. (1994) administered a 30-minute...
massage to the elbow flexors/extensors of subjects two hours after eccentric exercise and noted a significant decrease in muscle soreness. They suggested this was due to interruption of neutrophil emigration, a critical step in the inflammatory response and/or an increase in cortisol levels, a potent anti-inflammatory substance. However, Wenos, Brilla, and Morrison (1990) noted no decrease in DOMS symptoms when massage was administered to the quadriceps femoris muscle immediately post-exercise.

It would be very helpful to athletic participants if a method could be found to decrease DOMS. The purpose of this study was to compare massage and light aerobic exercise treatments administered immediately after eccentric exercise to determine which, if either, was more effective in combating the problems of DOMS. These treatments were chosen due to the fact that (a) both have met with some degree of success and (b) in most cases, both are easily administered and, with a minimal amount of instruction, can be performed by the subject and can be performed at any juncture post DOMS. The reason the author has chosen to administer treatment immediately after exercise is that many patients or athletes, due to possible physical or cognitive impairments, such as aphasia or mental retardation, may be unable to self-administer treatments. Also, if there is a question of compliance, the therapist or trainer may elect to perform treatment themselves. In most instances, for numerous reasons it would not be practical to ask patients or athletes to return at a later time so that treatment could be administered.
CHAPTER II
REVIEW OF THE LITERATURE
Related to DOMS

Exercise, especially strenuous, unaccustomed exercise, can lead to muscle micro-damage at the sub-cellular level. Evidence of this includes (a) morphological change (Friden, Sjostrom, & Ekblom, 1981, 1983; Newham et al., 1983; Jones, Newham, Rounds & Tolfree, 1986), (b) DOMS (Asmussen, 1956; Clarkson, Byrnes, McCormick, Turcotte, & White, 1986; Edwards, Mills & Newham, 1981), (c) performance decrements (Newham, Mill, Quigley & Edwards, 1983; Rodenburg et al., 1993; Hamill et al., 1991), and (d) increased muscle protein levels, especially creatine kinase (CK) in the blood (Newham, Jones & Edwards, 1983; Clarkson, Litchfield, Graves, Kirwan, & Byrnes, 1985). This damage is not permanent and repair does take place (Friden et al., 1981; Ebbeling & Clarkson, 1989). In fact, not only does repair take place but subsequent bouts of exercise at the same intensity result in little or no damage (Clarkson et al., 1985; Clarkson & Trembley, 1988; Newham, Jones & Clarkson, 1987). The exact cause of exercise induced damage and repair is not well understood. Two basic mechanisms have been offered to explain the initiation of exercise induced muscle damage. One mechanism is a disturbance in metabolic function and the other describes mechanical cellular disruption.
Metabolic Mechanisms

During prolonged submaximal eccentric exercise, metabolic events, such as ischemia or hypoxia, which result in changes in ion concentration, ATP deficiencies and waste accumulation have been proposed to explain muscle damage induced through exercise (Armstrong, 1984; Byrnes & Clarkson, 1986; De Vries, 1966). This theory, which was first described by De Vries (1961), proposes that exercise may cause ischemia in the working muscle, which would result in the production of pain substances. If too much of these substances were to accumulate in the muscle, pain endings would be stimulated. The resulting pain would then produce more reflex spasms prolonging the ischemia, initiating a pain-spasm-pain cycle (De Vries, 1966). He based this theory on his observations that resting subjects with DOMS had higher electrical activity on surface Integrated Electromyographs (IEMGs) (De Vries, 1961, 1966).

Other researchers have noted increased resting IEMG activity as well, but the magnitude of electrical activity was not related to soreness perception (McGlynn, Laughlin & Rowe, 1979). These authors used a bipolar surface electrode placement, which De Vries (1966) stated was not sensitive enough to pick up electrical activity in sore muscles and that surface unipolar electrodes should be used. However, Abraham (1977) stated that under similar conditions bipolar electrodes were three times more sensitive than unipolar. Also, using the same unipolar technique as De Vries, Newham et al, (1983) did not find an increase in the IEMG of sore muscles. It should also be mentioned that De Vries' results may not relate to "classical" exercise induced DOMS as his subjects had a wide variety of "accidentally induced muscle pain."
Studies performed on animals have also shown muscle damage in ischemic situations other than exercise. In one study researchers cut off the blood supply to rats' quadriceps femoris muscles to induce ischemia. A three-hour occlusion resulted in a loss of cross-striations, fragmentation, and inflammation within 48 hours following the return of blood flow. A one-hour occlusion resulted in only mild to moderate changes with the cross-striations being preserved (Ebbeling & Clarkson, 1989).

Cell injury induced by ischemia resembled cell damage following exhaustive endurance exercise (Ebbeling & Clarkson, 1989). Changes seen in the gastrocnemius muscle of marathon runners included disruption of the sarcolemma, contractile apparatus, mitochondria and sarcotubular system (Ebbeling & Clarkson, 1989). Marathon running may produce an ischemic situation in the working muscle which may cause direct damage or indirect (secondary) damage by delaying the clearance of lactic acid and/or other waste products. However, during running eccentric actions are performed by the leg extensors as the foot hits the ground and decelerates the center of mass (Armstrong, 1986). Researchers have suggested that this eccentric activity during endurance running may also cause muscle damage (Armstrong, 1986) and metabolic changes may increase the damage (Newham et al., 1983).

If lactic acid were responsible for exercise induced muscle damage, then muscles that contract concentrically and fatigue quickly would show more damage than muscles that are used eccentrically. In fact, the opposite appears to be true. Comparing skeletal muscle damage in rats following eccentric (downhill running) and concentric (uphill running) exercise, Armstrong, Oglivie, & Schwane (1983) found that running downhill
had a lower metabolic cost and produced greater muscle damage. Schwane, Watrous, Johnson & Armstrong (1983) used the same exercise protocol on human subjects and found a significantly increased level of lactic acid in those who exercised concentrically and yet did not suffer DOMS. The opposite was true for the downhill runners. Thus, it would seem lactic acid is not a primary initiator of DOMS.

Mechanical Mechanisms

The main argument against the metabolic hypothesis is that greater muscle damage has been found after eccentric exercise when compared with concentric exercise of the same intensity (Newham et al., 1983). Eccentrically exercised muscles require less energy than muscles exercised concentrically, but sustain greater damage as evidenced by DOMS (Newham et al., 1983), morphological changes (Newham et al., 1983) and plasma protein increases (Newham et al., 1983). IEMG activity was also lower during eccentric exercise which suggests that fewer fibers are recruited to produce large forces (Ebbeling & Clarkson, 1989). Therefore, muscles produce greater tension per fiber when under eccentric load.

Several studies have employed a step test to compare concentric and eccentric exercise (Newham et al., 1983). Subjects were instructed to step up with one leg (concentric contractions) and step down with the opposite leg (eccentric contraction) thus keeping the workload equal for each leg. Concentrically contracted muscles showed no morphological changes while eccentrically contracted muscles showed marked myofibrillar disorganization. Force decrement (as assessed post-exercise) and pain were also greater in the muscles exercised eccentrically (Newham et al., 1983).
The most direct evidence of structural damage has been provided by histological and ultrastructural analysis (Armstrong et al., 1983; Friden et al., 1981, 1983; Newham et al., 1983). The z-band in normal skeletal muscle appears as a well organized woven basket or square lattice (Hoppeler, 1986). Following eccentric exercise the z-band has shown streaming, broadening and even total disruption (Friden et al., 1981). Friden et al., (1981) have suggested that the z-band may be the weak link in the myofibrillar chain. Other changes noted include myofibrillar and sarcolemmal disruptions (Armstrong et al., 1983, Friden et al., 1981), A- and I-Band widening (Armstrong et al., 1983, Friden et al., 1983, Newham et al., 1983), as well as increased mitochondrial volume density and organelle displacement (Friden et al., 1983; Armstrong et al., 1983; and Jones et al., 1986).

Since CK is found almost exclusively in skeletal and cardiac muscle, its presence in serum or plasma is a strong indicator that muscle damage has occurred (Armstrong, 1986). Clarkson et al., (1986) and Ebbeling & Clarkson (1989) have all noted an increase in the levels of circulating CK after eccentric exercise and to a lesser extent after isometric exercise. However, since CK levels peak four to seven days post-exercise and pain is greatest 24-72 hours post-exercise, it is unlikely that CK plays a role in the production of pain associated with DOMS (Newham et al., 1983; Clarkson et al., 1986).

Hough (1902) suggested that DOMS was the result of mechanical stress placed on the muscle and its components and pointed especially to the connective tissue as the site of damage. Asmussen (1956) also hypothesized that DOMS was due to the over-stretching of the muscles elastic components. He found that soreness was most localized
to the tendonous attachments. More recently, Newham et al (1983) also reported that soreness after eccentric exercise occurred in the area of the myotendinous junction and concluded that the soreness was due to damage from mechanical stress, predominantly to the connective tissue.

Abraham (1977) noted a significant positive correlation between urinary excretion of hydroxyproline (OHP) (a product of connective tissue breakdown and indicators of collagen synthesis) and reports of soreness. Other studies have also shown an increase in OHP, but the increase was not significant. It should be noted, however, that in these studies the course of OHP excretion was only followed for 48 hours and the intensity of soreness was not reported (Cleak & Eston, 1992).

Damage and Repair Processes

Whether damage is initiated through metabolic or mechanical means, the sequence of events that follow are not well defined due to the variety of methods used to induce DOMS and the different periods of assessment following insult (Ebbeling & Clarkson, 1989). Friden et al. (1981) found significantly less z-band disruption on day seven following repeated stair descents than on day two indicating repair was taking place. The same researchers found evidence of immediate damage after exercise to the vastus lateralis muscle and three days later the damage had increased (Friden et al., 1983). Lipofuscin granules indicative of lysosomal activity (Friden, Kjorell, & Hornell, 1984) and polyribosomal complexes indicating protein synthesis and muscle repair (Friden et al., 1983) were seen in the disruption area. Six days after exercise, muscle fibers appeared essentially normal.
This change in fiber appearance suggests that damage progresses after exercise but is not permanent (Armstrong et al., 1983, Friden et al., 1986, 1983). Factors that may influence the damage and repair process are intracellular calcium (Ca++) influx (Armstrong et al., 1983; Clarkson & Trembley, 1988; Friden et al., 1983; Newham et al., 1983), inflammation (Smith, 1991), connective tissue damage (Armstrong et al., 1983; Asmussen, 1956), decreased energy sources (Ebbeling & Clarkson, 1889) and the presence of cytoskeletal and myofibrillar proteins in histological samples (Friden et al., 1984).

**Calcium**

High levels of Ca++ in muscle cells can trigger a series of events leading to necrosis (Ebbeling & Clarkson, 1989). It is hypothesized that the surface membrane is damaged when actin and myosin fibers are pulled apart during eccentric contraction allowing extracellular Ca++ to enter (Armstrong, 1984; Newham et al., 1983). This increase in Ca++ may explain the mitochondrial swelling seen by Friden et al. (1983). In an attempt to maintain homeostasis mitochondria sequester the excess Ca++. Due to the overload, oxidative phosphorylation is reduced and ATP production decreases (Ebbeling & Clarkson, 1989) initiating a sequence of events beginning with an impaired ability of the ATP dependent pumps to extrude Ca++ (Armstrong, 1984).

Ebbeling & Clarkson (1989) found that protein degradation was lowest in incubated rat skeletal muscle when no Ca++ was added and increased with increasing Ca++ concentrations. Also, when dantrolene, an inhibitor of Ca++ release from the sarcoplasmic reticulum, was added protein degradation was reduced. Thus, it would
appear that Ca++ from extra and intracellular sources effect protein turnover in skeletal muscles.

Some studies have reported a spontaneous shortening of muscles damaged by exercise, but this shortening was not caused by an increase in electrical activity (Clarkson & Trembley, 1988). Clarkson & Trembley (1988) had subjects perform eccentric exercise with the forearm flexors and then measured the elbow angle with subjects' arms hanging relaxed at their sides. This angle continued to decrease for two days indicating a shortening of the forearm flexors. They hypothesized that this shortening was due to an accumulation of Ca++ in the damaged fibers although they did not speculate as to how Ca++ caused the shortening.

There is also some evidence which points to eccentric exercise causing damage to the sarcoplasmic reticulum. Significant decreases in force production have been noted, especially at low frequencies of electrical stimulation (Newham et al., 1983). This has been attributed to insufficient Ca++ release from damaged sarcoplasmic reticulum, leading to a decreased ability of the muscle fiber to produce force (Newham et al., 1983). Although an attractive hypothesis, the effect of Ca++ needs further study to define its role in the DOMS phenomena.

Inflammation

Inflammation is the body's response to any type of tissue injury. The purpose of inflammation is to initiate healing of the damaged tissue (Smith, 1991).

Inflammation is biphasic, involving a vascular and cellular response. The vascular response begins with a five to ten minute vasoconstriction and is followed
several hours later by an extended period of vasodilation and increased vascular permeability (Smith, 1991).

The cellular response consists of mainly two types of white blood cells, polymorphic neutrophils (PMN's) and monocytes (Smith, 1991). Within one to four hours after injury PMN's concentrate at the site of injury, although this time may vary with the severity of the injury. PMN's secrete a variety of agents that aid in the breakdown of dead tissue cells in the area (Smith, 1991).

Several hours after PMN mobilization, monocytes migrate to the area, rise steadily in number and are maintained up to 48 hours. They then leave the blood and enter the tissue where they mature into macrophages, disposing of necrotic tissue and removing foreign bodies, a vital role in the healing process (Smith, 1991).

Smith (1991), suggested that the macrophages may be indirectly responsible for the pain associated DOMS. She proposed that, during an acute inflammatory response, macrophages synthesize a substance called prostaglandin E (PGE). Although PGE does not cause pain directly, it sensitizes pain receptors producing a state of hyperalgesia (increased sensitivity). This heightened state of sensitivity would allow chemical, mechanical, or thermal stimuli, previously benign, to activate pain receptors (Smith et al., 1991). Citing her own laboratory studies and those of another research, she reported finding similar time courses for increases in PGE and DOMS and suggested a positive relationship between the two. However, she did not give any further details concerning these studies.
As with Ca++, the inflammatory hypothesis is very attractive. However, several studies have failed to show an association between inflammation and DOMS. Armstrong et al. (1983) used a downhill running protocol with rats and although, on biopsy, macrophages were found in the area, PMN's were rarely seen in the involved tissue. Since PMN's are an important part of the inflammatory process they concluded that the muscle pathology did not involve inflammation in the classic sense (Armstrong et al., 1983). Friden et al. (1983) also failed to find an association between DOMS and inflammation in human studies. Following eccentric bicycle exercise, biopsies of the vastus lateralis muscle were taken and no monocytes were found in the tissue (Friden et al., 1983).

Jones et al. (1986) reported finding inflammatory cells in the muscles of human subjects after undergoing a fatiguing bout of eccentric exercise. However, the inflammatory cells were seen much later than would be normally anticipated. Armstrong (1986) suggested that the reason for this delay was that the damage was initiated at a site away from the muscle belly, where the biopsies were taken. The inflammatory cells then spread throughout the surrounding tissue, thus disrupting the normal time course. Some researchers have suggested that the site of initial damage may be the connective tissue of the myotendinous junction.

**Connective Tissue**

As stated earlier, OHP is a by-product of connective tissue collagen breakdown. Armstrong et al. (1983) stated OHP and other by-products of collagen breakdown may act as chemotactic agents for monocytes to travel from the blood into the tissue. The
animal work of Fritz and Stauber (1988) suggested that the invading cells may be myogenic in origin. Proteoglycans, major components of connective tissue, are essential for regulation of the myogenic process including satellite cell release, myoblast fusion and myotubular orientation. Thus, connective tissue may play a regulatory, as well as structural, role in the damage and repair process (Fritz & Stauber, 1988). The same authors noted that proteoglycans were localized to the endomysium in normal muscle, but were absent between one and three days after injury. Within five days after injury proteoglycans levels were essentially normal. This suggests that the extracellular matrix is an important part of the repair process as it provides structural support for regenerating cells (Fritz & Stauber, 1988).

Energy Sources, Cytoskeletal and Myofibrillar Proteins

Limited data exists on the role of energy sources in the damage and repair process. Because persons with certain diseases where muscle glycogen is compromised show severe rhabdomyolysis (disintegration of muscle fibers) in response to endurance type exercise, it has been suggested that glycogen depletion may initiate muscle damage. However, since eccentric exercise does not lead to total glycogen depletion, it is not likely that this would produce the damage seen after eccentric work in healthy subjects (Ebbeling & Clarkson, 1989).

However, the availability of glycogen may be important in the repair process. Several researchers have found a significant reduction of glycogen immediately after eccentric exercise. Samples taken as much as ten days later showed that, not only were levels not restored, but were, in fact, lower. Since soreness still persisted, it was
suggested that a reduction in glycogen synthesis may impair the repair process (Ebbeling & Clarkson, 1989). Also involved in the repair process may be the intermediate filament protein, desmin (Friden et al., 1984). Evidence suggests that this protein links the z-bands to one another and to the sarcolemma (Ebbeling & Clarkson, 1989). Friden et al. (1984), using microscopy with an antibody to desmin, found longitudinal extensions in biopsies taken three days after eccentric exercise. These were taken as an indicator of cytoskeletal disorganization. They hypothesized that there is an increased turnover of cytoskeletal proteins following muscle damage and desmin may be important in myofibrillar protein reorganization (Friden et al., 1984). Another study has shown a substantial increase in four unidentified proteins which may also reflect enhanced protein synthesis necessary for repair (Ebbeling & Clarkson, 1989).

**DOMS and Accompanying Symptoms**

**Pain and Swelling/Stiffness**

As stated earlier, the pain associated with DOMS increases in intensity during the first 24 hours after exercise and may last five to seven days, depending on the intensity of exercise (Armstrong, 1984; Byrnes & Clarkson, 1986; Cleak & Eston, 1992). This pain is most intense in the myotendonous junction of the affected muscle (Asmussen, 1956; Newham et al., 1983; Abraham, 1977). Three possible explanations have been suggested for the localization of pain.

One theory suggests that soreness may be the result of damage to the muscle and/or connective tissue fibers (Asmussen, 1956; Abraham, 1977; Friden, Sbakianos, & Hargens, 1986). Since muscle fibers near the myotendonous junction are oriented
obliquely, this makes them highly susceptible to the high stress of eccentric work (Friden et al., 1986). However, since muscle fibers are considerably more elastic in nature than connective tissue, Asmussen (1956) suggested that connective tissue fibers may be even more susceptible to the high stress and injury.

Edema has also been suggested as a cause of the pain associated with eccentric exercise. However, the evidence supporting this is contradictory (Ebbeling & Clarkson, 1989). Some studies found that limb volume was increased from 24 to 72 hours after eccentric calf exercise, but other researchers were unable to find any significant correlation between limb volume and soreness of the elbow flexors. Both studies used volumetric measurements to procure their results (Ebbeling & Clarkson, 1989).

Using a more sophisticated technique of slit catheter measurement, Friden et al. (1986) found a significant increase in tissue pressure in the anterior tibial compartment of the leg two days after exercise. However, others failed to find a significant increase in intramuscular pressure when using the same technique on the elbow flexors (Ebbeling & Clarkson, 1989). Newham (1988) stated that the conflicting results were a result of compliance differences in the respective compartments. The anterior tibial compartment has a relatively low compliance, whereas the elbow flexor compartment is more compliant and distensible (Newham, 1988).

Since anti-inflammatory drugs do not significantly reduce muscle soreness (Donnelly et al., 1990), the edema noted by several researchers may not be due to inflammation in the classical sense. Friden et al. (1983) suggested an alternative theory. Since fiber disruption can lead to the formation of degraded proteins and release of
protein bound ions, these substances may lead to an increase in the osmotic pressure causing fluid buildup. This increase in pressure may irritate free nerve endings and be perceived as soreness (Friden et al., 1983). This ties in well with Byrnes & Clarkson's (1986) theory as to the cause of pain associated with DOMS.

The sensation of pain in skeletal muscles is carried by group III myelinated and group IV unmyelinated nerves (Armstrong, 1984; Byrnes & Clarkson, 1986). These nerves are particularly dense in the region of the connective tissue of the myotendonous junction. Since group IV fibers carry dull, diffuse pain and are twice as common as group III fibers, Armstrong (1984) suggested these were primarily responsible for the sensation of delayed onset pain. Byrnes & Clarkson (1986) agreed and suggested that substances such as bradykinin, prostaglandin, serotonin, histamine and potassium produced or released with muscle damage activated those nerves causing the perception of pain. Armstrong (1984) also suggested that the delay in onset of pain was due to the fact that when cells die, time is needed for any or all of these substances to accumulate.

**Strength Loss**

Cleak & Eston (1992) had 26 physical therapy students perform eccentric exercise using the elbow flexors. Isometric strength measurements were taken immediately after exercise and at 24 hour intervals for 11 days. A reduction in strength was noted immediately after exercise and was greatest at 24 hours post-exercise. Eleven days post-exercise, isometric strength remained 20% lower than pre-exercise values (Cleak & Eston, 1992).
Clarkson & Trembley (1988) also induced DOMS of the elbow flexors of eight female subjects. Subjects were assigned to groups that performed either 24 or 70 maximal eccentric contractions. Both groups demonstrated maximum strength loss at day one post-exercise. However, the group performing 24 maximum contractions had strength return to baseline five days post-exercise. The 70 maximum contraction group showed a significant strength loss remaining on day five post-exercise.

Newham et al. (1987) superimposed electrical stimulation over maximal voluntary contractions to determine whether motivational factors or pain intolerance was responsible for the reduced ability to generate force. They found that not only were maximal voluntary contractions decreased but those elicited by electrical stimulation alone were as well (Newham et al., 1987). This was particularly evident at low frequencies of stimulation and was considered to be due to the damaged sarcoplasmic reticulum (Newham et al., 1983).

It is unclear at this time what the mechanism is behind the loss of strength noted after eccentric exercise (Ebbeling & Clarkson, 1989). Pain was thought to be responsible, but Newham et al. (1983) and Cleak & Eston (1992) have shown that the pain course does not follow that of strength loss. The theory proposed by Newham et al. (1993, 1987) that sarcoplasmic reticulum damage resulting in less Ca++ for action potentials is certainly plausible. However, this author has been unable to find any study which will support their theory. Another possibility is that mechanical damage to the muscle fibers may decrease their inherent ability to develop tension (Ebbeling & Clarkson, 1989).
Altered Kinematics

Hamill et al. (1991) used a 30-minute bout of downhill running on a treadmill to induce DOMS in ten subjects. Using a high speed video camera, they noted several kinematic alterations during subsequent runs at 48 and 120 hours post-DOMS. Most significant were the changes at the ankle and at the knee during both support and swing phases, as well as at the hip during support phase (Hamill et al., 1991). Both hip and knee flexion were reduced during the initial portion of support phase resulting in a decreased ability to attenuate shock. An increase in ankle dorsiflexion during support phase was also noted. The authors speculated that the increase in ankle dorsiflexion may be a compensation to reductions in ROM at the hip and knee (Hamill et al., 1991). The authors did not speculate as to the cause of these alterations. Cleak & Eston (1992) suggested that altered kinematics coupled with strength decrements may put athletes at risk for injury during periods of DOMS.

Role of Massage in Recovery From DOMS

Massage has long been heralded as a "cure-all" for muscle soreness and injury. However, very few scientific studies have been undertaken to document its effectiveness. Dukes & Ronto (1995), suggested that some of the benefits of massage are: (a) reduction of muscle tension, (b) quick recovery from the effects of fatigue, (c) optimum adaptation to training stimuli, (d) rapid restoration of body energy, (e) improved ability to renew physical activity without the athlete wasting unnecessary energy and time and, (f) improved general sedative effect. They claimed these benefits could be achieved because massage can: (a) increase local blood flow to joints hastening drainage from the region
reducing peri-articular swelling, (b) produce muscular relaxation, (c) increase lymphatic and venous return, (d) rapidly remove waste products and, (e) loosen and stretch tight tendons (Dukes & Ronto, 1995). Massage may also effect the oxidation and regeneration process in the muscles since it may increase the oxygen supply, thus facilitating the muscle repair process (Dukes & Ronto, 1995).

Wyke, (1980) suggested massage may also reduce pain through neurological mechanisms. This may be accomplished through stimulation of mechanoreceptors found in the soft tissue. These receptors synapse on interneurons in the spinal cord which, in turn, synapse on the nociceptive (pain) afferent neurons. The interneurons have an inhibitory effect on the nociceptive neurons. In order for the brain to perceive pain, these neurons need to undergo an excitatory response. Thus, the inhibitory synapse dampens the perception of pain in the brain (Wyke, 1980).

Recovery massage typically is composed of the basic movements of effleurage followed by petrissage and ending with effleurage (Cafarelli & Flint, 1992). Effleurage is a technique which accustoms the subject to the physical contact of the therapist. It is used to enhance relaxation, prepare the subject for further soft tissue manipulation and distribute skin lubricant (Cafarelli & Flint, 1992). Petrissage is used to mobilize tissue fluids, stretch adhesions, press metabolic waste products out of the area and increase local blood supply (Cafarelli & Flint, 1992). Effleurage and petrissage together may stimulate muscle contraction and relaxation rhythmically with the strokes. This action squeezes the venous and lymphatic vessels and forces the venous blood and lymphs toward the heart (Cafarelli & Flint, 1992).
Smith et al. (1994) studied the effects of a 30-minute athletic massage on subjects two hours after intense eccentric exercise to the forearm flexors/extensors. They theorized that by performing massage at this time a critical step in the inflammatory process, emigration of PMN's, would be interrupted preventing further damage and, thus, reducing soreness. Muscle soreness was assessed at 8, 24, 48, 72, and 120 hours post-exercise using a zero to ten-point pain scale. Result showed the massage group suffered significantly less soreness when compared to the control group (Smith et al., 1994).

Weber, Servido, & Woodall (1974), using an exercise protocol similar to Smith et al. (1994), also studied the effects of massage on muscle soreness. Treatments were administered immediately after exercise and again 24 hours after exercise. In this study, the elbow flexors were massaged for eight minutes. The authors were unable to detect any significant differences in perceived soreness between treatment and control subjects.

Role of Exercise in Recovery From DOMS

It is thought by many that metabolic by-products, especially lactic acid, are the precipitating events in DOMS. Evidence suggests that after a bout of intense exercise a bout of light exercise will hasten the removal of lactic acid (Powers & Howley, 1990, p. 54-63). This removal may be due to an increase in blood flow, the “pumping” action of the muscle, or both. It has been further suggested that the intensity of the light exercise bout should be between 30% and 40% of the subjects VO\(_2\) max (the body’s maximal capacity to deliver and utilize oxygen) (Powers & Howley, 1990, p. 54-63). However, it should be noted that the suggested intensity of recovery exercise is relative to full body exercise.
As with massage, the use of exercise in combating DOMS has met with mixed results. Donnelly et al. (1992) elicited a DOMS response on the elbow flexor/extensors. Subjects in the experimental group performed a second bout of exercise similar to the first but maximum torque was limited to 50% of that produced during the first bout. Also, 25 contractions were performed during the second bout, compared to 70 performed initially. The light exercise session produced neither temporary nor long-lasting reduction in muscle soreness.

Isabell et al. (1992) also induced DOMS in the elbow flexors/extensors of subjects. In their study they compared the effects of ice massage, ice massage with exercise and exercise on muscle soreness. Treatments were administered at 0, 4, 6, 24, 48, 72, and 96 hours post-exercise. Their results, although not statistically significant, showed that perceived soreness in the exercise group was less intense, peaked sooner and diminished more rapidly than the other treatment and control groups.

This corroborated, to a degree, the findings of Hasson et al. (1989) who used a bench stepping protocol to elicit a DOMS response in the quadriceps femoris muscle of ten subjects. At 24 hours post-exercise, the experimental group performed a high velocity, isokinetic exercise regiment. Exercise consisted of 20 maximum voluntary knee flexion/extensor at 300°/second with a three-minute rest period repeated six times. Soreness was assessed using a pressure probe with a grid placed over the knee extensors. Results showed that the experimental group suffered significantly less soreness when compared to the control group. The authors did not speculate as to the mechanism behind their findings.
Limitations of Previous Studies

All of the previous studies have two major limitations: (1) small treatment group size and (2) the subjective nature of the variable. Pain ratings may differ significantly between subjects. What one person perceives as a “three” may be perceived as “five” to a different person. Also, the change in pain from day to day may be perceived differently. One person may rate that change as a “one” and another person may rate the same change as a “three”. The only way this author knows to compensate for this variability is to have a large sample size so the variability “evens out.” The largest treatment group size used in the above studies was ten (Weber et al., 1994). This author does not believe this to be a sufficient amount of subjects per group to complete a meaningful statistical analysis.

Summary

The mechanisms which lead to DOMS are not yet clear. Initial damage may be due to metabolic waste products and hypoxia or brought on by high mechanical stress placed on muscles and connective tissue. Recent evidence points to the latter as the major cause.

Once initial damage has occurred, it continues to increase for several days. However, the damage is not permanent and repair does take place. This continuation of damage and repair is believed to be the result of intracellular Ca++ influx, an inflammatory response, glycogen depletion, as well as the presence of cytoskeletal and myofibrillar proteins. It is not clear as to the extent each plays a role.
DOMS has been strongly associated with eccentric exercise and, to a lesser extent, isometric exercise. This is thought to be due to the fact that during eccentric exercise fewer muscle fibers are recruited to produce large forces leading to the production of higher forces per muscle fiber.

Soreness arises within the first 24 hours and may last from five to seven days. Concomitant with the soreness is a loss of strength, swelling/stiffness and altered kinematics.

Massage has been touted as a "cure-all" for muscle pain and injury. Because few scientific studies have been done, its effectiveness has not been clearly documented. As with massage, exercise has also met with varying degrees of success in alleviating the signs and symptoms of DOMS.

Hypothesis

It is believed that the use of massage and exercise helps to clear metabolic waste products from the area of injury. Since evidence suggests that the accumulation of metabolic waste products is not the precipitating event in DOMS, the author hypothesizes that no difference in muscle soreness will be seen when massage or exercise is used post-exercise to alleviate the signs and symptoms of DOMS.
CHAPTER III
PROCEDURE

Research Site

This study was conducted in the physical therapy department at Saint Mary's Hospital in Grand Rapids, Michigan.

Subjects

Fifteen subjects (10 females and 5 males) were recruited from the Grand Valley State University (GVSU) student body and faculty. The subjects ranged from 23 to 39 years of age with a mean of 28.6 years of age.

Exclusion criteria for this study were anyone who (a) were less than six months status-post fractures, strains or sprains of the upper extremities, back or neck, or currently suffered from any of the above; (b) suffered from rheumatoid or osteoarthritis of the upper extremities, back or neck; (c) had neurological or neuromuscular disorders which may impair pain perception or muscle contractibility; (d) lifted weights regularly (three or more times a week) or participated in athletic events which entailed moderate to heavy upper extremity work (i.e., wrestling or football); (e) had a heart disorder or suffered from hypertension or, (f) suffered from osteoporosis.

Procedures

Each subject underwent a brief neuromuscular screen (Appendix C) to rule out any abnormalities before participating in this study. This screen was performed by the
researcher. Each eligible subject then read and signed a consent form (Appendix A) as
approved by the GVSU Human Subjects Review Committee.

The subjects were informed that they would undergo vigorous eccentric exercise
to both arms and would then receive massage to one arm and light exercise to the other
arm. The subjects were randomly assigned to one of two groups, massage to the
dominant arm or exercise to the dominant arm, according to the randomly prepared list.
This was to reduce, to the extent possible, the effect arm dominance may have on DOMS.
Thus, the massage treatment (MT) group consisted of eight subjects who received the MT
to their dominant arm and seven subjects who received the MT to their nondominant arm.
The exercise treatment (ET) group consisted of eight subjects who received the ET to
their nondominant arm and seven subjects who received the ET to their dominant arm.

In order to produce a DOMS response on the subject's upper extremity, each
subject performed eccentric exercise using a Biodex Isokinetic Dynamometer ® (BID)\(^1\).
The BID is a computer operated machine which was used to flex/extend the subject's
elbows at a constant speed allowing maximum force to be generated by the subject
through the full ROM. BID resistance (torque) was set at 140 ft.-lbs. so that no subject
could overpower the machine and stop the movement arm during exercise.

For the exercise bout the subjects were seated and their upper arm positioned and
secured in approximately 45° of shoulder horizontal abduction and flexion in accordance
with the BID instructions. Shoulder flexion varied slightly because of differences in the

\(^1\) Biodex Corporation, Shirley, NY, 19864)
subjects' sitting heights. The BID axis of rotation was aligned with the approximate axis of rotation for elbow flexion and extension (the humeral trochlea and capitulum) according to the BID instructions. The hand grip of the BID movement arm was adjusted so that the subjects' wrists were in approximately five degrees of flexion. This appeared to be the most comfortable position for the subjects. The BID was then engaged and each subject's arm was moved passively through their ROM. The subjects were asked if they were comfortable during the movement. If they stated they were not, positional adjustments were made until comfort was achieved. Because each subject's ROM varied to a small degree, the BID was set to move a 100° range (negative 5°-10° of elbow extension to 105°-110° of elbow flexion). This ROM was chosen arbitrarily, but was comfortable for all participants. Maintaining the BID in the passive mode with the speed set at 30°/second, subjects were instructed to perform 10 eccentric and 10 concentric contractions at approximately 50% of their maximum strength. This was used as a warm-up exercise and to familiarize the subjects with the BID's movement and speed.

Following the warmup and familiarization procedures, the exercise program was begun. The subjects performed five sets of 20 eccentric contractions at 30°/second with a one minute rest period between each set. Scoring windows were set automatically on the computer screen and were based on the first contraction of each set. This helped to keep contraction force consistent. Each subject was instructed to give the first contraction of each set "all you've got." Scoring windows were set at 90%-100% of the first contraction during the first exercise set and 70%-100% of the first contraction during the subsequent sets.
The subjects were instructed to resist the BID as it straightened their elbow, to relax and to not assist the machine as it returned to the starting position. They were also instructed to watch the computer screen and to keep the torque curves inside the scoring windows as much as possible. If the torque curves began falling below the scoring windows, verbal encouragement was added. No subject was able to keep the torque curves inside the scoring windows for the full 20 contractions. Although this exercise protocol was different from all previous studies, it was tested on three pilot subjects and produced a DOMS response. The ET arm was always exercised first. This was done as a time saving measure. While the subjects completed the treatment, the BID was set up to exercise the other arm.

The ET group performed one-armed cycling on an upper body ergometer (UBE) as the treatment. Subjects cycled in a clockwise direction (relative to the right arm) for eight minutes at a rate of 60 revolutions per minute (rpm). This protocol matches that of Weber, et al. (1994). Subjects were seated with the UBE axis of rotation aligned with the axis of rotation of the glenohumeral joint and the hand positioned in approximately 90° of pronation according to the manufacturer’s instructions.

After performing the complete experiment on six subjects, it was noticed that each subject complained that the UBE treatment arm had already developed considerably more soreness than the massage treatment arm. It was also noted that the subjects had great difficulty completing the UBE protocol and demonstrated an immediate decrease in elbow extension. This was particularly apparent in the weaker subjects. An informal check of these subjects’ soreness the next day revealed that the UBE treatment arm
developed soreness more quickly and to a greater extent. It was hypothesized that the UBE may, in fact, increase the DOMS response. However, without a control group to compare with, there would be no way to show this. In fact, if the UBE was increasing the DOMS response, it would appear during statistical analysis that the MT was a more effective treatment. In an effort to investigate if the UBE exercise protocol was increasing muscle soreness, a second exercise protocol was instituted.

The second ET instituted consisted of elbow flexion/extension in the supine position with the arm elevated above the level of the heart. This position was used so that gravity could aid in the venous return of metabolites to the circulatory system. The subjects were instructed to slowly flex and extend the arm for eight minutes, the same amount of time as the UBE group. The subjects who performed this ET did not have the complaints that the UBE group had. At the end of the study eight subjects had performed the flexion/extension exercise and seven subjects had performed the UBE exercise.

After completion of the treatment, the subjects then performed the same eccentric exercise protocol to the contralateral arm and received the massage treatment to the biceps brachii muscle. The massage consisted of three minutes of light effleurage followed by three minutes of kneading, three minutes of wringing, three minutes of milking and ended with three minutes of light effleurage. The 15 minutes massage time was consistent with that of Dukes & Ronto (1995) and in between that used by Smith, et al. (1994) and Weber, et al. (1994). Subjects were positioned supine during the massage with the arm elevated above the level of the heart, again to aid venous return of metabolites to the circulatory system.
Subjects were then instructed to avoid heavy upper extremity work during the 48 hour measurement period. Subjects were also instructed not to use analgesic creams or medications and to avoid being under the influence of alcohol when assessing their pain.

Measurement

Each subject assessed their muscle soreness before DOMS exercise, and again at 8, 24 and 48 hours post-exercise. Muscle soreness was assessed by having subjects fully extend their arms and palpate the myotendonous junction of the biceps brachii. They rated their soreness using a zero to 10-point scale (Appendix B). A score of zero meant no pain and a score of ten meant unbearable pain.

A separate measurement sheet was used for each soreness assessment. This was to decrease the influence previous measurements may have had on subsequent recordings. Each recording was sealed in an envelope supplied by the researcher. When all measurements were completed the envelopes were sealed in a large envelope and placed in a box located in the GVSU physical therapy department.
CHAPTER IV
RESULTS
Statistical Analysis

In order to detect any significant differences in pain perception between the UBE (n=7) and the flexion/extension (n=8) exercise groups, a two-sample t-test was used at each time interval. A difference score (UBE exercise pain score minus MT pain score) was calculated for each member of the UBE group and the mean differences were found. The same method was applied to the flexion/extension exercise group resulting in a mean difference score for that group. It was these means that were tested at each time interval.

In order for the two-sample t-test to be valid three assumptions must be met: (1) the variables must be independent, (2) the two groups must have equal variances, and (3) the two groups must have a normal distribution. Independence is assumed since the arms treated with UBE and flexion/extension exercise are not on the same person and thus, the pain scores were not dependent of each other. The SAS t-test was used to test the equality of the variances and normality was assessed using the Wilk-Shapiro test. A nonparametric test, the Wilcoxon Sum Rank test, was used to check the results of the two-sample t-test.

The next group of subjects that were compared were those who received the MT to one arm and the UBE treatment to the other arm. Since these scores came from the same subject, they were no longer independent and the two-sample t-test is no longer valid. Because of this a paired t-test was used. The variable assessed was the same
variable assessed in the first series of tests, UBE pain score minus massage pain score. The only assumption that must be met when using the paired t-test is normality. This was assessed during the first series of tests.

When assessing the scores of the group receiving flexion/extension exercise and the MT, independence was again lost. Therefore, a paired t-test was again used to assess the mean differences within this group. The variable assessed was also the same as in the first series of tests, flexion/extension pain scores minus massage pain scores. An appropriate nonparametric test was again used to check the results of the paired t-test. In this case the Sign Test was used.

To date, statisticians do not agree as to when to use parametric tests, such as the t-test, and when to use nonparametric tests, such as the Wilcoxon Sum Rank test and the Sign test. When using parametric tests certain assumptions must be met, as discussed earlier in this section. However, parametric tests are generally considered powerful enough to withstand even major violation of these assumptions without compromising the validity of the results, even when using ordinal data as this study does (Portney & Watkins, 1993, p. 357). When using t-tests it is also possible to calculate the power each test has. In light of the power the t-tests have over nonparametric tests and the fact that the power of each test can be calculated, the author chose to make the t-test the primary statistical tool.

Also of interest was how much power the t-tests demonstrated. Power is defined as the probability of rejecting the null hypothesis (no significant differences between groups) in favor of the alternative hypothesis (significant differences between groups)
given that the alternative hypothesis is true. A power of .80 or greater is considered good. A power analysis was performed using a significance level of 0.05 on the t-tests of each group at the 8 and 48 hour time intervals. A sample size calculation at each 8 hour time interval was also performed to determine how many subjects would be needed to give the t-tests a high power.

UBE Versus Flexion/Extension Groups

There are three assumptions that must be met before the two-sample t-test is valid. Independence has already been discussed. The SAS t-test determined that the two groups were of equal variance and the Wilk-Shapiro test performed on each variable showed that each was normally distributed. Thus, all assumptions were met and all tests using the two-sample t-test were considered valid.

The two-sample t-test performed before exercise and at the 8, 24 and 48 hour post-exercise time intervals resulted in p-values of 0.3019, 0.6115, 0.4424, and 0.6019 respectively. Because of the high p-values it was concluded that there were no significant differences between the mean pain differences of the UBE group and the flexion/extension group at either time interval. The Wilcoxon Sum Rank test performed at each time point verified the results of the two-sample t-test. See Table 1 for two-sample t-test results.

UBE Versus Massage Groups

As stated in Chapter III, the only assumption that must be met when using the paired t-test is that of normality. This was assessed in the previous series of tests and all groups were found to be normally distributed.
Table 1

Two-sample t-test Results--UBE vs. F/E Groups

(Mean Pain Differences)

<table>
<thead>
<tr>
<th>Time</th>
<th>N</th>
<th>Mean</th>
<th>Std Dev</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before DOMS exercise</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>UBE</td>
<td>7</td>
<td>-0.142857</td>
<td>0.377964</td>
<td></td>
</tr>
<tr>
<td>F/E</td>
<td>8</td>
<td>0.000000</td>
<td>0.000000</td>
<td>0.3019</td>
</tr>
<tr>
<td>8 hours</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>UBE</td>
<td>7</td>
<td>0.857143</td>
<td>1.214986</td>
<td></td>
</tr>
<tr>
<td>F/E</td>
<td>8</td>
<td>0.500000</td>
<td>1.414214</td>
<td>0.6115</td>
</tr>
<tr>
<td>24 hours</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>UBE</td>
<td>7</td>
<td>-0.142857</td>
<td>1.214986</td>
<td></td>
</tr>
<tr>
<td>F/E</td>
<td>8</td>
<td>0.375000</td>
<td>1.302470</td>
<td>0.4424</td>
</tr>
<tr>
<td>48 hours</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>UBE</td>
<td>7</td>
<td>0.000000</td>
<td>1.732051</td>
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<tr>
<td>F/E</td>
<td>8</td>
<td>0.375000</td>
<td>0.323899</td>
<td>0.6019</td>
</tr>
</tbody>
</table>

UBE= Upper Body Ergometer Exercise Treatment
F/E= Flexion/Extension Exercise Treatment

The results of the paired t-tests performed on the mean differences between the UBE and massage arms before exercise and at the 8, 24 and 48 hour post-exercise time intervals resulted in p-values of 0.3559, 0.4362, 0.7663 and 1.000 respectively. As a
result of the high p-values it was concluded that there were no significant differences between the mean pain difference scores of the UBE and MT arms at either time interval. The Sign test supported these conclusions. See Table 2 for paired t-test results.

Table 2

<table>
<thead>
<tr>
<th>Time</th>
<th>N</th>
<th>Mean</th>
<th>Std Error</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Before DOMS exercise</td>
<td>7</td>
<td>-0.142857</td>
<td>0.142857</td>
<td>0.3559</td>
</tr>
<tr>
<td>8 hours</td>
<td>7</td>
<td>0.857143</td>
<td>0.459222</td>
<td>0.4562</td>
</tr>
<tr>
<td>24 hours</td>
<td>7</td>
<td>-0.142857</td>
<td>0.459221</td>
<td>0.7663</td>
</tr>
<tr>
<td>48 hours</td>
<td>7</td>
<td>0.0</td>
<td>0.654654</td>
<td>1.000</td>
</tr>
</tbody>
</table>

UBE= Upper Body Ergometer Exercise Treatment
MT=Massage Treatment

Flexion/Extension Versus Massage Groups

As with the previous paired t-test, normality was assessed in the first series of tests and the groups were found to be normally distributed. For the before exercise time period, all scores for both the massage arms and the flexion/extension arms were zero. Since there was no differences, a t-test was not necessary.

The results of the paired t-tests performed on the mean differences between the flexion/extension and massage arms at the 8, 24, and 48 hour post-exercise time intervals resulted in p-values of 0.3506, 0.4423 and 0.2849 respectively. Due to the high p-values,
it was again concluded that there was no significant differences between the mean pain differences scores of the flexion/extension and MT arms at either time interval. Once again, the Sign test supported these conclusions. See Table 3 for paired t-test results.

Table 3

<table>
<thead>
<tr>
<th>Time</th>
<th>N</th>
<th>Mean</th>
<th>Std Error</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>8 hours</td>
<td>8</td>
<td>0.500000</td>
<td>0.500000</td>
<td>0.3506</td>
</tr>
<tr>
<td>24 hours</td>
<td>8</td>
<td>0.375000</td>
<td>0.460493</td>
<td>0.4423</td>
</tr>
<tr>
<td>48 hours</td>
<td>8</td>
<td>0.375000</td>
<td>0.323899</td>
<td>0.2849</td>
</tr>
</tbody>
</table>

F/E=Flexion/Extension Exercise Treatment

MT=Massage Treatment

Power Analysis and Sample Size Calculations

The power analysis performed on the two-sample t-test at the 8 and 48 hour time intervals resulted in powers of 0.20 and 0.09-0.11 respectively. Sample size calculations at the 8 hour time interval showed that in order to achieve a power of 0.80 using the two-sample t-test, a sample size of 48 subjects would be needed.

The power analysis performed on the paired t-tests of the UBE vs. MT group at the 8 hour time interval resulted in a power of 0.46. Since there was no mean pain difference at the 48 hour time interval, neither a power analysis nor a sample size calculation could be performed at this time interval. The sample size calculation for the
UBE vs. MT group at the 8 hour time interval demonstrated that a sample size of 17 would be needed to achieve a power of 0.80.

The power analysis performed on the paired t-tests of the flexion/extension vs. MT group at the 8 and 48 hour time interval resulted in a power of 0.15 and 0.15-0.20 respectively. Sample size calculations performed at the same intervals demonstrated that, in order to achieve a power of 0.80, a sample size of 66 subjects would be needed at the 8 hour interval and a sample size of 48 subjects would be needed at the 48 hour time interval. See Table 4 for power analysis and sample size calculation results.

Table 4

<table>
<thead>
<tr>
<th>Group</th>
<th>Time</th>
<th>Power</th>
<th>Sample Size</th>
</tr>
</thead>
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<tr>
<td>UBE vs. F/E</td>
<td>8</td>
<td>0.20</td>
<td>48</td>
</tr>
<tr>
<td>UBE vs. F/E</td>
<td>48</td>
<td>0.09-0.11</td>
<td>73</td>
</tr>
<tr>
<td>UBE vs. MT</td>
<td>8</td>
<td>0.46</td>
<td>17</td>
</tr>
<tr>
<td>F/E vs. MT</td>
<td>8</td>
<td>0.15</td>
<td>66</td>
</tr>
<tr>
<td>F/E vs. MT</td>
<td>48</td>
<td>0.15-0.20</td>
<td>48</td>
</tr>
</tbody>
</table>

*Power calculated at significance level of p=0.08

**Sample size calculated at a statistical power of 0.80

UBE= upper body ergometer exercise treatment
F/E= flexion/extension exercise treatment
MT= massage treatment
CHAPTER V
DISCUSSION
Relative to the Research Question

The purpose of this study was to compare massage and light exercise treatments administered immediately after eccentric exercise to determine which, if either, was more effective in combating the problems of DOMS. The hypothesis of this study was that no differences would be found in perceived pain between the light exercise and the massage groups. If the results of the t-tests, Wilcoxon Sum Rank Test and Sign Test were all that were considered then we would conclude that the research hypothesis were true.

Unfortunately, when the results of the power analysis were considered, the same conclusion can no longer be drawn. The largest power for the groups and time intervals analyzed was .46. This means that the correct conclusion can be drawn only 46% of the time. In order for the t-tests to achieve an appropriate power (.80), the sample size needed to be between 17 and 66 subjects (see Table 4). If, in fact, the hypothesis is true and neither treatment was more effective than the other, the possibility that both treatments were equally effective cannot be ruled out since there was no control group.

The lack of a control group resulted from an effort to increase the sample size. Those subjects who were to have acted as the control group were added to the treatment groups. Thus, there was no control group to compare with the treatment groups. Therefore, there was no way to compare the effectiveness of either treatment group.
Relative to the Literature

Because of the different variables that were assessed, the variation among the treatment protocols used and the fact that the conclusions drawn from this study are not generalizable outside of this group of subjects it is difficult to make comparisons to past research on DOMS. Smith, et al. (1994), Isabell et al. (1992), Dukes & Ronto (1995), Weber et al. (1994), and Hasson et al. (1989) used the mean pain scores of their subjects as the variable that was assessed. This study used the mean differences in pain scores between arms as the variable that was addressed. Hasson et al. (1989), and Dukes & Ronto (1995), elicited DOMS responses in the quadriceps femoris muscles. Weber et al. (1994), Isabell (1992) and this study chose to use the biceps brachii muscle to elicit a DOMS response. Smith et al. (1994) eccentrically exercised both the triceps and biceps brachii muscles in their study.

Protocols also differed in time of treatment after exercise, duration of treatment, number of treatments and type of exercise used for treatment. Smith et al. (1994) administered a 30 minute massage two hours after exercise while Weber et al. (1994) administered an 8 minute massage immediately after exercise and again at 24 hours after exercise. In this study a 15 minute massage was administered immediately after exercise. The massage application immediately after exercise was chosen because it may be more clinically relevant. In other words, it may be more practical to massage a patient or athlete immediately than to have them return two or 24 hours later.

Isabell et al. (1992) administered exercise treatments at 0, 2, 4, 6, 24, 48, 72 and 96 hours post-exercise. Their protocol was similar to the present study in that their
exercise treatment consisted of flexion/extension of the elbow against gravity for 15 minutes. However, in their exercise treatment subjects exercised for 20 seconds and rested for 40 seconds. In this study the subjects exercised continuously for eight minutes. Weber et al. (1994) administered UBE exercise treatments immediately after exercise and again 24 hours after exercise. The UBE protocol was identical to that used in this study. Hasson et al. (1989) used a high speed (300°/second) isokinetic exercise regimen as a treatment. This exercise treatment was administered 24 hours after DOMS exercise. Although they claimed success, the differences between their treatment protocol and the one used in this study precludes any comparison.

The measurement tools used in previous studies also varied widely. Hasson et al. (1989) used a very complicated system of grids and probes to measure muscle soreness. Isabell et al. (1992) used the Talag scale. This scale rates pain from one to seven and has verbal descriptors at each interval. Dukes & Ronto (1995) and Smith et al. (1994) used the same scale used in this study. However, Dukes & Ronto (1995) and Smith et al. (1994) used the scale to compare pain differences between subjects. In this study the scale was used to compare pain differences within subjects. Since in this study it was assumed that reliability within subjects is greater than reliability between subjects, this may have made the results of this study more valid and reliable. However, until studies are done to assess the reliability of the pain scale used in this and the previously mentioned studies comparisons are again difficult to make.
Limitations

The limitations of this study were greater than originally anticipated. This was, in part, due to the changes that were made during the course of the study. At the start of this study 24 subjects were recruited. Because of a breakdown in the BID only 15 were able to complete the study. Adding the flexion/extension group to the study further reduced the number of subjects in each group so that by the end of the study the largest group consisted of only eight subjects. This small number of subjects per group, combined with the fact that the subjects experienced little difference in pain between arms at each time interval following treatment, created a situation where the t-tests did not have sufficient power to detect significant differences between groups. In fact, with the power scores that were generated, there is a higher probability of drawing the wrong conclusion than drawing the right conclusion.

Another limitation was the subjective nature and thus, the variability, of each subject’s pain measurements. One person’s perception of a rating of “two” may have equaled another person’s rating of “four” and so on. What may have been of more consequence to this study, since this study was concerned with pain differences, was that one person may have rated a pain difference between arms as “one” and another person may have perceived the same difference as a “two.” In order to have offset this variability, a large sample size would have been needed.

Another limitation to this study may have been the measurement tool. Although much research has been done on the reliability and validity of ordinal scales, these scales typically incorporate verbal descriptors at each interval such as function, strength or
assistance which are then transposed into numerical scores. The scale used to measure pain in this study had no such descriptors. For this study it was assumed that since each subject was the best judge of his/her own pain and was assessing the pain between his/her own arms, the reliability and validity of this measurement scale would be high. Although the reliability of the scale is still assumed to be high, there is a question as to the scales validity. The question of the scale’s validity resulted from an informal conversation with one of the subjects after the experiment was completed. This particular subject rated her pain as 9 out of a possible 10. This would indicate that she was in extreme pain. However, she stated that she did not experience a great deal of pain, but that it was the loss of function that bothered her the most. This may have occurred because neither the measurement tool nor the researcher precisely defined what was meant by pain in this study. Therefore, it may have been that the scale did not actually measure pain alone, but rather a combination of symptoms.

Also, unlike the flexion/extension exercise, the UBE protocol was not tailored to the individual. In other words, the UBE protocol may have seemed like light exercise to the stronger individuals, but to the weaker subjects it may have been quite a workout. Completing the UBE exercise after the arm was already fatigued may have enhanced the DOMS response in some way in the weaker subjects.

There was also the chance that subjects participated in an activity before the end of the pain measurement period which may have had an impact on the DOMS response. Subjects were instructed to avoid heavy upper extremity work during the measurement period but their everyday activities or job responsibilities may have required such
activity. Subjects were also asked not to use analgesic creams or medications and to be free of the influence of alcohol when taking pain measurements. There were no other attempts to control what the subjects did after treatment.

Conclusions/Implications to Practice

The results of this study indicated that neither treatment was better or worse than the other. However, considering the low power demonstrated by the t-tests combined with the other limitations discussed above, the conclusions that were drawn from the results are not generalizable to other populations.

Although this study holds no implications for clinical practice, perhaps past studies do. For instance, it is known that eccentric exercise is the foremost cause of DOMS. If it appears that DOMS may present a problem for a patient or athlete, the therapist might want to use eccentric exercise with caution. But, since a bout of DOMS has been shown to have a protective effect against further bouts of DOMS for up to six weeks, perhaps the best defense against DOMS is a regular eccentric workout.

Suggestions for Further Research

One suggestion for further research would be to replicate this study with a larger sample size and use a control group. This would eliminate the low power of the t-tests that was demonstrated in this study and would allow for determining whether either treatment was effective. The exercise treatment protocol should be based on the subject’s individual capabilities, not set at a fixed resistance for a subject. This might eliminate the possibility that the treatment procedure was actually making the DOMS worse. Another
suggestion for replication of this study would be to find a DOMS measurement tool of known reliability.

Although this was certainly not a frivolous study, it may be that it was a little before its time. At this time there have been few studies that have been successful in combating DOMS. To begin doing comparisons at this time may be premature. This author would suggest concentrating on developing DOMS treatment protocols such as Smith et al. (1994) or Hasson et al. (1989) which have shown some promise. Once DOMS treatment protocols have been developed that are consistently effective, comparisons to determine the best treatment approach would be much more successful.
REFERENCES


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Appendix A
Informed Consent Agreement
INFORMED CONSENT AGREEMENT

I understand that this is a study comparing the effectiveness of massage therapy and light aerobic exercise in decreasing the discomfort associated with delayed onset muscle soreness and that the results attained are expected to help various health care professionals prescribe effective exercise protocols for their patients.

I also understand that:

1. Participation in this study requires a bout of vigorous arm exercise which is designed to create slight to moderate muscle soreness which may last five to seven days.

2. After exercise I will receive a 15-minute recovery massage to one arm and complete eight minutes of light aerobic exercise in the form of one-armed bicycling to the other arm.

3. I will be asked to report the degree of muscle soreness that I perceive before exercise and at 8, 24, and 48 hours after treatment using the form supplied to me by Kenneth Newland.

4. Although this exercise is relatively safe for apparently healthy individuals under the age of 45, there are certain conditions which may increase the risk of injury. Therefore, I should consult with my physician before participating in this study if I suffer from any of the conditions listed below:

* less than six months after fractures, strains, or sprains of the upper extremities, back or neck
* rheumatoid or osteoarthritis of the upper extremities, back or neck
* heart disorder or high blood pressure
* osteoporosis
* neurological or neuromuscular conditions which may impair pain perception of muscle contraction

5. I should promptly report to Kenneth Newland any exercise related abnormalities I may experience during the course of the experiment. If I experience any abnormalities my participation in the experiment will be ended immediately.

6. My participation in the study will be kept strictly confidential and data will be coded so that identification of individual participants will not be possible.

7. A summary of the results will be made available to me upon my request.

I acknowledge that:

"I have been given an opportunity to ask questions regarding this research study and that these questions have been answered to my satisfaction."

"My participation in this study is voluntary and that no compensation is being offered or is available for my participation."

"I may withdraw at any time by calling the Chair of the Human Research Review Committee at (616) 895-2472 and Kenneth Newland at (616) 372-3664, without penalty or loss of any benefits to which I may be entitled."
"I hereby authorize Kenneth Newland to release the information from the study to scientific literature. I understand I will not be identified by name."

"I may contact Kenneth Newland at any time if I have any questions."

I acknowledge that I have read and understand the above information, and that I agree to participate in this study.

______________________________  ______________________________
Witness                              Participant's Signature

______________________________  ______________________________
Date                                  Date

____ I am interested in receiving a summary of the study results.
Appendix B

Pain Scales and Recording Form
PAIN SCALE AND RECORDING FORM

Assigned Subject Number ___________  Sex _________  Age___________

Dominant arm______________________

Recording Period Relative to Exercise (Check one)

_____ Before exercising
_____ Eight (8) hours after treatment
_____ Twenty-four (24) hours after treatment
_____ Forty-eight (48) hours after treatment

Using the scale below, please circle the number which best represents the severity of muscle soreness you are experiencing. PLEASE ASSESS RIGHT ARM FIRST.

Right Arm:

0 1 2 3 4 5 6 7 8 9 10

no pain

Excruciating pain

Left Arm:

0 1 2 3 4 5 6 7 8 9 10

no pain

Excruciating pain
Appendix C

Neuromuscular Screen
**NEUROMUSCULAR SCREEN**

__________ Assigned Subject Number

**STRENGTH OF UPPER EXTREMITIES**

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</table>

**RANGE OF MOTION OF UPPER EXTREMITIES**

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<tr>
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**UPPER EXTREMITY SENSATION**

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**UPPER EXTREMITY REFLEXES**

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