

A COMPARISON OF THE EFFECTS OF A  
STRETCHING AND A COLD BEFORE STRETCHING TREATMENT  
ON THE RECOVERY OF RANGE OF MOTION  
AND PERCEPTION OF SORENESS  
FOLLOWING DELAYED MUSCLE SORENESS

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AN ABSTRACT OF THE THESIS OF

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Title: A Comparison of the Effects of a Stretching and a  
Cold Before Stretching Treatment on the Recovery of Range of  
Motion and Perception of Soreness Following Delayed Muscle  
Soreness.

Abstract approved:

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The purpose of this study was to determine the effects of two types of treatment, static stretch and cold prior to static stretch, on the recovery of range of motion of the ankle and perception of soreness of the calf muscle following exercise that causes delayed muscle soreness. Sixty subjects, male and female, ranging in age from 18 to 25, performed 30 sets of a plantar/dorsiflexion movement on one leg. Each set consisted of 15 seconds of exercise followed by 15 seconds of rest. Each subject was measured for ankle range of motion and perception of soreness before exercise and at 24 and 48 hours after exercise. The subjects were assigned to one of three groups: control (no postexercise treatment), stretching (performed six 30 second static stretches), and a cold application before stretch (immersed leg in 57 degree F. water for 15 minutes before

performing the same stretching routine). Treatments were given immediately after exercise and at 3 and 24 hours after exercise.

A fixed-effects model for a two-factor ANOVA was used to analyze the data with a  $p < 0.1$  level of significance. The results indicated no significant differences between groups in relief from soreness or recovery of range of motion. The mean of all three groups decreased in range of motion at 24 hours and slightly increased at 48 hours. The control group showed the greatest recovery of range of motion at 24 and 48 hours, with the cold and stretch group having the slowest recovery during that time. The control and stretching groups decreased in soreness from 24 to 48 hours, with the control group having the greatest amount of relief, although soreness was still apparent. The cold and stretch group had a greater amount of soreness at 48 hours compared to their 24 hour measurement.

The results of this study suggest that treatment with cold plus stretching might have a psychological effect on perceived soreness during the time period in which the treatments are administered (i.e. the first 24 hours). The cold plus stretch group recorded the lowest perception of soreness at 24 hours. While the other two groups decreased in soreness at 48 hours, the cold plus stretch group increased. The control group showed the greatest rate and extent of recovery of range of motion and perception of soreness at 48 hours, although not statistically significant. No treatment prevented the occurrence of DMS.

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## TABLE OF CONTENTS

	Page
LIST OF TABLES .....	vi
LIST OF FIGURES .....	vii
CHAPTER	
1. INTRODUCTION .....	1
Introduction .....	1
The Problem .....	9
Statement of the Problem .....	10
Statement of the Hypotheses .....	10
Significance of the Study .....	10
2. REVIEW OF RELATED LITERATURE .....	13
Introduction .....	13
Theories of Delayed Muscle Soreness ...	13
Lactic Acid Theory .....	13
Muscle Spasm Theory .....	15
Torn Tissue Theory .....	18
Connective Tissue Damage .....	20
Therapeutic Modalities .....	22
Cold .....	23
Stretching .....	23
Combinations .....	24
Measurements of Pain .....	27
Summary .....	31
3. METHODS AND PROCEDURES .....	34

Population and Sampling .....	35
Exercise Procedure to Induce DMS .....	37
Measurement Procedures .....	39
Reliability and Validity Tests .....	39
Range of Motion Measurement .....	40
Soreness Perception Measurement .....	42
Treatment Procedures .....	42
Analysis of Data .....	44
Assumptions and Limitations .....	48
4. ANALYSIS OF DATA .....	50
Introduction .....	50
Range of Motion Findings .....	50
Soreness Perception Findings .....	55
Summary .....	59
5. SUMMARY, DISCUSSION, CONCLUSIONS, RECOMMENDATIONS, & SUGGESTIONS .....	61
Summary .....	61
Discussion .....	63
Conclusions .....	66
Recommendations .....	67
Suggestions .....	68
REFERENCES .....	69
APPENDIXES .....	74
A. Subject Information Sheet .....	74
B. Informed Consent .....	76

C. Photograph of Exercise .....	79
D. Schematic of Range of Motion Instrument..	81
E. Perception of Soreness Form .....	83
F. Reliability Data for ROM Instrument .....	85
G. Photograph of Markings on Foot .....	87
H. Photograph of Use of ROM Instrument .....	89
I. Range of Motion Recording Form .....	91
J. Range of Motion Raw Data .....	93
K. Photograph of Stretching Procedure .....	95
L. Raw Data for Perception of Soreness .....	97



## LIST OF TABLES

	Page
1. Treatment and Measurement Design .....	37
2. Statistical Design .....	47
3. Group Mean & Standard Deviation for ROM .	51
4. ANOVA Summary Table for Range of Motion .	54
5. Mean and Standard Deviation Results for Perception of Soreness .....	56
6. ANOVA Summary Table for Perceived Soreness .....	57

## LIST OF FIGURES

	Page
1. Group Difference From Preexercise for Range of Motion .....	53
2. Group Mean Pain Perception Changes .....	58

## Chapter 1

### INTRODUCTION

An interest in physical fitness has spread during the last few years in the United States. This can be seen by the increased numbers who are participating in road races, triathlons, and aerobics. The advantages these activities have on developing and maintaining a healthy body have been well documented and highly promoted, however, methods of treatment and prevention of the pain and stiffness that often follow working out need to be researched in more depth.

A common occurrence following participation in physical exercise that is more vigorous than usual, especially in untrained individuals, is muscular soreness. There are two types of exercise-related muscle soreness: acute and delayed. Acute soreness arises during and immediately following exercise that is of sufficient intensity to produce a buildup of metabolic waste products, such as lactic acid and potassium. This soreness continues until the exercise intensity is reduced or work stops completely, both of which result in increased blood flow to the exercised muscles. Once blood flow rises, the levels of metabolic by-products decrease and the soreness is removed (Abraham, 1979). The second type of exercise-induced muscle discomfort is delayed soreness. It is not characterized by rapid onset or recovery. This

delayed muscle soreness (DMS) usually appears eight or more hours following the exertion, increases for one or two days, then gradually disappears in about five to seven days. Symptoms associated with this delayed muscle soreness include localized discomfort, a decrease in strength of the affected muscles, and a decrease in range of motion due to muscle tension. These symptoms peak in intensity 24 to 48 hours postexercise (Asmussen, 1979).

Delayed muscle soreness can be very discouraging for someone who does not participate in regular exercise and becomes involved in an occasional pickup game on weekends, for example. Once DMS occurs, it seems that very little can be done to get rid of the soreness for the next few days. In athletics, DMS can be a deterrent in the attainment of an athlete's optimal fitness, especially if a high degree of fitness must be achieved within a limited amount of time. Following the onset of this soreness, one certainly does not feel like engaging in further activity. For the weekend exercise enthusiast, that may merely be bothersome. But for an athlete, any situation which limits one's ability to practice can have a serious detrimental effect on performance. Therefore, the aim of this study is to investigate treatments which may prevent the occurrence of or reduce the recovery time associated with delayed muscle soreness.

What actually causes DMS is not totally clear. Since

Hough's (1902) initial study in 1902, the pain associated with delayed muscle soreness has been studied by various groups of investigators, resulting in four theories attempting to explain the condition. These theories are: lactic acid accumulation, (Asmussen, 1956), muscle spasms (deVries, 1961), torn tissue (Hough, 1902), connective tissue damage (Abraham, 1977). Current literature has cast doubt on the validity of the lactic acid theory due to lack of supporting evidence. Support for the muscle spasm theory decreased with the emergence of evidence for the theories regarding structural damage to either the muscle tissue itself or to the connective tissue. The muscle spasms were thought to be a process which occurs following the initial structural damage and were not the causal factor for DMS itself (Friden, Sjostrom, & Ekblom, 1983, Francis, 1983).

Although several studies have shown evidence of some form of structural damage, they have failed to determine why the pain does not follow the damage immediately, as experienced in other injuries. Brendstrup (1962) hypothesized that since the tissue damage triggers an inflammatory process, the time required for the formation of edema to take place could explain the delay between the trauma and the perception of soreness. Knight (1978) stated that after any musculoskeletal injury, pressure due to inflammation or damage to the nerve fibers causes a

muscle to spasm in order to protect itself from further injury. This pressure causes pain, and the body reacts by increasing muscle spasm, which then increases the pain, etc. He appropriately called this the "pain-spasm-pain" cycle. In the case of minor injuries such as DMS, the gradual increase of this cycle may fit the time course in the 24-48 hours delay of DMS.

DeVries (1986) contends that there is still some merit for the muscle spasm theory based on an entirely different mechanism for the production of pain. He indicated that localized spasm caused compression of blood vessels within the muscles. This compression of blood vessels results in ischemia which triggers the perception of pain. The pain then causes further muscle spasm which in turn causes more ischemia and pain. Eventually, the lack of blood flow to the muscles causes an inflammatory process which may lead to tissue damage.

Although the theories differ in regard to the actual cause of DMS, a common agreement is that an inflammatory reaction takes place at the site of the affected tissues. Brendstrup (1962) stated that anyone experiencing delayed muscle soreness, regardless of its duration or intensity, is probably suffering from some degree of acute inflammation. However, the direct cause of inflammation has not been determined. If inflammation is the initial reaction that takes place with the onset of DMS, as

proposed by the tissue damage theories, then treatments known to reduce inflammation should be beneficial in preventing development of further symptoms, such as pain and stiffness. If other factors proposed by the spasm theory, such as muscle spasms and ischemia, eventually result in an inflammatory reaction, then early treatment for reduction of inflammation may fail to yield positive results. In the case of DMS, discovering successful treatment techniques may lead to finding which mechanisms are responsible for its occurrence.

The most noticeable symptom of DMS is the onset of soreness or pain. Pain is thought to be the body's warning sign that injury is about to occur. This seems to be in contrast with the theories that structural damage is the initial phase since the pain is delayed. Increases in pain in DMS have been moderately correlated with decreases in strength (Talag, 1973), and decreases in range of motion (Yackzan, Adams, and Francis, 1984). Therefore, in light of the muscle spasm theory, if the pain-spasm-pain cycle can be stopped before any damage occurs, then it would seem reasonable to assume that range of motion would remain unaffected. Decreases in strength and range of motion may be due to the inhibition of effort due to the presence of pain and not because of any physiological damage to the tissues (Friden, 1984).

Many therapeutic modalities have been advocated for

treatment of all types of musculoskeletal trauma. Although there is general disagreement over their uses, cryotherapy (the use of cold), thermotherapy (the use of heat), and exercise have been the most popular forms of therapy used by athletic trainers.

Cryotherapy (cold treatment) has become the most popular modality to use in most musculoskeletal injuries, especially immediately after and up to 48 hours following the injury. Cryotherapy is known to decrease muscle spasm which prolongs the relaxation of the muscle, thereby increasing range of motion and allowing earlier mobilization of the muscles (Knight, 1978). Cryotherapy also decreases blood flow which further reduces inflammation and edema, and decreases pain (Lehmann, 1982)). Since physiological effects of cold treatments are inversely related to the symptoms of DMS, research suggests that cryotherapy should be an ideal treatment choice (Francis, 1983).

Exercise is a popular form of treatment used to rehabilitate an injured muscle. In acute injuries, exercise may not be utilized immediately, but establishing an exercise program, especially in the form of stretching, as soon as possible is one of the most important phases in returning a person to normal activities in the shortest amount of time (Houglum, 1977). Exercise, in the form of stretching has been shown to relieve symptoms such as pain



and spasm after exercise that caused muscular soreness (deVries 1961).

Use of some form of a cold application along with exercise is a technique that was popularized in the 1960's by Hayden (1964) and Grant (1964). They found positive results using cold treatments prior to range of motion exercises in military patients who sustained sprains, strains, and contusions during training. They both cited that active exercise is what rehabilitated an injured area; ice acts only to decrease pain and allow for an earlier active motion. Yackzan, Adams, and Francis (1984) found that cold treatment alone had no significant effect on reducing the pain or increasing range of motion associated with DMS. Prentice (1982) examined the effects of the combinations of cold, heat, and stretching (static and PNF) and found positive results in each combination for reducing delayed muscle pain. Treatments using cold with static stretch proved to be the most effective treatment combination.

It is interesting to note that, although cold therapy is known to relieve symptoms associated with DMS, the use of cold alone has failed to produce positive results as a treatment. Cold continues to be used in combination with other forms of treatment. Since cold also provides an anesthetic effect when applied, it may prove to have more of a psychological effect than physiological effect in treatment of DMS.

Since it is known that DMS usually takes five to seven days to disappear, it seems evident that finding an effective method of preventing development of or speeding the recovery from DMS is an essential factor in maximizing the efforts of athletic training and performance. In most cases, some form of treatment after injury seems to speed the recovery time as shown in the studies by Hayden (1964) and Grant (1964). They reduced the recovery time from sprains, strains, and contusions by several days using cold and exercise over no treatment or rest. In some cases, however, simple rest seems to be the only answer to recovery. This may be due to the fact that an effective treatment has not been found for that particular injury or that the body's process for repair after injury takes a certain amount of time regardless of any form of treatment applications.

Although various studies have shown stretching and cold with stretching to be effective in treating DMS, no studies have investigated the differences between stretching as opposed to cold with stretching. The purpose of this study is to determine if there is a difference between the use of a stretching protocol and the use of cold application plus a stretching protocol on the recovery of range of motion and/or relief from pain following exercise designed to induce delayed muscle soreness. By using the same stretching technique in both cases, this

study is designed to determine if the use of cold has any added benefit in relieving or preventing symptoms associated with delayed muscle soreness.

### The Problem

Research concerning effective means of treating muscle soreness indicates that both cold with stretching and stretching alone have produced some desired effects. However, no studies have been found which compare the effectiveness of treatment by stretching alone to that of treatment by use of cold plus stretching.

Most individuals who engage in a type of physical activity to which they are unaccustomed are unaware that they may experience discomfort several hours after cessation of the exercise. Therefore, they will probably fail to consider any type of immediate treatment in an effort to prevent delayed muscle soreness from occurring. In cases where DMS can be anticipated, such as in athletics, a proper treatment can be administered with the intent of preventing the occurrence of delayed muscle soreness. The information from this study will enable anyone who suspects the onset delayed soreness following exercise to make a more valid decision when selecting methods for preventative treatment of delayed muscle soreness.

### Statement of the Problem

Is there a significant difference between stretching alone and stretching after cold water immersion on the extent and rate of recovery of range of motion following exercise that causes delayed muscle soreness?

Is there a significant difference between stretching alone and stretching after cold water immersion on the rate and extent of perception of soreness following exercise that causes delayed muscle soreness?

### Statement of the Hypotheses (Null Forms)

There is no significant difference between stretching alone and stretching after cold water immersion on the rate and extent of recovery of range of motion following exercise that causes delayed muscle soreness.

There is no significant difference between stretching alone and stretching after cold water immersion on the rate and extent of perception of soreness following exercise that causes delayed muscle soreness.

### Significance of the Study

Although exercise should be done at a level that avoids delayed muscle soreness, it is very difficult for a coach or any individual to know at which point continuation of exercise will cause DMS. By determining which of the

of exercise will cause DMS. By determining which of the two methods is more effective in treating DMS, that treatment can be prescribed as a precautionary measure following a workout that is thought to cause DMS. This may alleviate or prevent the symptoms associated with delayed muscle soreness, thus reducing the amount of time a person spends recovering from DMS, thereby increasing effective training time.

Various studies have investigated several combinations of cryotherapy and exercise in attempts to obtain relief or to rehabilitate an injury to the musculoskeletal system. However, most of the results have been empirical due to the difficulty of studying acute injuries in a controlled situation. For experimental purposes dealing with DMS, however, it is possible to induce soreness in subjects in order to help control as many intervening variables as possible. Nearly all previous research which induced soreness did so by forcing the muscle to exertion far beyond that to which it is accustomed, usually to the point of exhaustion or total fatigue. This study is designed from a more practical stand point in that the type of soreness experienced in athletes after some workouts is usually of the mild form. The exercise used has been found to cause mild soreness and does not exercise the subjects to total fatigue (Bobbert, Hollander, and Huijing, 1986). No studies have been found using the techniques and procedures commonly used in

training rooms and advocated by Knight (1978) for the use of cryotherapy on musculoskeletal injuries. Knight suggests that immediate treatments for soft tissue injuries should be administered two to five times the first 24 to 48 hours. Most of the studies have not initiated a treatment until symptoms became apparant (24 hours postexercise) or used only one or two treatments for the duration of the study. Since prevention of, or quicker relief from, soreness is the aim of the study, three treatments were administered within the first 24 hours. The treatment times were designed to simulate a practical approach of a routine that an an athlete might take as a precautionary measure for early treatment of delayed muscle soreness. That is: apply a treatment immediately following an afternoon workout; apply the treatment once later that evening; and apply the treatment again prior to the workout the following day.

## Chapter 2

### REVIEW OF RELATED LITERATURE

#### Introduction

The review of related literature is divided into three parts. Part one includes the rationale behind the four theories relating to delayed muscle soreness, along with the support that exists for the mechanisms responsible for the occurrence of the known symptoms. Part two deals with the research on the uses of therapeutic modalities, specifically cold and stretching which were used in this study. Part three provides an insight into how research has dealt with measuring pain in human subjects.

#### Theories of Delayed Muscle Soreness

##### Lactic Acid Theory

There are generally two types of muscular pain associated with severe exercise: immediate pain which occurs in the final stages of exercise and lasts up to a couple of hours after the exercise has stopped and the residual or delayed type which becomes apparent eight or more hours following exercise. Both of these types of pain were first thought to be a result of lactic acid accumulation (Francis, 1983). The symptoms of stiffness, tenderness, decreased strength, and edema have been

associated with both the immediate pain (Brendstrup, 1962, Abraham, 1979) and the delayed pain (Assmussen, 1956, Friden, 1984).

The theory that lactic acid accumulation causes fatigue and pain immediately following severe exercise has been proven and is generally undisputed in current research. Even though there have been no studies to substantiate it, the belief that lactic acid may in some way be a contributing factor in the onset of delayed soreness still exists after it was first proposed nearly 50 years ago (Armstrong, 1984).

There are considerable data that argue against the lactic acid theory. The most convincing evidence comes from the muscle contractions that cause the greatest degree of soreness. It is undisputed that the eccentric phase of muscle contraction is the primary cause of muscle soreness. An eccentric type of exercise requires a lower amount of oxygen consumption and ATP production than a concentric contraction at the same workload. Also, fewer motor units are recruited for the same amount of work and the buildup of waste products is much less than in a concentric contraction (Friden, 1984). Therefore, more strain can be placed on a muscle which is acting eccentrically before fatigue forces the subject to stop.

Recently, Waltrous, Armstrong, and Schwane (1981) performed a study which also cast doubt on the validity of



this theory. They ran subjects on a treadmill for 45 minutes at a 0 and -10 degree incline. They found that when the subjects ran on the flat treadmill at 78%  $\text{VO}_2$  max., they showed a significant accumulation of lactic acid but none reported delayed muscle soreness. However, when the subjects ran downhill at the same speed, requiring 58%  $\text{VO}_2$  max., the subjects accumulated only small amounts of lactic acid but reported severe soreness.

Current studies trying to relate lactic acid to delayed muscle soreness are scarce. Within the last 10 to 15 years, researchers have focused their efforts on testing other ideas concerning the cause of delayed muscle soreness.

### Muscle Spasm Theory

Electromyography (EMG) has been used in numerous studies to measure the electrical activity of the muscles. There are three different EMG techniques that have been used in research concerning delayed muscle soreness. They include surface electrodes, unipolar and bipolar, which are placed on the skin over the area of muscles to be measured, and needle electrodes which are inserted into the muscle. In 1961 and 1966, deVries reported experiments in which he measured the electrical activity in sore muscles using a unipolar electrode technique. He has proposed that his findings support the theory that muscle pain initiates a

positive feedback cycle leading to muscle spasms, which in turn causes more pain. The magnitude of this pain-spasm-pain cycle is dependent upon the number of motor units involved. He indicated that the level of pain symptoms and the recorded EMG activity were directly related. Abraham (1977) tried to reproduce deVries' study using bipolar electrodes and found no significant relationships between the amount of EMG activity and muscle soreness. DeVries contends that the bipolar and needle EMG's are not sensitive enough to record the electrical activity. However, Newham, Mills, Quigley, and Edwards (1983) used unipolar EMG and failed to find any localized spasm during sensation of pain as reported by deVries. The validity for the use of EMG as a measuring instrument for the degree of soreness in muscles remains in question.

With the increase of research findings supporting the theory that some structural damage has occurred causing delayed muscle soreness, most researchers have dismissed the spasm theory as a probable cause. DeVries (1986) still believes that the spasm theory is the cause of the soreness. It is commonly accepted in the medical profession that a major function of pain is to protect the body from further injury. Delayed muscle soreness does not seem to fulfill this role since the pain does not immediately follow the exercise. Through research, an untrained muscle has shown its inability to completely

relax following exercise that causes fatigue (Petajan and Eagan, 1968). The contracted condition of the muscle causes increased intramuscular pressure (ischemia) which limits the recovery process of that particular area. Edema is also present around the affected muscles. Though it is not known what causes edema, Brendstrup (1962) concluded that it could be (1) intracellularly provoked by chemical processes in the muscle cell, (2) extracellularly provoked by injury outside the muscle fibers, especially the connective tissue, or (3) extracellularly provoked by an increase in permeability of the capillaries. The accumulation of waste products then causes the muscle to spasm and the pain-spasm-pain cycle is born. DeVries contends that muscle damage occurs because of the inability of waste products to be removed from the area due to ischemia. The delay in pain is a result of the time it takes for ischemia to have a damaging effect on the involved tissues.

In a study by Bobbert, Hollander, and Huijing (1986), 11 subjects performed exercise resulting in DMS in the gastrocnemius of one leg. The other leg served as the control. The exercise consisted of 30 sets of 15 seconds of toe-raises on a 2.0-cm thick board with a 15-second rest interval. Soreness perception and EMG levels were reported at 24, 48, and 72 hours postexercise. They found that the increase in soreness perception was not accompanied by an

increase in resting EMG level. Although their conclusions indicated that soreness perception was not related to a tonic localized spasm, they did not completely dismiss the possibility of the muscle spasm theory. All 11 subjects reported soreness from the exercise with three having increases in EMG activity corresponding to their level of perceived soreness. They concluded that there is a large interindividual variation in resting EMG levels of sore muscles. Thus, it is imaginable that the muscle spasm theory is responsible for soreness perception provided that the spasm is severe enough to cause partial occlusion of blood vessels, which thereby leads to ischemia.

### Torn Tissue Theory

In 1902, exercised induced muscle soreness was first described and studied by Hough (1902). He found that this kind of pain did not vary with intensity of fatigue but that it was closely associated with mechanical tensions in the muscles. He theorized that an untrained muscle group subjected to prolonged periods of work may experience structural damage and change in muscle morphology. Until the last few years, this theory was generally speculative. Recently, however, several studies have added new significance to this idea (Francis, 1983). Friden, Sjostrom, and Ekblom (1981) found structural damage to the contractile apparatus of the muscle fibers (Z-bands) in the

thigh following exercise which caused delayed muscle soreness. Twelve male physical education students ranging in age from 18 - 32 reported severe soreness 18-72 hours following a 30 minute exercise on a bicycle ergometer modified for use in eccentric work. Work intensity for the exercise was the same as the one which during concentric exercise corresponded to 80% to 100% of individual  $\text{VO}_2$  max. Two subjects failed to maintain the stipulated time due to complete exhaustion. Muscle biopsies were obtained from three subjects immediately postexercise, from six subjects three days postexercise, and from three subjects six days postexercise. Z-band disorganization was apparent immediately following exercise in 32% of the fibers, while corresponding values three and six days after exercise were 52% and 12% respectively.

Although this study proved that damage occurred to the tissues, the question of whether the mechanical overload was primarily or secondarily responsible for the Z-band disruption remains unclear. The alterations in permeability of the cell membrane due to a series of chemical disturbances resulting from high mechanical forces produced by eccentric exercises may have caused a weakening of the Z-band material. This chemical imbalance could provoke edema and soreness, leading to further damage as shown 3 days postexercise. This study offered another explanation for the increase in damage three days

postexercise. It may be due to persisting tension which could stretch and further damage the already defective material. Armstrong (1984) stated that these chemical alterations are very complicated and will require more studies dealing with the basic cellular and biochemical mechanisms involved in delayed muscle soreness. In short, damage to the muscle fibers has been found to be related to delayed muscle soreness, but the causative factors underlying the mechanisms responsible for the damage remain unclear.

#### Connective Tissue Damage .

The last prominent theory that has been investigated as an explanation of delayed muscle soreness is that connective tissue, including tendons, is damaged during muscular contraction. In a study designed specifically to investigate this theory, Abraham (1977) correlated urinary excretions of hydroxyproline (OHP) with reports of muscular soreness. OHP is known to be a specific breakdown product of connective tissue. He observed that OHP levels were highest at 48 hours in subjects who reported maximum soreness following eccentric contractions. Subjects who performed concentric contractions had no significant changes in levels of OHP. A study by Tullson and Armstrong (1968) provided additional support when they observed an increased inflammatory response in the connective tissue

elements of skeletal muscle in untrained rats 24 hours following a prolonged low intensity exercise session. In another study involving rats, Brendstrup (1962) demonstrated that the development of edema in experimental animals agreed in time (6 to 24 hours) with the appearance of the soreness of muscle in man following exercise. He stated that soreness may be related to the tension which the edema causes in the fibers of the connective tissue.

Newham, Mills, Quigley, and Edwards (1983) conducted an experiment on four subjects who performed a step test in which one leg contracted concentrically while the contralateral muscle contracted eccentrically. They concluded that the damage was due to the extreme mechanical loading of the elastic component during an eccentric contraction. Subjective measures of pain were reported in the eccentrically contracted leg with no pain reported in the concentrically contracted leg. Strain gauge measurements showed the area of soreness was located at the distal, medial, and lateral parts of the quadriceps (i.e. the areas of musculo-tendinous attachments). These findings were in agreement with the conclusions of Asmussen (1956) that the cause of pain and tenderness is due to the over-stretching of elastic non-contractile tissues. This also supports Abraham (1977) who reported rises in hydroxyproline in subjects who performed eccentric contractions.

It is apparant that each proposed theory of delayed muscle soreness, with the exception of the lactic acid theory, agrees that some form of damage occurs at the site of the exercised muscle. Symptoms of edema formation or inflammation, muscle tension, decreased strength, and soreness are evident regardless of the theory proposed. The main difference between the spasm theory and the tissue theories is which comes first, tissue damage or muscle tension.

### Therapeutic Modalities

Various forms and application techniques of modalities have been proven effective in reducing the recovery time of nearly all types and degrees of injuries. In the case of acute injuries, the initial goal is to prevent further damage from occurring. The physiological basis behind any type of treatment following injury is to accelerate the healing process of the body. The healing time depends mainly on the blood supply, which carries the necessary elements such as nutrients, oxygen, and inflammatory cells to the site of the injury (Southmayd & Hoffman, 1981). If given the time, the body has the ability to heal most injuries. The use of therapeutic modalities is based on the idea of reducing that amount of time.



## Cold

A number of physiological responses to cold have been found to have therapeutic importance concerning delayed muscle soreness and soft tissue trauma in general. Lehmann (1982) identifies cold treatment (cryotherapy) as being successful in decreasing edema production. As stated earlier, there is formation of edema with the onset of delayed muscle soreness regardless of which theory is proposed. Knight (1978) states that the use of cryotherapy will decrease muscle spasm. Local application of cold causes a decrease in sensory nerve impulses and decreases the stretch reflex, resulting in a local anesthesia. This causes a decreased pain sensation and the decreased stretch reflex results in decreased muscle spasm, thus the pain-spasm-pain cycle is broken. Even a very short-term interruption of the pain stimulus may break the cycle and provide relief for a prolonged period.

## Stretching

Stretching has been shown as being beneficial in treating delayed muscle soreness. DeVries (1961) has found that stretching was successful in relieving soreness following exercise that caused muscular soreness. Two women and 15 men ranging in ages from 20-29 performed four minutes of wrist hyperextensions using a 9 pound weight. Both arms were exercised simultaneously at a cadence of 30

wrist hyperextensions per minute. Immediately following the exercise and at 2, 6, 20, and 22 hours following the exercise, the wrist flexors, and then the wrist extensors, were statically stretched for one minute on the experimental arm of each subject. Subjects recorded their degree of soreness at 4, 8, 24, 48, and 72 hours postexercise. He found lesser degrees of soreness in the experimental arm than the control arm at every observation time but only the 24 and 48 hour times were found to be statistically significant. DeVries (1966) also found lower EMG activity and relief from soreness 48 hours after repeated arm curls in subjects who performed stretching exercises as opposed to subjects who did not stretch . Although Abraham (1977) did not find any differences in EMG activity at 24 or 48 hours in 11 subjects who performed repeated arm curls to exhaustion, he did find static stretching to have a significant effect on pain reduction. However, the stretching provided relief for only a couple of minutes.

### Combinations

Cold in combination with static stretch has been recommended for reducing muscle spasm or decreasing delayed muscle soreness. Cold is applied over the painful muscle using ice massage, ice packs, or through cold water immersion. Immediately following the cold application, the

exercise is carried out with the idea of returning the muscle to its normal resting length. For decreasing muscle spasm, Knight (1978) recommends the combination of cold and static stretching as being a proper method for breaking the pain-spasm-pain cycle. Grant (1964) reported that over a two and one-half year period, he treated more than 7,000 patients who suffered from various musculoskeletal injuries with cold followed by mobilization exercises. Although his study was not a controlled experiment, he believes that the fact that over 80 percent of the patients received less than three treatments attested to the validity that early motion was the key to recovery and that the cold treatments acted only to relieve pain in order to allow that early motion.

Prentice (1982) used EMG to evaluate the effectiveness of heat and cold in conjunction with either static stretching or a technique of proprioceptive neuromuscular facilitation (PNF) stretching to determine which combination was most effective in relaxing muscles which exhibited delayed muscle soreness. Twenty-four hours after totally fatiguing the hamstring muscles on a Universal knee machine, 50 subjects were assigned to one of the possible combinations of treatments. After EMG recordings were taken, the subject applied either heat or cold packs for 20 minutes then immediately performed 3 sets of 10 seconds of either static stretch or PNF stretch. The

results indicated that the use of cold followed by static stretch was more effective than the other methods in reducing delayed pain. Cold followed by either form of stretching was more effective than either treatment using heat. He also concluded that both stretching exercises were equally effective in reducing muscle pain.

The implications for using cold and stretching in conditions such as muscle spasm may have some validity as shown by research. However, Yackzan, Adams, and Francis (1984) investigated the effectiveness of 15 minutes of ice massage in reducing delayed muscle soreness to determine the optimal time within a stated protocol. Three groups (10 per group) receiving ice immediately after exercise, those receiving it 24 hours after exercise, and those receiving it 48 hours after exercise were studied. The exercise used to induce soreness was a repeated eccentric contraction of the elbow flexor muscles to complete exhaustion. Pain scores were determined by the use of a 7-point rating scale and elbow range of motion was assessed using a full circle plastic goniometer. There were no consistent patterns of significance differences on the comparisons between the treated versus the untreated arms and of the treated arms that were treated at different times. This study failed to demonstrate that cold alone was effective in preventing the occurrence of or relieving pain in delayed muscle soreness.

### Measurements of Pain

There have been a variety of methods used in evaluating levels of pain in experimental and medical research. Attempts have been made to measure pain as non-subjectively as possible with the use of instruments. One of these methods employs the use of a strain gauge pressure probe. A round-ended wooden probe is attached to a strain gauge which measures the amount of pressure applied to specific areas of a subject's muscle. The wooden probe is slowly pressed against the skin and the subject is asked to indicate verbally when the sensation of pressure changes to one of discomfort. Numerous sites can be tested on the same muscle group, showing the degree and distribution of tenderness (Newham, Mills, Quigley, and Edwards, 1983). DeVries (1966) found that increased muscle electromyographic (EMG) activity correlated closely with levels of pain in his subjects. Studies by McGlynn, Laughlin, and Rowe (1979) and Cobb, deVries, Urban, Leukens, and Bagg (1975) reported comparable findings. However, similar studies (Abraham, 1979, Bobbert, Hollander and Huijing, 1986) failed to find consistent correlations between EMG readings and the subject's pain sensations. Prentice (1982) concluded that perception of pain is subjective and tends to be variable from one individual to another. Consequently, it is probable that each subject

could rate levels of pain differently, thus reducing the reliability of rating scales. Prentice feels that the best non-subjective measurement of pain used in research thus far, dealing with delayed muscle soreness, is the EMG measurements of muscular activity.

The assessment of subjective responses to pain require an accurate, reliable, and sensitive method. A valid recording made by the subject seems to be the preferred method of doctors and psychologists over other types of external measuring devices (Huskisson, 1974). Bond (1979) states that although self reports of pain are subjective measurements, they are likely to be more reliable than having a person not experiencing the pain try to evaluate the level of pain in another person. He suggests the use of a visual analogue scale which allow the patients to indicate the amount of pain experienced by marking on a 10-cm long line. The distance of the mark from the left end of the line is measured in millimeters and called a pain score. The only descriptive markers on the line were at each end of scale, the left side states 'no pain' and the right side states 'pain could not be worse'. He felt this scale was more sensitive in determining changes in pain than the use of an analogue scale which divides the line into levels or fixed points such as, some pain, considerable pain, extreme pain, etc. He advocates the use of the visual analogue scale for measurements of chronic

pain over long periods of time, or with patients rating effectiveness of pain-relieving drugs where the subject uses the scale several times in one day. Levine, Gordon, Jones, and Fields (1978) found the visual analogue scale to correlate better than 95% with verbal reports of change in pain levels. However, Joyce, Zutshi, Hrubes, and Mason (1975) performed a study comparing the two types of scales, a visual analogue scale (VAS) and a fixed point scale (FPS). The VAS consisted of a 10-cm long line with the extremes marked with "no pain" and "pain could not be more severe". The FPS was the same but added the words "some pain" and "considerable pain" equally divided along the line for use as reference marks for the subjects. To provide an external standard of reference, the efficacy of two doses of each of two commonly used analgesics were compared in patients attending a busy outpatient department for the relief of chronic inflammatory pain. The results indicated that scores showed very little difference between the type of scale used to mark pain. The VAS seemed to be more difficult for the patient to understand and may, therefore, be less reliable than a FPS. They concluded that a VAS could be a more sensitive measurement for patients who repeatedly used the scale, such as in rating chronic pain over an extended period of time. They suggested the use of a FPS for patients who were not familiar with or accustomed to filling out pain measurement

scales.

Researchers using self-reports for measuring levels of pain involving delayed muscle soreness have used a variety of unique scales for recording soreness changes. It was found that as the studies using rating scales became more current, the scales more closely resembled a visual analogue scale. Asmussen (1956) simply used three scores: no soreness, palpable changes, and soreness. DeVries (1961) used a chart on which subjects indicated degrees of soreness given four magnitudes: none, mild, moderate, and severe. Talag (1973) used a 0-7 rating grid with units of .25 between each division. Subjects were able to indicate soreness in 29 different degrees of magnitude. Bobbert, Hollander, and Huijung (1986), assessed soreness scores using a Visual Analogue Scale with the extreme ends marked with "I do not have any soreness" and "My soreness could not be worse".

Although the studies mentioned have all used different methods of subjectively measuring soreness in their subjects, their purpose and intent was similar. There is no indication of one method being more valid than the other. Some form of a 10 cm analogue scale, where the scores can be measured in greater divisions (0-99) may be more sensitive to differences in soreness. Placing reference marks on the analogue scale may prove useful for subjects not familiar with marking an analogue scale and



provide consistency to the interpretation of various distances along the line.

### Summary

The mechanisms that cause delayed muscle soreness are poorly understood and, therefore, most studies thus far have been of a descriptive nature. Although conflicting studies are abundant, each researcher has presented evidence supporting his findings. Another problem concerning the studies involving delayed muscle soreness, besides the uncertainty of the causative factors, is the inconsistency of procedures for inducing soreness, and varying methods of measuring pain and tissue damage.

It is generally agreed that the most predictable way of producing delayed muscle soreness is to have the muscles perform eccentric contractions, and that the degree of soreness is related to both intensity of the muscular contraction and the duration of the exercise. Therefore, subjects need not be exercised to exhaustion or total fatigue in order to induce soreness.

Most research agrees that structural damage occurs with exercise that causes delayed muscle soreness, and that edema formation and muscle tension are evident during that process. Therefore, treatments known to alleviate muscle tension and edema in the form of cryotherapy and exercise

have been tested and found successful.

The lactic acid theory is known to cause fatigue and pain immediately following exercise due to the buildup of metabolic wastes, but research has not been able to substantiate its relation to delayed muscle soreness. The muscle spasm theory proposes that that the structural damage is due to the inability of the muscle to relax following an increase in muscle force. This causes ischemia, which leads to edema and tissue destruction. The torn tissue theory suggests that structural damage within the muscle cell following high mechanical forces is due to a series of chemical disturbances which become toxic, provoke edema, and eventually cause destruction and soreness. The connective tissue theory is supported by the fact that most soreness is apparent in the areas where the connective tissue is located. It is thought that excess tension produced by eccentric contractions damages the elastic non-contractile parts of the muscle. This creates an inflammatory response and edema formation which puts pressure on the nerve fibers causing inflammation or the formation of edema which in turn causes pain.

Treatments known to be beneficial in preventing or relieving symptoms associated with delayed muscle soreness include stretching and the use of a cold modality. These too have come under some dispute as to their effectiveness as a treatment. With the formation of edema and the

possibility of muscular spasms, a cold treatment seems to be an ideal choice for alleviating symptoms of delayed muscle soreness. Studies have also demonstrated success with relieving soreness through static stretching exercises alone.

A variety of methods have been used to subjectively measure pain associated with delayed muscle soreness. Several forms of scales have been designed by researchers that allow subjects to distinguish various levels of pain. The use of an analogue scale seems to be more sensitive and reliable than other methods used. Furthermore, an analogue scale which has equal divisions along the line to assist the subject in selecting a consistent level has been proposed. Thus, a graded analogue scale appears to be best suited for measuring soreness in subjects with delayed muscle soreness.

## Chapter 3

### METHODS AND PROCEDURES

The purpose of this study was to compare two types of treatments for relieving symptoms associated with the onset of delayed muscle soreness. Data were collected and statistical procedures were utilized with the intention of answering the question: Is the use of a cold and stretch treatment more beneficial than a stretching treatment alone for relief from soreness and the recovery of range of motion (ROM) following exercise that causes delayed muscle soreness? The rate at which the treatments affected ROM and perception of soreness, as well as the extent to which the treatments affected ROM and perception of soreness, were examined. Research indicates that symptoms of delayed muscle soreness peak in intensity between 24 and 48 hours (Asmussen 1956). This study was designed to apply two treatments for the relief of symptoms associated with DMS within the first 24 hours postexercise. Measurements of perception of soreness and range of motion were taken at 24 and 48 hours to determine what effects the early treatments had on those measurements.

The treatment procedure was designed to simulate a realistic and practical approach of a routine that an athlete might take as a precautionary measure for early treatment of delayed muscle soreness. If an athlete completed a workout and anticipated the possibility of it

causing soreness the following 24 to 48 hours, a probable treatment procedure might be as follows: apply a treatment immediately following the workout, which is usually in the late afternoon; apply the treatment once later that evening; and apply the treatment again before the workout the following day.

### Population and Sampling

Sixty male and female students ranging in age from 18 to 25, who were enrolled in PE 100 Lifetime Fitness classes at Emporia State University, volunteered to take part in the study. The subjects met the following requirements in order to qualify for participation in the study: they must have been free of any injury of the lower leg or foot within the last 3 months and they could not have been involved in a varsity sport at Emporia State. For the duration of the experiment, the subjects were asked to abstain from any type of exercise activity to which they were unaccustomed. They were also instructed not to stretch or apply any form of treatment such as heat, cold, or massage to the involved leg during the 48 hours of the investigation. Prior to their consent, they were informed of the reason for the experiment, the amount of time involved, the procedures used, and the risks involved. They were given the opportunity to ask any questions

concerning the treatments and procedures used in this study. They were informed that they could withdraw from participation in the study at any time. All subjects then filled out the subject information sheet (Appendix A) and signed an informed consent form (Appendix B) in accordance with the policy used by Emporia State University.

Each of the 60 subjects was randomly assigned to one of three groups (20 per group). Group I served as a control. Although they performed the exercise to induce soreness, no postexercise treatment was administered. Group II received the stretching treatment. Group III received the cold and stretching treatment. A description of the three groups' involvement for each session is shown in Table 1. After being assigned to one of the three groups, each subject worked out an appropriate schedule for the required sessions. In order to avoid conflicts with classes, subjects were allowed to schedule the final three sessions with a leeway of plus or minus one hour around the prescribed time.

Due to the unique measuring instrument used for determining range of motion in the ankle, only the left leg of each subject was included in the study. This instrument will be described in detail later in the chapter. Through the design of the exercise protocol (i.e. allowing each subject to choose their frequency and amplitude of the toe-raises), it is assumed that the stronger the subject's

Table 1. Treatment and Measurement Design

	Time After Exercise (h)			
	0	3	24	48
Group I			X	X
Group II	S	S	XS	X
Group III	C	C	XC	X

S = Stretching treatment

C = Cold with stretching treatment

X = Soreness and ROM measurements

Note: All subjects performed the same exercise to induce soreness and were pretested for ROM and soreness perception.

legs, the more repetitions and greater intensity would be performed, therefore, eliminating the need to establish a dominant or stronger leg for each subject.

#### Exercise Procedure to Induce DMS

Research has not proposed a standard procedure for experimentally inducing muscle soreness in human subjects. Nearly all of the studies found by the investigator have involved a design in which subjects performed an exercise consisting of a high number of repetitions, usually to the

point of total fatigue. However, the exercise used for this study was patterned after the design used by Bobbert, Hollander, and Huijing (1986). After two pilot studies by the researcher, it was established that this exercise was successful in causing mild soreness in the gastrocnemius. The nature of this type of exercise also permitted several subjects to perform the exercise simultaneously, and it was of low enough intensity to enable the subjects to complete the exercise without a high risk of serious injury.

The delayed muscle soreness was induced by having the subject repeatedly lift and lower the body by plantar- and dorsiflexion of the ankle on the left leg. A 2.0-cm board was placed under the ball of the foot. The subjects were instructed to keep the knee of the exercising leg extended and the non-experimental leg free from bearing any weight while performing the exercise (Appendix C). They were also asked to plantarflex as much as possible, then return the heel to the floor in a controlled manner. The exercise lasted 15 minutes, using intervals of 15 seconds of exercise followed by 15 seconds of rest. During the rest period the subject was allowed to step off the board and stand on both feet. The subjects performed at least 8 and no more than 12 plantar-/dorsiflexion repetitions per 15 seconds. To assist in establishing the proper frequency, a verbal cadance allowing completion of 10 repetitions was given for the first two exercise intervals. Throughout the



subsequent exercise bouts, the subjects were responsible for completing the proper number of repetitions.

### Measurement Procedures

Range of motion and perception of soreness measurements were obtained for the left leg of each subject preexercise, and at 24 and 48 hours postexercise. The 24-hour measurements were taken prior to administering any treatment that session. The measurements of range of motion were taken using a modified version of a goniometer designed by the researcher. A drawing of the instrument is shown in Appendix D. The soreness perception was acquired by using a 10-cm long graded analogue scale (Appendix E). Each subject used his/her own subjective judgement, free from any interaction with any other individual, while grading the soreness.

### Reliability and Validity Tests

The range of motion measuring instrument was tested for reliability using a test-retest on 20 subjects. A Product-moment correlation was used on the paired scores which were taken approximately one hour apart. An  $r=.958$  was found. The raw data from this test are shown in Appendix F.

The construct validity was evaluated by demonstrating

the instrument to four individuals for their approval. These included two physical educators with Ph. D.'s, a certified athletic trainer, and a professor outside the field of physical education. Studies by Rivera (1979) and Leighton (1942) found reliability scores on ankle range of motion using the Leighton Flexometer to be .927 and .99 respectively. The measuring device used for this study proved to be as reliable as the flexometer, which is a popular instrument used in research for measuring joint range of motion. The preexercise mean for total range of motion for the 60 subjects involved in this study was 66.0 degrees. The American Academy of Orthopaedic Surgeons (1985) compiled data from four sources and found the average range of motion for the ankle to be 66 degrees.

#### Range of Motion Measurement

The instrument used to measure ankle range of motion consisted of a plastic goniometer attached to a 2' x 2' sheet of plexiglas in such a way that the only movement that could occur to the protractor and horizontal arm of the goniometer was a vertical adjustment. This adjustment was necessary in order to align the axis of the goniometer with the center of the lateral malleolus of each subject. As soon as the proper position was set, the protractor and horizontal arm were locked into place. The sliding mechanism was produced by gluing a two inch piece of

plexiglas at a right angle to the fixed horizontal arm of the goniometer. This vertical piece was fit between two pieces of plexiglas which were glued to the large sheet of plexiglas. This formed a track which allowed the entire goniometer to move vertically. The 2' x 2' sheet of plexiglas was attached to a wooden table with two screws.

Each subject was marked by placing a dot, with indelible ink, on the center of the lateral malleolus and on the posterior aspect of the fifth metatarsal on the left foot (Appendix G). The subject was seated on the table with the leg fully extended and the heel approximately three inches past the edge of the table, or until the dot on the malleolus was aligned with the axis of the goniometer. The malleolus was in slight contact with the large sheet of plexiglas on the opposite side of the fixed goniometer. The subject was instructed to keep the leg straight and dorsiflex as far as possible. The adjustable arm of the goniometer was then aligned with the dot on the fifth metatarsal and a measurement was taken (Appendix H). The subject repeated the procedure by plantarflexing, taking care to keep the leg straight and the achilles tendon in contact with the edge of the table. Any errors during the measurement performed by the subjects were verbally corrected by the researcher during the first and second trials. Three trials were given and the third one was recorded. Fieldman (1968) and Harvey and Scott (1967)

found that reliability of flexibility scores was higher on the third or fourth trials. Both measurements, extreme plantarflexion and dorsiflexion, were recorded in degrees using the full 0-180 degree scale on the protractor. The difference between the two measurements was used as data for total range of motion (Appendix I). The raw data of the range of motion and the degree of loss or gain at 24 and 48 hours from preexercise are shown in Appendix J.

#### Soreness Perception Measurement

For the determination of soreness perception, the subjects were asked to score on a graded analogue scale the amount of soreness perceived in the gastrocnemius. This scale was 10 cm in length, with two divisions equally spaced along the line for use as reference marks by the subject. The subject was instructed to place a vertical line through the scale at the place that best indicated the amount of soreness experienced in the calf muscle at that time. For the purpose of transforming the subject's marks into workable data, the researcher measured the marks to the nearest millimeter, starting from the left end of the scale. Thus, a score from 0 to 99 was possible.

#### Treatment Procedures

Group I served as a control and received no

postexercise treatment throughout the study. After completing the exercise to induce delayed muscle soreness, they returned at 24 and 48 hours postexercise for range of motion measurements and to chart their soreness perception.

Group II performed a stretching program immediately following exercise and at 3 and 24 hours postexercise. The stretching program consisted of a static stretch of the gastrocnemius (Appendix K). The subjects stood approximately four feet away from a wall, placed the hands against the wall while keeping the left leg straight and the heel in contact with the ground. This gently but forcibly placed the calf muscles of the left leg on stretch. They stretched up to a point of slight discomfort and held that position for 20 seconds, followed by a 20 second rest. They repeated this procedure three times, took a 1-minute rest and repeated the procedure for a total of six stretches.

Group III followed the same guidelines as group II for the stretching program but added a cold water treatment prior to stretching. The left leg was submerged up to a point slightly below the patella. The water was held at a constant temperature of 57 degrees F. After 15 minutes of cold treatment, the subjects quickly removed their leg, dried it with a towel and performed the stretching routine.

### Analysis of Data

The range of motion and soreness perception data were compiled and computed for statistical comparisons of the group means. A fixed-effects model for a two-factor analysis of variance was computed on an Apple IIe computer for each of the two groups of data. A level of significance of  $p < 0.1$  was selected as the level of acceptance. This level was chosen with the idea of uncovering a general difference between the postexercise treatments of stretching and cold with stretching. If one of these treatments proved to be superior in preventing or providing quicker recovery of range of motion or perception of soreness at this level, then further studies might use a lower level of significance to compare the effectiveness of specific levels of treatments (Minium, 1978). That is, future studies could compare various degrees and durations of stretching and cold water immersion.

Although the independent variables of stretching and cold with stretching were under investigation for their differential effects on range of motion and perception of soreness, the control group was an important part of the statistical design. The control group was used in order to assure that soreness did occur from the exercise and that any observed difference in recovery rate of the stretching and/or cold with stretching groups was not the same as the

control group (ie. no postexercise treatment). It simply assessed the effect of the natural healing process of the body without experimentally manipulating that process as designed in the stretching and cold with stretching groups. Although rest is considered a treatment for injuries, the control group in this study cannot be considered as a postexercise treatment since it was not controlled rest. That is, there were no levels or criteria established for the control group. The control group was used in the ANOVA design as a means of evaluating any differences between the two independent variables, stretching and cold with stretching. The difference in effect in the control group was subtracted from the stretching and cold with stretching groups. Any differences remaining were considered to be the effect that particular treatment had on the dependent variables, range of motion and soreness perception.

Although the research questions were formulated to find any significant differences between the stretching protocol and the cold with stretching protocol, the statistical hypothesis must include all three groups.

The hypotheses for the effects on range of motion are:

$$H_A : \mu_1 = \mu_2 = \mu_3$$

$$H_0 : \mu_1 = \mu_2 = \mu_3$$

The hypotheses for the effects on perception of soreness are:

$$H_A : \mu_1 = \mu_2 = \mu_3$$

$$H_0 : \mu_1 = \mu_2 = \mu_3$$

These statements indicate that in order to reject the null hypothesis, a significant difference must be found between the stretching group, the cold with stretching group and the control group. It is possible for the control group to be equal to one of the other groups and still find a significant difference between the stretching and cold with stretching group. It is also possible for the stretching and cold with stretching group not to be significantly different from each other while being significantly different than the control group. They could both be equally effective in preventing or providing quicker relief from DMS without one being more effective than the other. If this happens, then the research question that there is a significant difference between stretching alone and stretching after cold water immersion on the rate and extent of recovery from the dependent variables (each tested independently) will not be accepted.

The fixed-effects model ANOVA is necessary since the levels of the independent variables used in this experiment were arbitrarily chosen by the researcher. The results of the study can be generalized only to populations of subjects who would be clinically treated on the same specifically chosen levels (Linton & Gallo, 1975). It cannot be assumed that different water temperatures, treatment times, other stretching exercises, etc., would have the same effect on the dependent variables as in this



experiment.

A repeated measures design allows both independent variables, the stretching protocol and the cold with stretching protocol, plus the control group (no postexercise treatment) to be combined in the same study so that the effects of time across subjects can be evaluated. The repeated measures determined the rate at which each treatment affected the dependent variables by examining the differences each group has from 24 to 48 hours. Table 2 shows the design of the ANOVA used. In this design the two

Table 2  
Statistical Design  
A x (B x S) ANOVA

		Factor B	
		Time	
		24 hour	48 hour
Factor A	Control	S <sub>1</sub> . . . S <sub>20</sub>	
	Stretch	S <sub>21</sub> . . . S <sub>40</sub>	
	Cold & Stretch	S <sub>41</sub> . . . S <sub>60</sub>	

factors are: the different postexercise treatments, or the between-groups factor (A); and the differential postexercise treatment effect at 24 and 48 hours, the within groups factor (B). The variance of postexercise treatment effects within each group was evaluated to see if the groups came from similar or different populations. Any interaction between groups was found by determining if there were any group mean differences across time. Any interaction that does occur can be found by plotting the mean scores of each of the three groups at 24 and 48 hours on a graph. An interaction is shown if the lines of two groups intersect.

#### Assumptions and Limitations

It was assumed that the subjects were representative of the normal population distribution of college students between the ages of 18 and 25 and that a maximal effort was given by each subject during the exercise and all testing and measurement procedures. It was further assumed that subjects did not apply any form of treatment to the experimental leg during the course of the study. It was assumed that subjects were free from any injury or muscular limitation of the ankle and lower leg that might cause invalid data. Those with known injuries or muscular limitations were omitted from the study.

Limitations observed by the researcher were that some physical activity may have occurred during the testing period outside of the study due to activity classes and intramural programs. All subjects were not tested on the same day or time of day and all subjects had slightly different time periods between treatment and measurements. Because of the large sample size used in this study, subjects with common levels of fitness, such as all track athletes etc., could not be used. Although the exercise was designed to allow individuals to choose a frequency and amplitude which they could endure, the wide variation of fitness levels may have caused more soreness in some than in others.

## Chapter 4

### ANALYSIS OF DATA

#### Introduction

This chapter contains the results of the analysis of data from the comparison of the control group (no postexercise treatment) and the postexercise treatment groups, stretching and cold with stretching. The three groups' means were compared at 24 hours and at 48 hours for differences in ankle range of motion and perception of soreness. The statistical procedure used was a fixed-effects model for a two-factor analysis of variance. The level of significance selected for this study was  $p < 0.1$ . This chapter is divided into two major topic areas: (1) range of motion findings, and (2) soreness perception findings.

#### Range of Motion Findings

The raw data results of the range of motion measurements are presented in Appendix J. Table 3 reveals the group means and standard deviation scores for each of the three groups at 24 and 48 hours. All three groups showed a decrease in range of motion at 24 hours after

Table 3

MEAN AND STANDARD DEVIATION RESULTS  
FOR RANGE OF MOTION AS EXPRESSED  
IN DEGREES LOSS

	GROUPS TESTED					
	Control		Stretch		Cold & Stretch	
	24h	48h	24h	48h	24h	48h
Mean	-2.45	- .45	-3.05	-2.20	-3.90	-3.80
SD	7.51	8.29	7.20	10.54	7.72	11.16

exercise, indicating that the exercise did cause a negative effect on range of motion, a known symptom of delayed muscle soreness. The cold and stretch group showed the greatest loss in range of motion with a -3.90 degree loss, followed by the stretching group with a -3.05 degree loss. The control group recorded the smallest loss in range of motion at 24 hours with a mean score of -2.45 degrees. The difference between the three groups' range of motion scores at 24 hours was not statistically significant.

At 48 hours, all three groups indicated that they were recovering from the exercise by showing an increase in

range of motion from their 24-hour measurements. However, all groups were still below preexercise levels. The control group recorded the greatest rate of recovery from the exercise with a 24-hour score that showed an increase in range of motion of two degrees. The control group also recorded the greatest extent of recovery from the exercise by returning to within 0.45 degrees of the preexercise mean score. Figure 1 presents the mean difference from preexercise values of each group at 24 and 48 hours. As indicated by Figure 1, the stretching and cold with stretching groups had higher mean scores at 24 hours than the control group and maintained a higher level at 48 hours. Also, the rate of recovery for the stretching and cold with stretching groups was less than the control group. Although none of the differences were found to be statistically significant, there is an indication that the forms of postexercise treatment administered in this study may have had a negative impact on the recovery of range of motion following the onset of delayed muscle soreness. As shown by the group means at 24 and 48 hours in Figure 1, as the amount of postexercise treatment time increased, the rate and extent of recovery from the exercise decreased.

The ANOVA Summary Table for Range of Motion (Table 4) shows the F value of 1.312 proved insignificant for the within subjects comparison across time. The results of this study failed to reject the hypothesis that  $\mu_1 = \mu_2 = \mu_3$  for range of motion. Therefore, this study failed to

Figure 1

GROUP MEAN DIFFERENCE FROM PREEXERCISE  
FOR RANGE OF MOTION

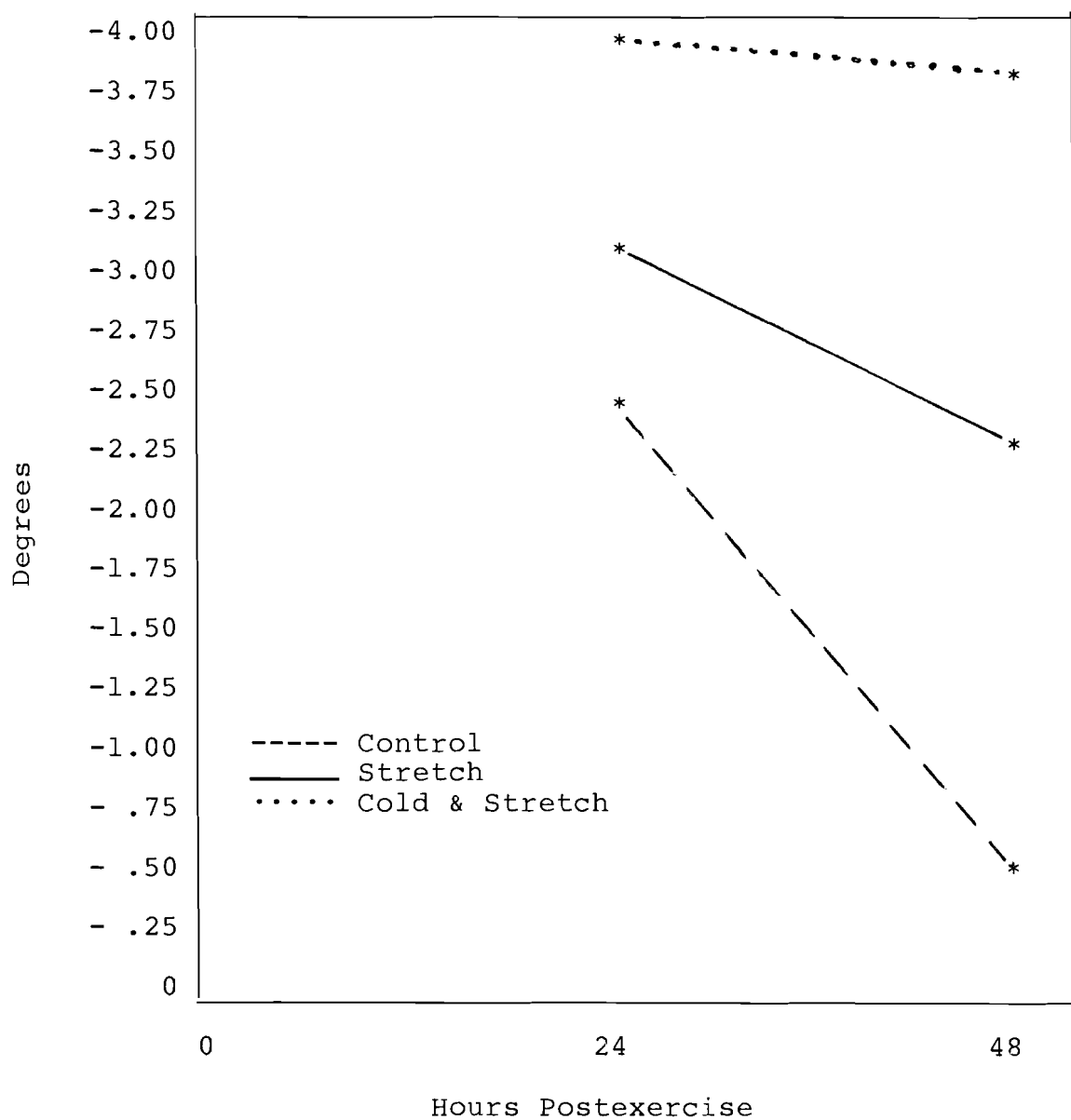


Table 4

## ANOVA SUMMARY TABLE FOR RANGE OF MOTION

Source	SS	df	MS	F
<u>Between Subjects</u>				
Treatments	115.217	2	57.608	.426
Error	7712.875	57	135.314	
<u>Within Subjects</u>				
Time	29.008	1	29.008	1.312
Treatment Time	18.317	2	9.158	.414
Error	1260.175	57	22.108	
Total	9135.592	119		



accept the research question that there is a significant difference between stretching alone and stretching after cold water immersion on the rate and extent of recovery of range of motion following exercise that causes delayed muscle soreness.

### Soreness Perception Findings

The raw data results for the perception of soreness measurements are presented in Appendix L. All subjects reported no soreness for the preexercise measurement, therefore the reported scores at 24 and 48 hours and the differences of preexercise from the 24 and 48 hours are the same. Table 5 reveals the group means and standard deviation scores for each of the three groups at 24 and 48 hours. Muscle soreness was apparent in all three groups at 24 hours. This gives evidence that the exercise was effective in producing some degree of delayed muscle soreness. However, this also proves that, within the stated research design, neither the stretching treatment nor the cold with stretching treatment was effective in preventing delayed muscle soreness.

The F value of 2.777 found in the ANOVA Summary Table (Table 5) indicated that an interaction did occur within groups across time. That is, at least one of the group means was different from the others between the 24 and 48 hour recordings. However, it did not indicate

Table 5

MEAN AND STANDARD DEVIATION RESULTS  
FOR PERCEPTION OF SORENESS

	GROUPS TESTED					
	Control		Stretch		Cold & Stretch	
	24h	48h	24h	48h	24h	48h
Mean	40.80	33.25	44.65	43.15	36.95	41.35
SD	23.62	25.90	26.45	30.71	36.95	34.12

specifically which mean was different. Figure 2 shows that the interaction occurred between the control group and the cold and stretching group from the 24 hour to the 48 hour measurements. The cold with stretching group mean was lower than the control group and the stretching group at 24 hours although not statistically significant. At 48 hours the cold with stretching group mean showed a significant increase in soreness. This indicates that the application of cold (in the cold with stretching group) may have caused the interaction to occur since the stretching group was not involved in any significant difference or interaction with the control group or the cold with stretching group. The only difference between the cold with stretching group and

Table 6

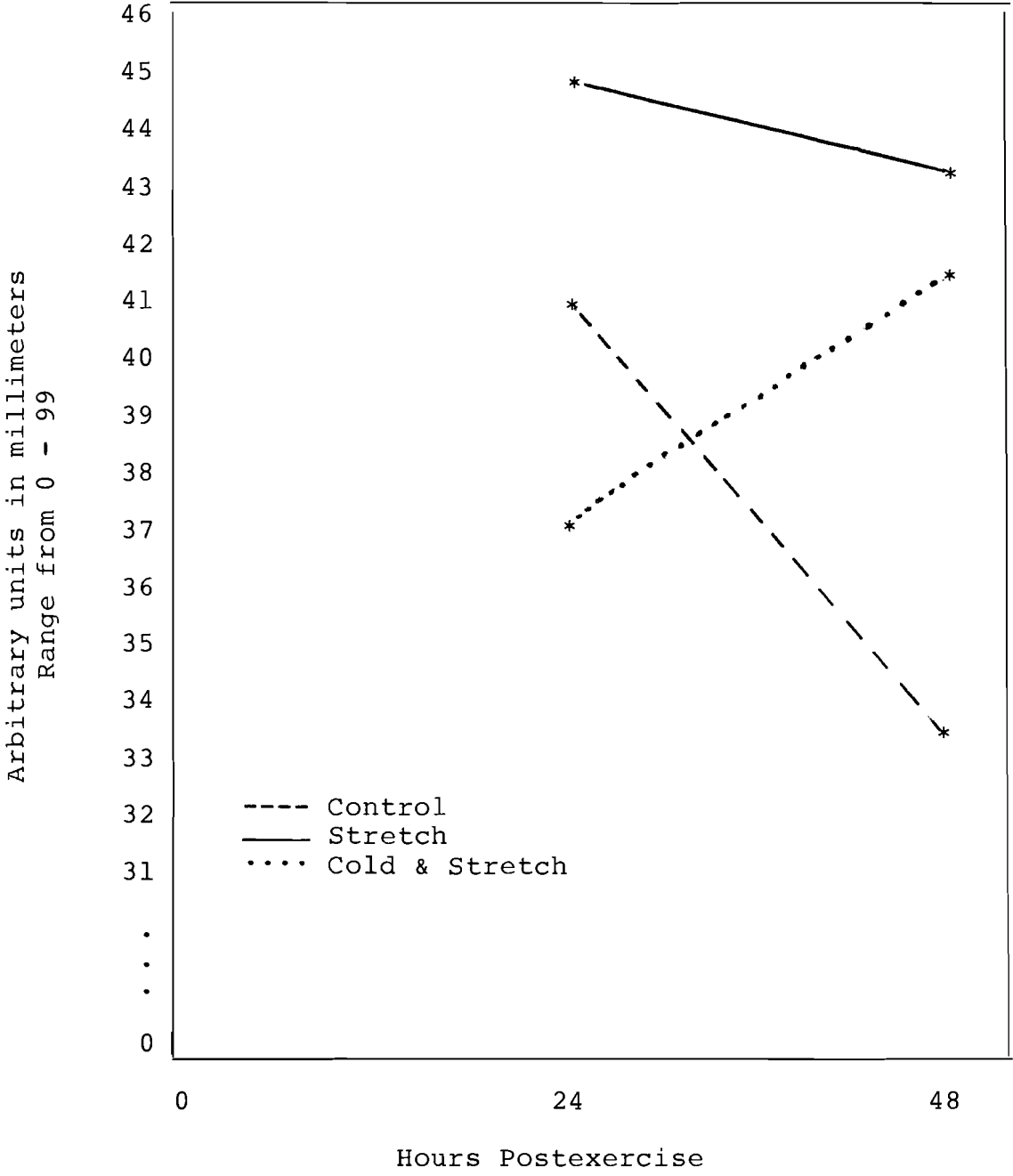
## ANOVA SUMMARY TABLE FOR PERCEIVED SORENESS

Source	SS	df	MS	F
<u>Between Subjects</u>				
Treatments	991.250	2	495.625	.335
Error	84334.175	57	1479.547	
<u>Within Subjects</u>				
Time	72.075	1	72.075	.561
Treatment Time	714.050	2	357.025	2.777*
Error	7329.375	57	128.586	
Total	93440.925	119		

\*significant interaction between treatments across time  
at  $p > 0.1$ .

Figure 2

GROUP MEAN PAIN PERCEPTION CHANGES



the stretching group was the application of the cold prior to the stretch.

### Summary

The group mean differences from preexercise scores of range of motion and soreness perception found that all three groups decreased in range of motion of the ankle and recorded some soreness at 24 hours after exercise. The stretching group recorded the highest level of soreness at 24 hours, decreased slightly but still showed the highest level of soreness at 48 hours. An interaction did occur between the control group and cold and stretching group from 24 to 48 hours. While the control group recorded a higher score than the cold and stretching group for soreness at 24 hours and decreased at 48 hours, the cold and stretching group actually increased in soreness from 24 to 48 hours to a level above the control group and just below the stretching group. No interaction occurred between any groups for the range of motion measurements. The cold and stretching group showed the greatest loss in range of motion, and the control group had the least amount of loss at 24 hours. All groups showed signs of recovery from soreness with an increase in range of motion at 48 hours, with the control group having the greatest amount of recovery. All three groups' 48 hour measurements were still below the preexercise mean at 48 hours, with the

control group closest to the preexercise level.

## Chapter 5

### SUMMARY, DISCUSSION, CONCLUSIONS, RECOMMENDATIONS, & SUGGESTIONS

#### SUMMARY

The incidence of delayed muscle soreness following exercise that is more vigorous than usual or involving repetitive eccentric contractions continues to pose a problem in the conduct of training and conditioning programs. At this time, the theoretical basis and the underlying mechanisms for its occurrence are not firmly established. Current research has generally agreed that some form of structural damage occurs. The controversy exists in identifying the causative factors of the structural damage. Another problem in dealing with this phenomenon of delayed muscle soreness is the difficulty encountered in finding a precise measurement of the degrees of soreness. Several investigators have observed muscular activity using EMG recordings and have found that an increase in activity in muscles correlated highly with soreness apparent in those muscles. However, subsequent investigators have failed to find a correlation in similar studies. The most consistent method of measuring soreness, although subjective, is allowing the subject to rate his/her soreness on some type of scale.

The physiological responses to a cold treatment have been considered effective in decreasing inflammation, swelling, edema, and muscle spasms. Stretching has also been found to reduce muscle spasms which aids in returning the muscle to its normal resting length. Since symptoms such as muscle spasm, edema, and inflammation have been associated with delayed muscle soreness, cold and stretching treatments should be an ideal treatment choice for delayed muscle soreness.

This study was completed to determine if a significant difference could be found between the stretching group and cold and stretching group, in terms of their ability to prevent or relieve symptoms of delayed muscle soreness in the calf muscle, caused by vigorous repetitive exercise. Scores on range of motion of the ankle and on the subject's perception of soreness were collected and analyzed as previously described in Chapters 3 and 4.

Sixty male and female subjects ranging in ages from 18 - 25 who were enrolled in lifetime fitness classes participated in the study. The exercise used to induce soreness in the calf muscles of the left leg was 30 sets of a plantar/dorsiflexion movement with a 2.0-cm board placed under the forefoot. Each set consisted of 15 seconds of exercise followed by 15 seconds of rest. The subjects were divided into three treatment groups: no treatment (control group), stretching, and cold with stretching. Six



30-second static stretches were used for the stretching group, while the cold and stretching group immersed the exercised leg in water at 57 degrees F. for 15 minutes prior to performing the same stretching program. All treatments were administered immediately following exercise and at 3 and 24 hours postexercise. A fixed-effects model for a two-factor analysis of variance was used to analyze the data with a  $p < 0.1$  level of significance.

### DISCUSSION

The null hypotheses tested in this study were: there is no significant difference between stretching alone and stretching after cold water immersion on the rate and extent of recovery of range of motion following exercise that causes delayed muscle soreness and, there is no significant difference between stretching alone and stretching after cold water immersion on the rate and extent of perception of soreness following exercise that causes delayed muscle soreness. The results of this study failed to reject either hypothesis.

In light of the evidence presented in some of the studies reviewed, these results were somewhat unexpected. DeVries (1961 & 1966) found relief from soreness using static stretching. Prentice (1982) found relief from soreness using combinations of cold and heat with static

and PNF stretches. Cold with static stretch was more effective than the other combinations although the other combinations were also effective. DeVries (1961) used subjects' perception of soreness as a measurement, but the other studies used EMG recordings as a measuring device for determining the level of soreness. In this study not only was there a lack of statistical significance in the stretching and cold with stretching treatments in decreasing soreness and increasing range of motion, the means of those groups showed an increase in soreness and a decrease in range of motion at 48 hours compared to the control group which received no postexercise treatment.

It should be noted that the results of this study can not be generalized beyond the levels of the independent variables chosen for this study. That is, other treatment times and application procedures of cold therapy and stretching techniques cannot be assumed to yield the same results as in this study. However, it would be reasonable to assume that similar stretching protocols would have a tendency to produce similar effects as a treatment for delayed muscle soreness. DeVries (1986), advocates the use of a two-minute static stretch two or three times a day for relief of soreness after it has occurred. His basis of using a stretching treatment is to break the pain-spasm-pain cycle which he believes is the initial cause of delayed muscle soreness. The static stretch in

this study was similar in that it was six 30-second stretches totaling three minutes. Therefore, the results of this study fail to add support to the muscle spasm theory as the cause of delayed muscle soreness.

Hayden (1964), Grant (1964), and Knight (1978) have supported the theory that a cold treatment with exercise is beneficial in rehabilitating a soft tissue injury. They also state that a cold treatment before exercise merely numbs the area and the exercise is responsible for the speed of recovery. The interaction between the cold and stretching treatment group and the control group in this study gave some indication that cold does reduce or inhibit the perception of pain to some degree. The conclusion made from this study is that some subjects who received the cold treatment psychologically thought it should help. When the subjects reported their 24-hour perception of soreness, they had received two cold and stretching treatments prior to that time. There was approximately 20 hours between the second treatment and when they charted their 24-hour marks. When they charted their soreness at 48 hours, there was approximately 23 hours between the last treatment and their 48 hour recording. A possible physiological explanation for the increased soreness at the 48-hour measurement is that the three cold treatments, in the cold with stretch group, within the first 24 hours decreased metabolism, upset the pain-spasm-pain cycle, and/or prevented edema

formation. The cold is thought to be the influencing factor since the stretching group failed to exhibit a similar pattern. When the cold with stretching treatments ended, the edema continued to increase, and/or the spasms slowly returned by the 48-hour measurement period. Thus, the cold may have simply caused further delay of the onset of the symptoms.

The large standard deviations in the soreness perception scores could be attributed to several factors not controlled in this study. Subject response to the treatments could have been influenced by their physical condition, pain threshold, body composition (affecting the penetration of the cold), ingestion of drugs, extra physical activity between the measurement periods outside the design of the study, etc.

The only study found measuring the effects of delayed muscle soreness on range of motion concluded that an increase in soreness perception was associated with a decrease in range of motion (Yackzan, Adams, Francis, 1984). The results of this study did not support that association.

### CONCLUSIONS

From the results of the statistical analyses and within the limitations of this study, the following

conclusions were made:

- (1) Muscular soreness can be induced without using exercises which force subjects to exercise to exhaustion or total fatigue.
- (2) An exercise routine of six 20-second static stretches with a 20-second rest interval was not effective in preventing or relieving the perception of soreness found in delayed muscle soreness.
- (3) An exercise routine of six 20-second static stretches with a 20-second rest interval was not effective in enhancing the recovery of range of motion found in delayed muscle soreness.
- (4) A 15 minute cold water treatment followed by an exercise routine of six 20-second static stretches with a 20-second rest interval was not effective in preventing or relieving the perception of soreness found in delayed muscle soreness.
- (5) A 15 minute cold water treatment followed by an exercise routine of six 20-second static stretches with a 20-second rest interval was not effective in enhancing the recovery of range of motion found in delayed muscle soreness.

#### RECOMMENDATIONS

The following questions and ideas are posed for

further investigation. Would a different type of stretching program be more effective in reducing symptoms of delayed muscle soreness? Would a colder water temperature or treatment time influence the outcome of the scores? Would the use of athletes from a common sport reduce the variance in scores because of similarities in body composition, metabolism rate, etc.? How much of an effect does subcutaneous fat thickness have on the outcome of the symptoms of pain? Would compression and/or elevation be effective in treating DMS? What would be the outcome of a comparison of two different intensities of exercise used to induce soreness?

### SUGGESTIONS

Based on the results of this study neither the cold and stretching nor the stretching treatment was found to prevent or obtain quicker relief from delayed muscle soreness. No treatment may be more effective than either the stretching or cold with stretching program used in this study. However, other stretching or cold applications with stretching may produce different effects.

## REFERENCES

## REFERENCES

- Abraham, W. M. (1977). Factors in delayed muscle soreness. Medicine and Science in Sports and Exercise, 9(1), 11-20.
- Abraham, W. M. (1979). Exercise induced muscle soreness. The Physician and Sportsmedicine, 7(10), 57-60.
- American Academy of Orthopaedic Surgeons, (1985). The foot and ankle: biomechanical evaluation and treatment. In J. A. Gould III & G. J. Davies (Eds.), Vol. 2. Orthopaedic and Sports Physical Therapy. St. Louis: C. V. Mosby Company.
- Armstrong, A. B. (1984). Mechanisms of exercise-induced delayed onset muscular soreness: a brief review. Medicine and Science in Sports and Exercise, 16(6), 529-538.
- Asmussen, E. (1956). Observations on experimental muscular soreness. Acta Rheumatologica Scandinavica, 2, 109-116.
- Bobbert, A., Hollander, P., & Huijing, P. (1986). Factors in delayed onset muscular soreness of man. Medicine and Science in Sports and Exercise, 18(1), 75-81.
- Bond, M. R. (1979). Pain: Its Nature, Analysis and Treatment. (pp. 26-30). New York: Churchill Livingstone.
- Brendstrup, P. (1962, August). Late edema after exercise. Archives of Physical Medicine & Rehabilitation, 401-405.
- Clarke, D. (1963, October). Effect of immersion in hot and cold water upon recovery of muscular strength following isometric exercise. Archives of Physical Medicine & Rehabilitation, 565-568.
- Cobb, C. R., deVries, H. A., Urban, R. T., Leukens, C. A., & Bagg, R. J. (1975). Electrical activity in muscle pain. American Journal of Physical Medicine, 54, 80-87.
- deVries, H. A. (1961). Prevention of muscular distress after exercise. Research Quarterly, 32, 177-184.



- deVries, H. A. (1966). Quantitative electromyographic investigation of the spasm theory of muscle pain. American Journal of Physical Medicine, 45, 119-134.
- deVries, H. A. (1986). Physiology of muscle soreness - cause and relief (pp. 473-488). In: Physiology of Exercise for Physical Education and Athletics (4th ed.). Dubuque, Iowa: Wm. C. Brown Publishers.
- Francis, K. T. (1983). Delayed muscle soreness: A review. The Journal of Orthopaedic and Sports Physical Therapy, 5(1), 10-13.
- Friden, J. (1984). Muscle soreness after exercise: Implications of morphological changes. International Journal of Sports Medicine, 5, 57-66.
- Friden, J., Sjoström, M., & Ekblom, B. (1983). Myofibrillar damage following intense eccentric exercise in man. International Journal of Sports Medicine, 4, 170-176.
- Fieldman, H., (1961, October). Relative contribution of the back and hamstring muscles in the performance of the toe-touch test after selected extensibility exercises. Research Quarterly, 39, 518-523.
- Grant, A. E. (1964, May). Massage with ice (Cryokinetics) in the treatment of painful conditions of the musculoskeletal system. Archives of Physical Medicine & Rehabilitation, 233-238.
- Harvey, V. P. & Scott, G. D., (1967, March). Reliability of measure of forward flexibility and its relation to physical dimensions of college women. Research Quarterly, 38, 28-33.
- Hayden, D. (1964). Cryokinetics in an early treatment program. Journal of American Physical Therapy Association, 44, 990-993.
- Hough, T. (1902). Ergographic studies in muscular soreness. American Journal of Physiology, 7, 76-92.
- Houghlum, P. (1977). The modality of therapeutic exercise: Objectives and principles. Athletic Training, 12(1), 42-45.

- Huskisson, C. E. (1974, November 9). Measurement of pain. The Lancet, 1127-1131.
- Joyce, C.R.B., Zutshi, D. W., Hrubes, V., & Mason, R. M. (1975). Comparison of fixed interval and visual analogue scales for rating cronic pain. European Journal of Clinical Pharmacology, 8, 415-420.
- Knight, K. L. (1978). Cryotherapy in sports medicine. In K. Scriber, & E. Burke (Eds.), Relevant Topics in Athletic Training (pp. 52-59). New York: Movement Publications.
- Kowal, M. A. (1983). Review of physiologiagl effects of cryotherapy. The Journal of Orthopaedic and Sports Physical Therapy, 5(2), 66-73.
- Lehmann, J. F., & de Lateur, B. J. (1982). Cryotherapy. In J. Lehmann (Ed.), Therapeutic Heat and Cold 3rd Ed. (pp. 563-602). Baltimore: Williams & Wilkins.
- Leighton, J. R. (1955, September). An instrument and technique for the measurement of range of motion. Archives of Physical Medicine and Rehabilitation, 571-578.
- Levine, J. D., Gordon, N. C., Jones, R. T., & Fields, H. L. (1978). The narcotic antagonist naloxone enhances clinical pain. Nature, 272, 826-827.
- Linton, M. & Gallo, P. (1975). The Practical Statistician: Simplified Handbook of Statistics. Belmont California: Wadsworth Publishing Co. Inc.
- McGlynn, G. H., Laughlin, N. T., & Filios, S. P. (1979). The effect of electromyographic feedback on EMG activity and pain in the quadriceps muscle group. Journal of Sports Medicine, 19, 237-244.
- McMaster, W. C. (1982). Cryotherapy. The Physician and Sportsmedicine, 10(11), 112-119.
- Millar, A. P., (1976). An early stretching routine for calf muscle strains. Medicine and Science in Sports, 8(1), 39-43.
- Minium, E., (1978). Statistical Reasoning in Psychology and Education (2nd ed.). New York: John Wiley & Sons.
- Moller, M., Ekstrand, J., Oberg, B., & Gillquist, J. (1985, March). Duration of stretching effect on range of motion in lower extremities. Archives of Physical Medicine & Rehabilitation, 171-173.

- Newham, D. J., Mills, K. R., Quigley, B. M., & Edwards, R. H. T. (1983). Pain and fatigue after concentric and eccentric muscle contractions. Clinical Science, 64, 55-62.
- Petajan, J. H., & Eagan, C. J. (1968). Effect of temperature and physical fitness on the triceps surae reflex. Journal of Applied Physiology, 25, 16-20.
- Prentice, W. E. (1982). An electromyographic analysis of the effectiveness of heat or cold and stretching for inducing relaxation in injured muscle. The Journal of Orthopaedic and Sports Physical Therapy, 3(3), 133-140.
- Rivera, M. L. (1979). Effects of static, ballistic and modified proprioceptive neuromuscular facilitation stretching exercises on the flexibility and retention of flexibility in selected joints. Unpublished master's thesis, University of Kansas, Lawrence, KS.
- Talag, T. (1973). Residual muscular soreness as influenced by concentric, eccentric, and static contractions. Research Quarterly, 44, 458-469.
- Tullson, P. & Armstrong, R. B. (1981). Muscle hexose monophosphate shunt activity following exercise. Experientia, 37, 1311-1312.
- Waltrous, B., Armstrong, R., & Schwane, J. (1981). The role of lactic acid in delayed onset muscular soreness. Medicine and Science in Sport and Exercise, 13(2), p. 80. (Abstract from the 1981 American College of Sports Medicine annual meeting).
- Yackzan, L., Adams, C., & Francis, K. (1984). The effects of ice massage on delayed muscle soreness. The American Journal of Sports Medicine, 12, 159-165.

APPENDIX A  
SUBJECT INFORMATION SHEET

## STUDENT INFORMATION SHEET

First	Last	Middle Initial
-------	------	----------------

SEX	
-----	--

HOME ADDRESS	
--------------	--

PHONE NUMBER	
--------------	--

Did you have any injury of your lower left leg that required treatment within the last 3 months? Yes ☐ No ☐

Were you involved in any varsity sport at Emporia State? Yes ☐ No ☐

Please indicate the group of sessions which you will be able to attend.

Tuesday	2:00-3:10		
	4:30-4:55	<input type="checkbox"/>	or for 25 min. between 4:15-5:30
	6:30-6:55	<input type="checkbox"/>	or for 25 min. between 6:30-7:30
Wed.	3:00-3:20		
Thur.	3:00-3:15		

Monday	2:00-3:10		
	4:30-4:55	<input type="checkbox"/>	or for 25 min. between 4:15-5:30
	6:30-6:55	<input type="checkbox"/>	or for 25 min. between 6:30-7:30
Tue.	3:00-3:20		
Wed.	3:00-3:15		

Tuesday	10:00-11:00		
	<input type="checkbox"/>		for 25 min. between 12:00 and 3:00
	<input type="checkbox"/>		for 25 min. between 1:30 and 6:30
			<b>NOTE</b> (There must be at least 1 hour between treatments.)
Wed.	10:00-1:00		for 30 minutes within that time.
Thur.	10:00-10:20		

None of these times work out. An alternate schedule needs to be arranged.

APPENDIX B  
INFORMED CONSENT FORM

## SUBJECT'S INFORMED CONSENT DOCUMENT

I, Dennis Weber, am requesting your voluntary participation in a study designed to compare the effectiveness of two types of treatment for the relief of symptoms associated with delayed muscle soreness. The specific objective is to determine if the use of a cold water treatment before stretching is more beneficial than stretching alone in the relief of discomfort and the recovery of flexibility after exercise that causes delayed muscle soreness.

The Division of Health, Physical Education, Recreation, and Athletics supports the practice of protection for human subjects participating in research and related activities. The following information is provided so that you understand the procedures of the study and therefore can make an informed decision whether you wish to participate in this study.

As a subject in this study you will be asked to volunteer enough time, at your convenience, for the completion of the activities involved within a 48-hour timeframe. There will be four different required sessions. The first session will last approximately one hour and fifteen minutes. Sessions two, and three will last thirty minutes. The final session will take approximately ten minutes to complete. You are being asked to submit to the procedures outlined below.

Session One: Only the left leg of each subject will be used as a part of this study. Prior to the exercise, a measurement of range of motion on the ankle of the experimental leg and charting of the amount of soreness perceived in the calf muscle will be done. The exercise lasts 15 minutes and will consist of performing toe raises on the experimental leg with a two inch board under the ball of the foot. The toe raises will be done at 15-second intervals followed by equal amounts of rest. This exercise routine is designed to cause some delayed muscle soreness, but not pain, in the exercised leg muscles. Group I will serve as a control and will not receive any type of treatment for the duration of the study, that is, Group I will only take part in sessions one, four, and five. Group II will perform six 20-second stretches for the calf muscles immediately following the exercise. Group III will follow the same stretching routine after immersing the lower leg in cold water for 15 minutes. Both treatments have been shown to relieve symptoms associated with delayed muscle soreness.

Session Two: Groups II and III will perform the same treatment procedure as performed in the first session (stretching for Group II and cold immersion plus stretching for Group III).

Session Three: All subjects will again undergo the range of motion test of the ankle and charting of the amount of soreness perceived in the calf muscle. Groups II and III will again receive the identical treatment as in the preceeding sessions.

Session Four: In this final session, all subjects will be tested for the range of motion of the ankle and charting of the amount of soreness perceived in the calf muscle.

In order to investigate the effects of treatment for delayed muscle soreness, some discomfort and mild soreness must be induced in the calf muscles. The procedures and treatments being used in this study have been safely performed in other studies.

Although all of the subjects will experience some muscular discomfort, the intensity and duration of the exercise have been designed to induce discomfort but not pain. Furthermore, if any subject experiences more discomfort than he/she cares to endure, the treatments will be initiated sooner or more frequently than scheduled in order to bring relief sooner.

As with any type of physical activity, there is the possibility of severe pain or injury. Some degree of tear, sprain, strain, or muscle cramps from the exercise is possible. Dizziness, nausea, and fatigue are other possible adverse effects. Also, the cold treatment used in this study might be uncomfortable for the first three to five minutes. The cold or pain associated with this treatment may be severe to some subjects. There is a risk of developing shortness of breath, nausea, or dizziness. At any time throughout the study, if any of these symptoms appear severe, the subject may drop out of the study as desired.

Your permission to use the data collected is requested for the purpose of conducting research for a thesis and for the use in any publications resulting from this study. All data will be recorded on coded recording forms and will be kept confidential. Data will be displayed and discussed in a manner which preserves the complete confidentiality of each subject. Only Dennis Weber, the primary investigator, will possess the list matching code numbers to the names of the participants.

"I have read the above statement and have been fully advised of the procedures to be used in this project. I have been given sufficient opportunity to ask any questions I had concerning the procedures and possible risks involved. I understand the potential risks involved and I assume them voluntarily. I likewise understand that I can withdraw from the study at anytime without being subjected to reproach.

---

Subject

---

Date



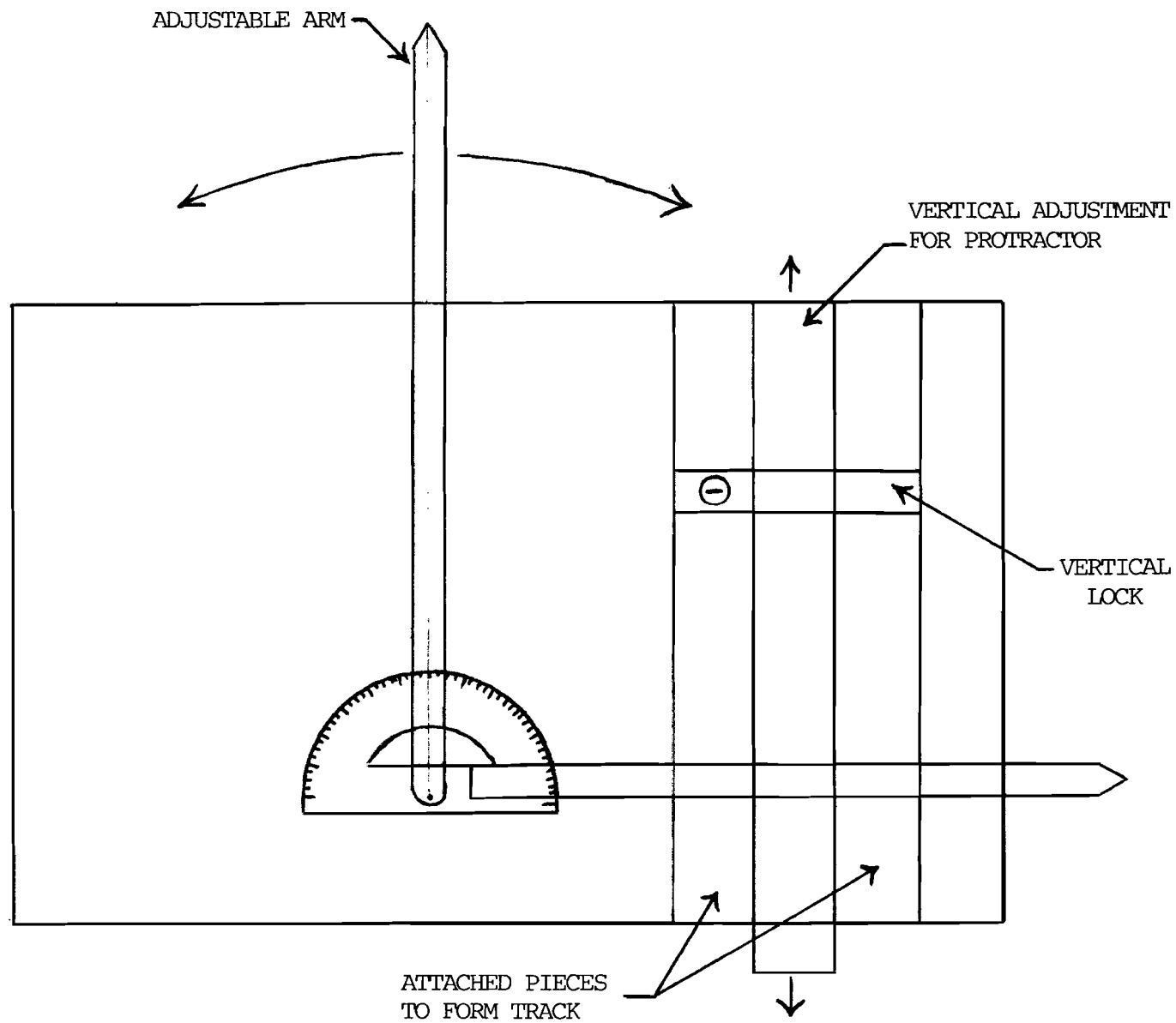
APPENDIX C  
PHOTOGRAPH OF EXERCISE



Exercise to Induce Soreness

## APPENDIX D

### SCHEMATIC OF RANGE OF MOTION INSTRUMENT



APPENDIX E

PERCEPTION OF SORENESS FORM

GROUP: Control (GI) Name \_\_\_\_\_

Stretch (GII) Code \_\_\_\_\_

Cold & Stretch (GIII) Date \_\_\_\_\_ Time \_\_\_\_\_


### INSTRUCTIONS

Please mark the amount of soreness felt in your calf muscle on the exercised leg by placing a ( | ) at the appropriate place on the line. Any soreness on the back of the leg from the knee to the ankle should be considered.

Use the definitions below as a reference in making your decision as to the degree of soreness.

- No soreness - No noticeable effects or symptoms.
- Mild - Soreness only apparent upon rubbing or pushing on the muscle.
- Moderate - Soreness apparent while walking or upon light stretching.
- Severe - Extreme pain felt and limits normal movement.

Pre-exercise



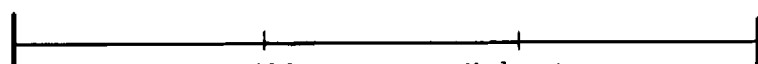
No Soreness      Mild Soreness      Moderate Soreness      Severe Soreness

24 Hours



No Soreness      Mild Soreness      Moderate Soreness      Severe Soreness

48 Hours



No Soreness      Mild Soreness      Moderate Soreness      Severe Soreness

## APPENDIX F

### RELIABILITY DATA FOR ROM INSTRUMENT

## Raw Scores:

## Reliability Test for Range of Motion Instrument

Subject	Test 1	Test 2
1	54	68
2	63	65
3	63	59
4	67	69
5	63	66
6	82	80
7	71	68
8	53	55
9	51	51
10	57	54
11	77	76
12	67	72
13	79	72
14	64	70
15	57	56
16	73	71
17	115	114
18	88	89
19	75	83
20	82	79

---


$$r_{xy} = .958$$

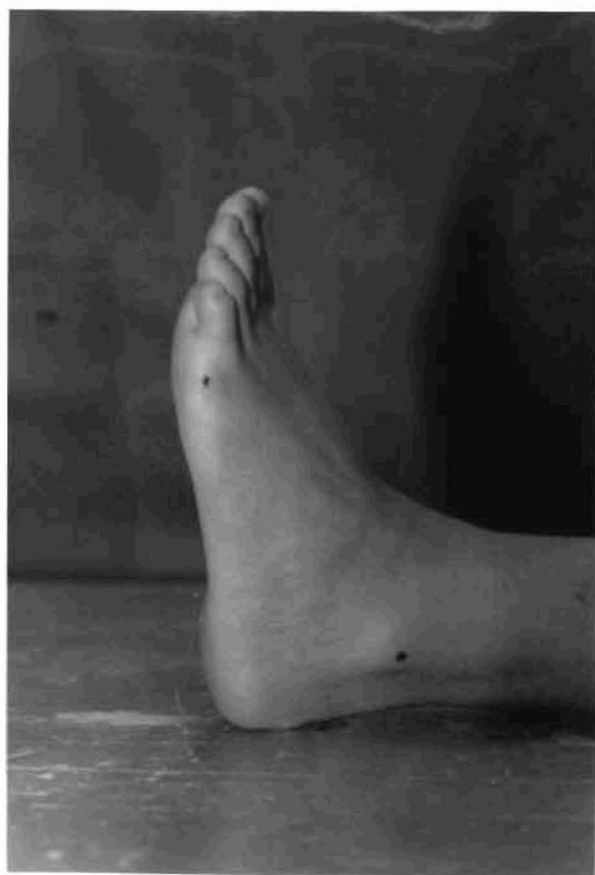
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Pearson r correlation was used.



APPENDIX G

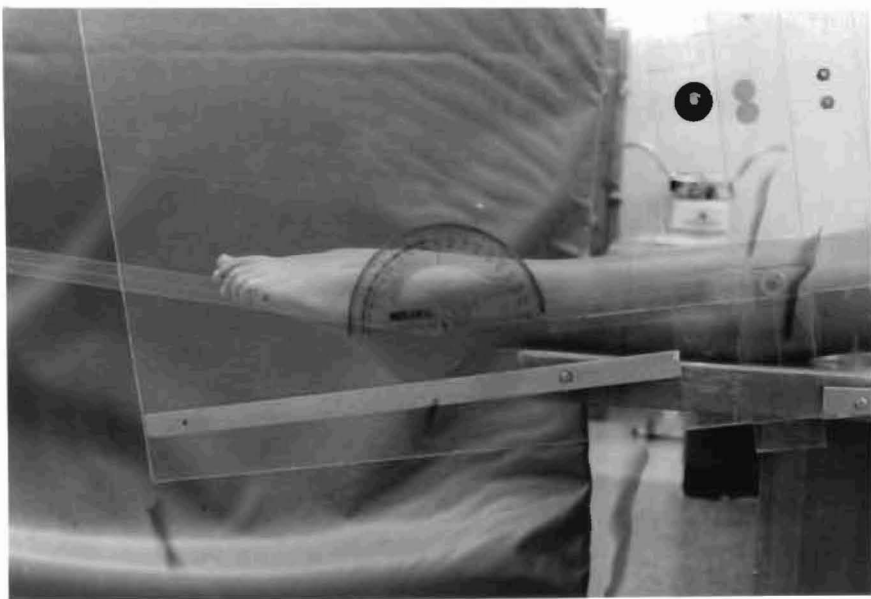
PHOTOGRAPH OF MARKINGS ON FOOT



Markings on Foot

APPENDIX H

PHOTOGRAPH OF USE OF ROM INSTRUMENT



Use of ROM Instrument

APPENDIX I

RANGE OF MOTION RECORDING FORM

## RANGE OF MOTION MEASUREMENTS

PREEX. \_\_\_\_\_ Plantar-flexion \_\_\_\_\_ Dorsiflexion \_\_\_\_\_ Diff.

24 H. \_\_\_\_\_ Plantar-flexion \_\_\_\_\_ Dorsiflexion \_\_\_\_\_ Diff.

48 H. \_\_\_\_\_ Plantar-flexion \_\_\_\_\_ Dorsiflexion \_\_\_\_\_ Diff.

APPENDIX J  
RANGE OF MOTION RAW DATA

CONTROL						STRETCH						COLD & STRETCH					
Subject No.	Pre-Exercise	24 Hours	48 Hours	24H-Preex.	48H-Preex.	Subject No.	Pre-Exercise	24 Hours	48 Hours	24H-Preex.	48H-Preex.	Subject No.	Pre-Exercise	24 Hours	48 Hours	24H-Preex.	48H-Preex.
1	48	59	57	11	9	21	71	77	76	6	5	41	74	64	67	-10	-7
2	74	79	71	5	-3	22	61	56	51	-5	-10	42	61	56	54	-5	-7
3	69	62	55	-7	-14	23	62	64	60	2	-2	43	65	62	64	-3	-1
4	68	69	74	1	6	24	73	83	73	10	0	44	57	54	60	-3	3
5	60	54	52	-6	-8	25	57	57	68	0	11	45	59	61	58	-2	-1
6	38	39	44	1	6	26	83	83	88	0	5	46	74	65	72	-9	-2
7	78	76	80	-2	2	27	63	60	58	-3	-5	47	71	69	71	-2	0
8	72	75	75	3	3	28	75	61	48	-14	-27	48	71	70	72	-1	1
9	59	57	59	-2	0	29	60	67	75	7	15	49	41	44	50	-3	9
10	53	39	40	-14	-13	30	75	55	56	-20	-19	50	77	74	74	-3	-3
11	69	71	59	2	-10	31	82	71	87	-11	5	51	72	80	73	8	1
12	58	57	59	-1	1	32	47	49	54	2	7	52	66	60	53	-6	-13
13	62	39	53	-23	-9	33	56	55	56	-1	0	53	63	66	73	3	10
14	82	82	88	0	6	34	56	55	64	-1	8	54	79	52	41	-27	-38
15	76	70	77	-6	1	35	62	61	62	-1	0	55	54	52	48	-2	-6
16	63	52	62	-11	-1	36	72	65	55	-7	-17	56	65	61	55	-4	-10
17	82	84	86	2	4	37	71	66	70	-5	-1	57	61	59	69	-2	8
18	49	53	66	4	17	38	62	57	61	-5	-1	58	75	57	52	-18	-23
19	72	72	78	0	6	39	79	69	70	-10	-9	59	67	67	70	0	3
20	63	57	51	-6	-12	40	82	77	73	-5	-9	60	67	68	67	1	0



## APPENDIX K

### PHOTOGRAPH OF STRETCHING PROCEDURE



Stretching Procedure

## APPENDIX L

RAW DATA FOR PERCEPTION OF SORENESS

Control			Stretch			Cold & Stretch		
Subject No.	24 Hours	48 Hours	Subject No.	24 Hours	48 Hours	Subject No.	24 Hours	48 Hours
1	39	21	21	24	9	41	0	0
2	59	33	22	66	87	42	0	0
3	65	33	23	0	0	43	49	64
4	17	16	24	80	67	44	26	32
5	0	0	25	78	41	45	66	48
6	65	66	26	40	32	46	63	40
7	23	31	27	87	66	47	0	0
8	5	2	28	75	99	48	0	0
9	10	30	29	37	50	49	57	43
10	67	74	30	74	88	50	60	45
11	63	66	31	8	0	51	65	90
12	54	35	32	1	0	52	76	87
13	60	68	33	31	18	53	4	4
14	33	0	34	48	48	54	43	76
15	36	12	35	16	10	55	34	66
16	45	32	36	49	57	56	43	58
17	6	0	37	40	27	57	1	0
18	66	66	38	53	42	58	81	99
19	37	14	39	46	62	59	42	66
20	66	66	40	40	60	60	29	9

\*All subjects recorded 0 on the pretest.

Scores are actually the number of millimeters measured from zero.

The scale ranges from 0 to 99.