THE EFFECTIVENESS OF STATIC STRETCHING

FOR THE PREVENTION AND THE TREATMENT

OF DELAYED MUSCLE SORENESS

by

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B.P.E., University of Calgary, 1968

A THESIS SUBMITTED IN PARTIAL FULFILLMENT

OF THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF ARTS

in the Faculty

of

Education

We accept this thesis as conforming
to the required standard

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September 1980

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ABSTRACT

The purpose of this study was to determine if static stretching could be used for the treatment and for the prevention of delayed muscle soreness. Twenty female subjects aged 20 to 35 years were assigned at random to two groups: Group A for treatment and Group B for prevention. Each group served as its own control and experimental group. Delayed muscle soreness was induced in all subjects following repetitions of concentric and eccentric contractions of the elbow flexors. Subjects in Group A during the experimental situation applied static stretching to the exercised arm after the soreness had developed, at 24 hours, 34 hours, 46 hours, 48 hours, 58 hours and 70 hours following the experimental exercise. Group B subjects in the experimental situation applied static stretching immediately and at 2 hours, 6 hours, 20 hours and 22 hours post-exercise. The McGill Pain Questionnaire was used to measure the perceived pain for each subject in the control and experimental situations at 24 hours, 48 hours and 72 hours following the experimental exercise. Results indicated that when compared

with the control group, each experimental group showed significantly lower levels of delayed muscle soreness. In the treatment Group A, major significance of .01 occurred at the 72 hour measurement period. In the prevention Group B, the exact location of the significant difference could not be determined. However, there was some statistical indication that the significant difference occurred at the 24 hour observation period.

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ACKNOWLEDGEMENTS

To my husband, Jerry, and my three children, Dennis, Jamie, and Cori, for their patience and understanding throughout this process of learning.

To my fellow graduate students, Jaye, Mary, Charlene, and Monty, for their support and encouragement.

To Judy Pitcher, for all her time and effort put into typing this thesis.

CHAPTER I

THE PROBLEM

Introduction

Delayed muscle soreness is a phenomenon that often arises when individuals are subjected to rigorous physical activity. Symptoms of pain, stiffness and soreness appear some hours after the exercise and may be correlated with an increased level of electrical activity in those muscles experiencing the soreness (de Vries, 1961b).

This type of muscular distress frequently disrupts fitness and training programs. Some authorities state that delayed muscle soreness is a major factor that keeps many people from participating in physical activity (Lamb, 1978). Many individuals are not able to cope with this type of muscle pain and therefore either quit or shy away from fitness and activity programs. Jensen (1979) states that this muscle soreness also affects highly trained athletes. Such individuals, highly motivated towards continuing intensive physical training, are usually able to endure the pain and continue participation. There may, however, be a delay in the training program. Komi and Buskirk (1972) reported that some individuals showed a significant decrease in their ability to exert maximal tension during the first week of conditioning. This was attributed to the presence of delayed muscle soreness.

De Vries attempted to alter the predictable pattern of muscle soreness. His studies (1961a, 1961b, 1966) suggested that static stretching could be used for the treatment and also for the prevention of delayed muscle soreness. Other researchers have not been able to obtain such results. Abraham (1977) noted that static stretching provided relief for only one to two minutes. McGlynn (1979) reported that his subjects had no significant decrease in perceived pain when they employed static stretching. Thus, the effectiveness of static stretching to reduce individual pain levels resulting from intensive, repetitive exercise has not been resolved.

Discrepancies in pertinent research results may be due to inconsistencies in methodology and design, such as demonstrated in the literature review. By modifying previous research design and utilizing improved instrumentation, a more reliable and valid study was attempted to determine the effectiveness of static stretching for the prevention and for the treatment of delayed muscle soreness.

Statement of the Problem

This study attempted to assess the effect of static stretching on the development of delayed muscle soreness. Specifically, its purpose was to determine if static stretching could prevent delayed muscle soreness, and to evaluate the effectiveness of static stretching as a method of treatment for delayed muscle soreness.

Hypotheses

To fulfill the purposes of the study, the following hypotheses were tested.

1. In the treatment section, there is no significant difference in pain levels between the experimental group and the control group at the 24 hour, 48 hour and 72 hour measurement periods.

2. In the prevention section, there is no significant difference in pain levels between the experimental group and the control group at the 24 hour, 48 hour and 72 hour measurement periods.

3. In the treatment section, there is no significant difference in pain levels between the control group and the experimental group.

4. In the prevention section, there is no significant difference in pain levels between the control group and the experimental group.

Delimitations

The delimitations of the study were:

1. the subjective response of the subjects' rating of pain;

2. the motivation of the subjects in the experimental exercise;

3. the willingness of the subjects to comply with instructions concerning their physical activity outside of the laboratory;

4. the lack of the researcher's control over what the subjects do from the time of the experimental exercise to the subsequent tests; and

5. the experimental exercise is not the only type of exercise that can cause delayed muscle soreness, however, it is considered to be a good representative so that generalizations can be made.

Limitations

The limitations of the study are:

1. the skill of the investigator in administering the McGill Pain Questionnaire; and

2. the skill of the investigator to communicate designated instructions to the subjects.

Definition of Terms

Static Stretching: Static stretching is a technique involving holding a stationary position for a period of time whereby the muscle to be stretched is placed in a position of greatest possible length. For this study the stretch position was held for two minutes.

Delayed Muscle Soreness: Delayed muscle soreness includes all symptoms of pain, stiffness or soreness that occurs in the exercised muscle from four hours to seventy-two hours after the exercise.

Unusual Activity: Unusual activity is any activity beyond that which the subjects are accustomed to in their normal daily routines.

CHAPTER II

REVIEW OF LITERATURE

Introduction

Skeletal muscle pain is a frequent companion of physical exertion and may be localized in the muscle tissue, the supporting connective tissue or both. Trauma of the muscle organ, such as contusion or strain, results in soft tissue injury and may require medical attention. However, two types of muscle pain induced through exercise are non-pathologic: immediate muscle soreness and delayed muscle soreness (Hough, 1902; de Vries, 1966a, Abraham, 1979).

Immediate soreness occurs during the exercise and immediately following it. Such pain is associated with the period of ischemia which accompanies contracting muscles (Rodbard, 1975). A noxious metabolite is released from the muscle and into the tissue fluid. Rodbard (1975) refers to this pain substance as a toxic catabolite which serves as a protective mechanism for the muscle. Dorpat (1955) postulates that such a catabolite or "Factor-P" is the diffusible potassium that travels outward across the cell membrane. Once it accumulates in concentration sufficient enough to exceed the pain threshold, the individual experiences increasing levels of fatigue and pain, until finally the exercise must be discontinued. The amount of accumulation depends on the form, intensity and duration of contraction (Dorpat, 1955). Pain may also be associated with the buildup of other metabolites which presumably apply pressure on the pain receptors. The accompanying discomfort continues until either the intensity of the exercise is reduced or the exercise is terminated. Ensuing relaxation of the muscle organ allows the blood flow to remove the by-products of muscle contraction.

Delayed muscle soreness develops some hours or even days after physical exertion. Symptoms of pain, soreness, and stiffness usually appear approximately twelve hours after the exercise, but may occur as early as four hours (de Vries, 1961a). These symptoms become more severe during the day following the exercise bout, peaking at 24 hours (McGlynn, 1979; de Vries, 1961a), or even 48 hours later post-exercise (Talag, 1973). Individuals are usually symptom-free after four to six days.

Physiology of Muscle Soreness

Investigation into delayed muscle soreness has resulted in speculations as to the possible cause or causes. Four theories proposed relate to the cause. As early as 1902, Hough set up ergographic studies contracting a muscle against a resistance. He found that delayed muscle soreness occurred more often when the muscle was subjected to high tensions. Also, soreness occurred more frequently if the rhythm was "jerky," that is, if the contraction to relaxation ratio was .5 sec/1.5 sec. He concluded that soreness originates through tissue rupture within the muscle. This "torn tissue" theory has been supported by other investigators (Hill, 1951; Astrand, 1977).

The thesis of muscle organ-structural damage has lacked histological evidence. In 1961 de Vries (1961a) advanced his hypothesis, the "spasm theory." He suggested that following intensive exercise the muscle develops an increasing inability to achieve complete relaxation, resulting in tonic spasms of motor units. Rationale for such an idea begins with the fact that exercise above a certain level causes a degree of ischemia in working muscles. The pain substance or "P-substance" that is released out of the muscle cells and into the tissue fluid accumulates, stimulating pain receptors. This pain initiates a reflex tonic muscle contraction, inducing further ischemia, and a phasic cycle of vasoconstriction and pain ensues.

Karpovich (1965) suggested that prolonged intensive work causes an excessive accumulation of waste products, producing physiological changes in muscle fibres. Such sarcoplasmic conditions include a rise in somatic pressure, resulting in the retention of water and local edema, with compression of nociceptors causing pain. Since some of these cellular chemical processes are slow, it may require several hours for soreness to develop.

The fourth theory is related to Hough's (1902) hypothesis but is much more specific. In Asmussen's (1956) experiments, subjects performed either positive or negative work. Soreness was much more common in subjects that had been doing the negative work. Also, pain seemed localized more in tendonous areas. He concluded that connective tissue of exercised muscle organs was the site of injury and accompanying pain. Asmussen's results indicated that muscle soreness apparently could not be the result of metabolite build-up, as the negative-working muscles developed more soreness and yet did much less metabolic work. Later researchers confirmed eccentric work as causing greater soreness (Komi and Buskirk, 1972; Talag, 1973).

Abraham (1977, 1979) found a significant correlation between peaks of maximum urinary hydroxyproline (a breakdown product of collagen fibre) excretion and the times of maximum perceived soreness. He concluded that delayed soreness is most closely related to irritation of the connective tissue.

It is evident that there is still a great deal of controversy over the cause of delayed muscle soreness. A recent suggestion is that both connective tissue damage and spasms likely contribute (Jensen, 1979; ATA, 1974).

Related Literature

Controlled research concerned with the prevention and treatment of delayed muscle soreness is limited. The probable explanation may be due to the elusive characteristics of the condition and the obvious difficulty of measurement.

There have been only two studies that have attempted to evaluate methods of preventing delayed muscle soreness. Staton (1952) felt that because ascorbic acid plays a prominent role in maintaining the cohesiveness of the cells of the blood vessels and other tissues, it might make muscle fibre more resistant to damage. The experimental group was fed heavy doses of ascorbic acid for 30 days. A sit-up test was used to measure soreness by comparing the number performed before and during the period of muscle soreness. When compared to a control and placebo group, results were not significant.

The second study involved the use of static stretching to prevent muscle soreness. De Vries (1961a) induced muscle soreness in both arms of the subjects through a repetitive series of wrist hyper-extensions. The subjects applied static stretching to the wrist extensors and flexors of the non-dominant arm immediately following the exercise bout and two hours, six hours, 20 hours and 22 hours following it. A static stretch of one minute duration was accomplished in each case. The results showed less muscular distress in the non-dominant arm at significant levels when measured at the 24 hour and 48 hour observation periods.

De Vries (1961b) also incorporated static stretching for the treatment of delayed muscle soreness. He used electromyography to measure muscle soreness and found increased electrical activity in those muscles feeling distress. When static stretching was applied for one to three minutes, the EMG readings were lowered and the individuals verbally expressed symtomatic relief. There was no instrument used to measure pain and because the subjects were referred to the researcher from other sources, he was unable to specify the times following the exercise that the static stretch technique was administered.

De Vries in a later study (1966) again reported positive findings. He induced soreness in the forearm flexors and 48 hours following the exercise, he measured the pain levels of the subjects. They then performed the static stretch for two minutes followed by a one minute rest. The sequence was then repeated. A pain measurement was taken and the subjects indicated symptomatic relief. The EMG recordings also verified his earlier findings. Because there were no serial measurements of pain taken it is not known how long the subjects experienced a diminished degree of muscle soreness.

Abraham (1977) induced soreness in the forearm flexors and measured pain 24 hours after the experimental exercise. The subjects then either stretched or flexed the arm and pain levels were immediately re-evaluated. This same procedure followed at 48 hours. Results showed that both procedures provided relief for one to two minutes. The duration of the stretch was not recorded. He reported no relationship between the development of muscle soreness and the magnitude of EMG activity in the exercised muscles. Static stretching as well, did not cause any change in the electrical activity of the muscles.

McGlynn (1979) introduced biofeedback as a method to reduce muscle soreness and compared it to static stretching. The subjects employed static stretching at six hours, 25 hours, 30 hours, 40 hours and 54 hours following the experimental exercise. The stretch was held for a period of two minutes, followed by a one minute rest. This sequence was repeated four times. The subjects pain levels were recorded immediately after the exercise, and at 24 hours, 48 hours and 72 hours following it. Results showed that when compared with a control group, both auditory biofeedback and static stretching significantly reduced EMG activity but had no significant effect on perceived pain.

Conflicting results of these studies make it difficult to determine how effective static stretching is for the treatment of delayed muscle soreness. A number of factors may contribute to these discrepancies. These include:

1. differences in the duration of the static stretch procedure;

2. differences in the designated times following the experimental exercise when static stretching was done;

3. the limited number of pain measurements;

4. studies by de Vries (1961b, 1966) and Abraham (1977) were terminated before any long term effects could be measured;

5. the experimental design needs to be more complete. De Vries (1961b, 1966) and Abraham (1977) did not use a control group for testing the use of static stretching. McGlynn (1979) did not use the most accepted procedure to measure the subjective pain response; and

6. the choice of instrument used to measure pain.

The instrument used to measure pain is a major criticism of the studies concerned with both the prevention and the treatment of delayed muscle soreness. These studies rated pain on a three point scale indicating mild, moderate and severe. Behavioral indices were then attached to these ratings. Kierns (1980), Research Officer for the Ministry of Health, British Columbia, has done extensive work in pain research incorporating various methods for measuring pain. He stated that the type of scale used in the studies to measure muscle soreness lacks validity and is not sensitive enough to note changes in pain.

In order to obtain the consistent results of assessment of static stretching procedures to prevent and treat delayed muscle soreness, it is necessary to implement consistent and reliable measurement procedures.

Static Stretch Mechanism

De Vries (1961a) developed the static stretch idea in support of his "spasm theory." He explains the rationale of this method through the interaction of the slow stretch with both the muscle spindles and the golgi tendon organs. Steady stretch has two effects on these proprioceptors. First of all, it depresses the monosynaptic response thereby reducing the stretch reflex.

Secondly, it activates the golgi tendon organs further depressing activity. These golgi tendon organs inhibit contraction and thus protect an overstrained muscle. In this way, static stretching disrupts possibilities of spasms by an inhibitory reflex arc.

There may be a relationship between the static stretch technique and the Melzack-Wall (1977) gate control theory of pain. This theory discusses low level types of stimulation as one of the sensory methods to control pain. Stimulation such as tactile stimuli, electrical stimuli, application of cold or heat and intense pressure selectively activate the large, A-beta fibres thereby blocking or inhibiting the pain mechanism. The stretching of the muscle may perform a similar function.

CHAPTER III

THE METHOD

The Design

The nature of the problem of the study required two separate experimental designs involving two different groups of people. Group A was arranged to evaluate the effectiveness of static stretching for the treatment of muscle soreness. In this group the experimental treatment was used only after delayed muscle soreness had developed. Group B was set up to test the usefulness of static stretching to prevent muscle soreness. The experimental treatment began immediately after the experimental exercise. Due to the nature of the subjective response, each group served as its own experimental and control group. Two weeks separated each situation. In this way, each subject's responses were compared in the experimental and control situation.

At the time of the first administration of the experimental exercise, each group was randomly divided in half. Five subjects in each group served as the experimental subjects and the other five were the control subjects. Two weeks later, the experimental exercise was again administered and the roles of the subjects were reversed. The purpose of this procedure was to prevent any sort of learning or training bias from occurring between the experimental and control situations. (See Figure 1).

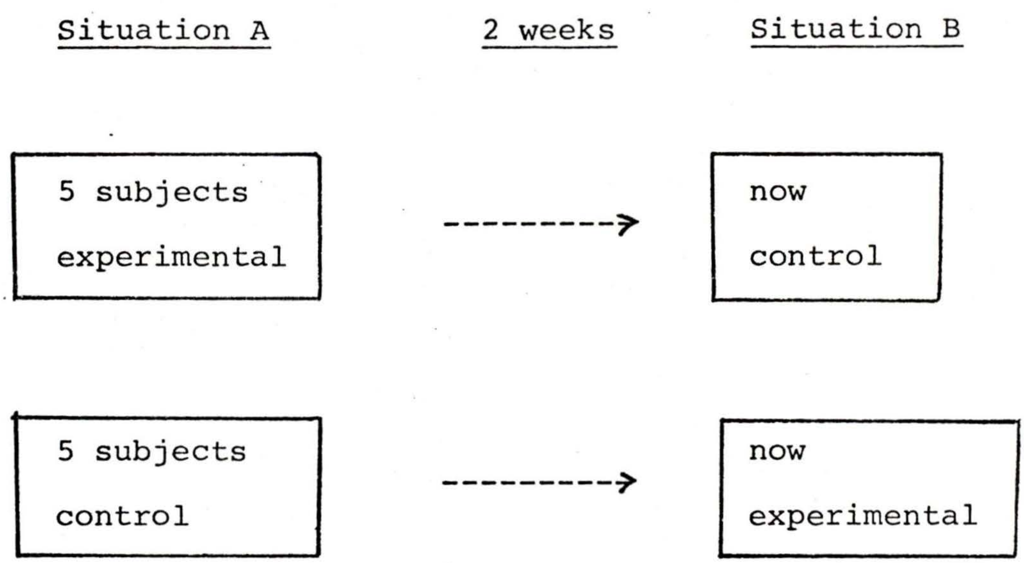
The measurement of pain levels for each subject were taken at designated time intervals. Previous studies by de Vries (1961a, 1961b, 1966) showed that static stretching decreased electrical activity of the muscle and also gave symptomatic relief. McGlynn (1979) however, noted that although the electrical activity decreased by using static stretching, individuals showed no drop in their levels of perceived pain. Abraham (1977) also noted that his subjects had no significant decrease in their perceived pain for any length of time after using static stretching. These discrepancies indicate that evaluation of static stretching depends heavily on an improved instrument for measuring pain levels of experimental subjects.

Sample Selection

The subjects were 20 female volunteers between the ages of 20 and 35 years. Subjects agreed not to engage in any unusual activity with their arms for two days prior to the experiment. Each person was questioned as to what types of physically-demanding activities they had been involved in within the previous two days. This was

Figure 1

Experimental Design



necessary to ensure that delayed muscle soreness did not occur as a result of some activity other than the experimental exercise bout. The subjects were then randomly assigned, according to a table of random numbers, to one of two groups: Group A or Group B.

The time factor between the experimental exercise and the subsequent tests was controlled for by giving each subject specific instructions regarding this period. These were as follows:

1. subjects were to refrain from participating in any unusual activities using their arms;

2. subjects were to refrain from taking any types of medication;

3. subjects were to refrain from attempting to use any other treatment method for the muscle soreness such as ice, massage or heat; and

4. subjects were allowed to shower only, and to refrain from entering hot baths, saunas, whirlpools.

Experimental Exercise

Previous studies (de Vries, 1961a, 1966; Talag, 1973; Abraham, 1977; McGlynn, 1979) had shown that the most acceptable and reliable method for attaining delayed muscle soreness was through a series of repeated contractions of a muscle organ to a point of fatigue. Intermittent rest periods enable experimental work bouts to continue longer and therefore a higher percentage of subjects experience soreness in the exercised muscle.

Delayed muscle soreness was induced in the forearm flexors - the biceps brachii, the brachialis, and the brachioradialis - of the non-dominant arm, using a one-arm dumbbell "curl." The subject was positioned so that no other part of the body could aid the flexors in the movement. The subject was seated in a chair and a portable arm rest was placed at the side of the chair of the non-dominant arm. The elbow was then positioned comfortably on the arm rest platform (13" x 7") and extended to a joint angle of 180°. In order to position the arm horizontally, the height of the arm rest was adjusted. Adhesive tape was applied to the wrist to prevent flexion and hyperextension of the wrist during the exercise.

The weight of resistance was placed in the subject's hand with the forearm supinated. The forearm was then flexed to a 45° angle at the elbow before being extended back to 180°. A goniometer was used to measure the joint angles. Thus both concentric and eccentric contractions were involved. The experimental exercise was divided into work and rest periods. A work period was termed a set and began with the maximum number of curls per set equalling ten. At the end of the set, the subject rested.

Each subject started the exercise with the weight of resistance that they could only perform ten executions. This weight was determined from the following set of weights: 25 lbs., 20 lbs., 15 lbs., 12 lbs., 10 lbs., 9 lbs., 8 lbs., 7 lbs., 6 lbs., 5lbs., 4 lbs., 3 lbs., 2 lbs. When the number of repetitions in a set equalled three, the weight was reduced. If the subject began with a weight of 15 pounds or more, the weight decrement for each succeeding set was five pounds. If the starting weight was less than 15 pounds, the weight decrement was two pounds. Rest periods between the sets with the initial weight were of one minute duration. When the weight of resistance was lowered the rest periods were 30 seconds in length. The subject continued until the number of curls again equalled three and the weight was reduced. This procedure continued for one hour. Then the weight was again reduced and the subject completed as many repetitions as possible. The total work performed was carefully equated during the control and experimental work bouts.

Experimental Treatment

In order to put the forearm flexors in a position of greatest possible length, a method adapted from that of de Vries (1966) was found effective. De Vries' (1966) technique of locking the hands behind the chair was modified because in preliminary trials it was noted that some subjects could not keep their elbows locked when their hands were in this position. The subject sat in a straight back chair and grasped a towel with both hands behind the back of the chair. The hands, with palms up, were positioned as close together as possible on the towel, while maintaining the elbows in a locked position. In order to gain the greatest possible hyperextension of the arm at the shoulder, an assistant grasped the subject's wrists and gently aided in the movement. This position of complete extension was held for two minutes, followed by a one minute rest. The sequence was then repeated.

Group A was involved with using static stretching for therapy, and therefore the stretching maneuvre was initiated after the soreness developed. Previous studies (Talag, 1973; Abraham, 1977; McGlynn, 1979) had shown that soreness appeared in all the subjects 24 hours after the experimental exercise, therefore, immediately following the 24 hour measurement, the static stretch procedure was performed. Following a stretching protocol suggested by de Vries (1974), the subjects performed the static stretch procedure two more times before the 48 hour measurement period, at 34 hours and 46 hours following the experimental exercise. Following the 48 hour testing period static stretching was done immediately, at 58 hours, and finally at 70 hours, two hours prior to the 72 hour measurement.

Group B subjects in the experimental situation employed static stretching for the prevention of muscle soreness. Therefore, they applied the procedure immediately after the exercise and at 2 hours, 6 hours, 20 hours, and 22 hours following the experimental exercise. These times were designated by de Vries (1961a) as being effective.

Each subject in the experimental situation of Group A and Group B received a form (Appendix A) which designated the necessary times of day that the experimental treatment was to be done and also the times that they were to be measured for pain perception. The times were recorded by the researcher once the experimental exercise had been completed. Subjects were required to check off each stage as it was accomplished. All subjects completed each requirement.

The researcher was unable to assist the subjects in performing the static stretch procedure at all the designated times. In Group A that was using static stretching for the treatment of delayed muscle soreness, the researcher assisted at the 24 hour and 48 hour time periods. For Group B using static stretching for the prevention of delayed muscle soreness, the researcher assisted each subject in the static stretch procedure immediately following the experimental exercise. The remaining times for the experimental treatment were performed by each subject at home.

Instrumentation

There are at present, no reliable physiological indices of pain (Hilgard, 1969). Because of this, researchers (Beecher, 1959; Melzack, 1977) seem to agree that the subjective response of the subject is more specific and sensitive for pain measurement. The instrumentation therefore, consisted of using the most acceptable and reliable method of measuring pain. This took the form of the McGill Pain Questionnaire which was developed by Melzack (1975). The McGill Questionniare is used by other researchers for various types of pain (Kierns, 1980; Leavitt, 1978, 1979). Kierns, the Research Officer for the Ministry of Health, British Columbia, has found the McGill Questionnaire to be the most reliable and valid in his pain research. Elton (1979) compared five different methods for measuring the subjective response of pain and found that the McGill Pain Questionnaire showed high reliability. In a practical situation, physiotherapists at the Gorge Road Hospital in Victoria, B.C., frequently find this instrument helpful.

Quantitative data can be obtained through three main measures.

1. Pain Rating Index (Rank). To obtain the PRI(R), subjects choose words from a series of adjectives that best describe the pain. The words in each group are placed in order of intensity and can be assigned a value. The word for least pain is given a value of one, the next word is given a value of two and so on. The sum total of the scale values of all the words chosen is then calculated to give the RRI(R).

2. Present Pain Intensity (PPI). The number-word combination chosen as the indicator of overall pain intensity at the time of administration of the questionnaire.

3. Number of Words Chosen (NWV). The total number of adjectives chosen is recorded.

Melzack (1975) found these three measures to show significant correlations. However, for measuring changes in pain levels the PRI(R) was the more valid and sensitive index. Therefore, the PRI(R) was the criterion measure used for the study (Appendix B).

The Melzack Pain Questionnaire was administered through an interview with the subjects. All instructions and interview procedures were the same for each subject. To aid in test-administration reliability and validity, the words were typed on a cardboard card and shown to the subjects so that they could easily recognize each word as it was being communicated by the researcher. In case some subjects were unfamiliar with the meaning of a particular word, the Gage Canadian Dictionary was used.

Data Collection

Data was collected for Group A and Group B through the use of the McGill Pain Questionnaire. Each subject in both groups had a total of six pain measurements taken, three in the experimental situation and three in the control situation. These were done at 24 hours, 48 hours, and 72 hours following the experimental exercise. Measurements were taken either at the laboratory or at the subjects home.

Data Analysis

Group A and Group B were treated separately as there was no need to make any data comparisons between these two groups. The treatment of the data for each group, however, was handled in a similar statistical manner.

The major statistical tool used was the nonparametric sign test. De Vries (1961a, 1966) and Melzack (1975) utilized this method of analysis for comparing changes in pain. The .05 level of confidence was accepted for significance. To maintain nonparametric consistency, the

Wilcoxon Matched Pairs Signed Ranks Test was used as a secondary statistical test of significance. This was used to verify significant results of the sign test and to identify further possibilities. If both tests indicated significance then the result was considered highly meaningful. When one test showed significance and the other insignificance, then the result was questionable.

These tests were used to determine if there were any significant differences between the control group and the experimental group in the treatment and the prevention section. Also, they were used to determine if significant differences occurred in the 24 hour, 48 hour, or 72 hour time periods between the control and experimental groups.

It was necessary to use nonparametric statistics throughout the analysis because of the non-normality of the scores. Problems arose when parametric statistics were utilized. When means were used for the establishment of confidence intervals, all results showed insignificance even though significance was indicated through other parametric tests. The use of means and t-tests also gave too much weight to specific scores. For example, when comparing the scores for the 24 hour measurement period in Group A, the elimination of one highly irregular pair of scores resulted in a change from insignificance to significance.

CHAPTER IV

RESULTS AND DISCUSSION OF RESULTS

Results

The results of this study are presented in two separate sections corresponding to the parts of the overall problem as stated in Chapter I.

Prevention of Delayed Muscle Soreness

Pain levels for each subject at the specific time periods, in both the control and experimental situations are presented in Table I. Medians for each subject and each time period are also shown.

A comparison was made between the control and experimental group by using the medians for each subject. The sign test indicated a significance of .05 as eight out of ten subjects experienced a lower level of pain in the experimental situation. The Wilcoxon Matched Pairs Signed Rank Test also showed significance at the .05 level. Thus the experimental group expressed a significantly lower level of muscular distress than the control group.

TABLE I: PAIN LEVELS FOR EACH SUBJECT AT SPECIFIC TIME PERIODS IN CONTROL AND EXPERIMENTAL SITUATIONS

	Control				Experimental			
	24 hr	48 hr	72 hr	Median	24 hr	48 hr	72 hr	Median
	28	24	7	24	13	6	0	6
	20	6	1	6	5	6	1	5
	19	8	0	8	5	5	0	5
	11	17	2	11	14	2	1	2
	12	11	6	11	6	2	0	2
	9	8	4	8	7	8	1	7
	14	12	8	12	9	10	10	10
	4	7	1	4	7	11	1	7
	8	7	2	7	4	3	2	3
	6	7	1	6	7	13	7	7
	132	107	32		77	66	23	
Median	11.5	8	2		7	6	1	

Further tests were made for each set of observations independently and the results are shown in Table II.

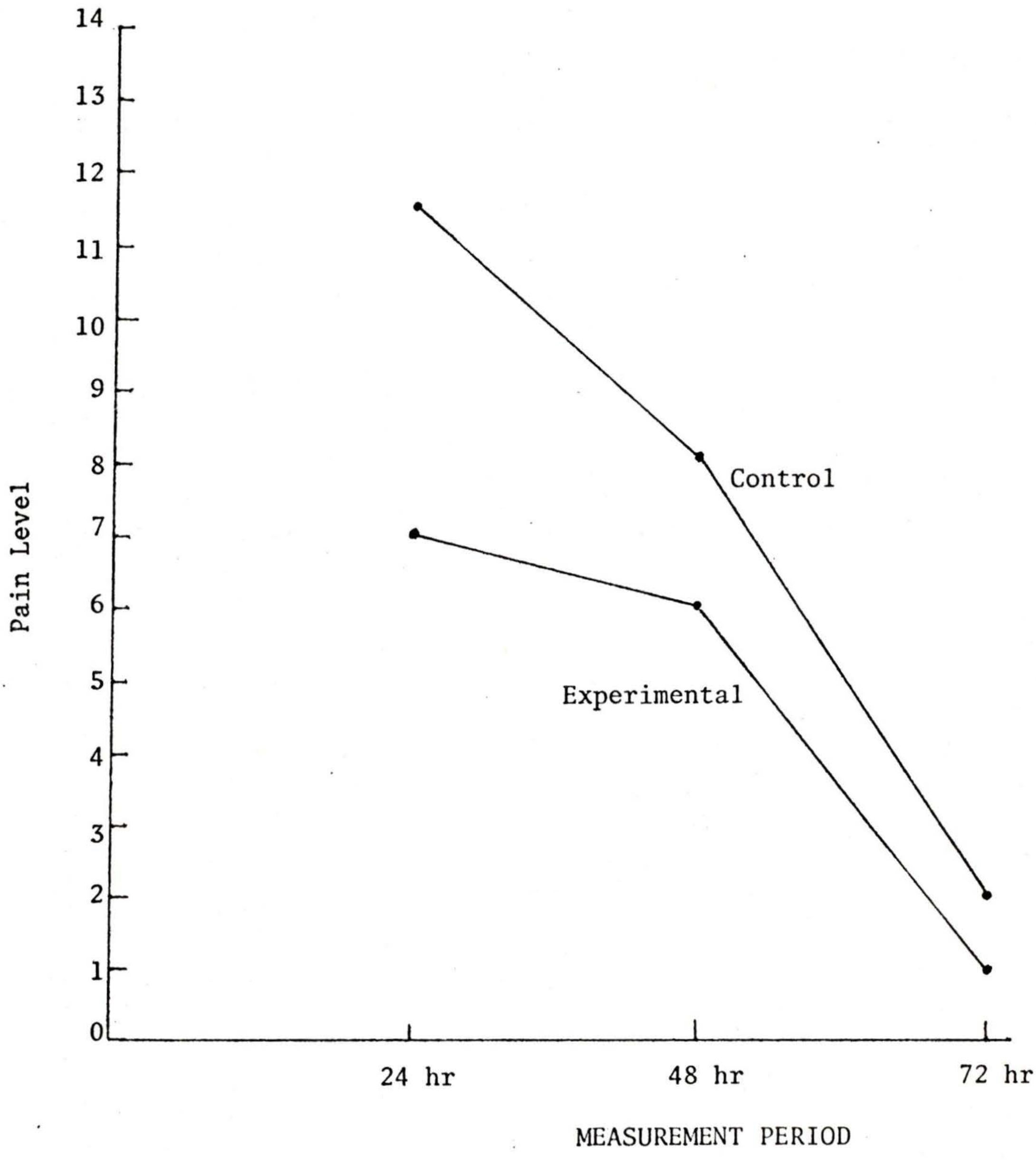
TABLE II: RESULTS OF INDEPENDENT SET OBSERVATIONS

Time	Sign Test	Wilcoxon
24 hr	7/10 = .17 (N.S.)	p = .05 (S)
48 hr	6/8 = .14 (N.S.)	p = N.S.
72 hr	4/6 = .34 (N.S.)	p = N.S.

The sign test shows no significance at any of the three time periods. The Wilcoxon test, however, indicates a significant level of .05 at the 24 hour observation period. One possible explanation for this is that the 24 hour time period contains more of the previously recorded significance between the experimental and control groups than the other time periods. A larger sample size may have resulted in the difference at the 24 hour measurement period being highly significant. As illustrated in Figure 2, the post-exercise 24 hour observation period had the greatest difference in median pain levels.

Figure II

Comparison of Median Pain Levels Between The Experimental And Control Group at Specific Measurement Periods



Treatment of Delayed Muscle Soreness

The recorded pain levels for each subject at each observation period in the control and experimental situations are shown in Table III. The medians for each person and each time period are also shown.

Medians for each subject were used to compare the control and the experimental groups. The sign test indicated that seven out of eight subjects had a decrease in pain levels during the experimental situation, a significance level of .03. The Wilcoxon test of this difference was significant at the .02 level. These results demonstrate that the experimental or stretch group had a significantly lower level of delayed muscle soreness than the control group.

Table IV presents the results of comparing the control and experimental groups at the specific observation points.

These results indicate a highly significant difference between the control and experimental group at the 72 hour observation period. The stretch group displayed an apparently considerably lower level of pain at this time than the control group.

TABLE III: RECORDED PAIN LEVELS FOR SUBJECTS AT EACH OBSERVATION PERIOD IN THE CONTROL AND EXPERIMENTAL SITUATIONS

	Control				Experimental			
	24 hr	48 hr	74 hr	Median	24 hr	48 hr	72 hr	Median
	17	15	7	15	15	11	5	11
	4	9	8	8	3	4	0	3
	10	4	1	4	8	4	0	4
	7	3	0	3	3	3	1	3
	8	4	2	4	4	1	0	1
	10	11	7	10	11	5	1	5
	8	6	2	6	9	3	1	3
	8	3	1	3	7	2	0	2
	4	5	0	4	12	3	0	3
	6	4	5	5	6	10	0	6
	82	64	33		78	46	8	
Median	8	4.5	2		7.5	3.5	0	

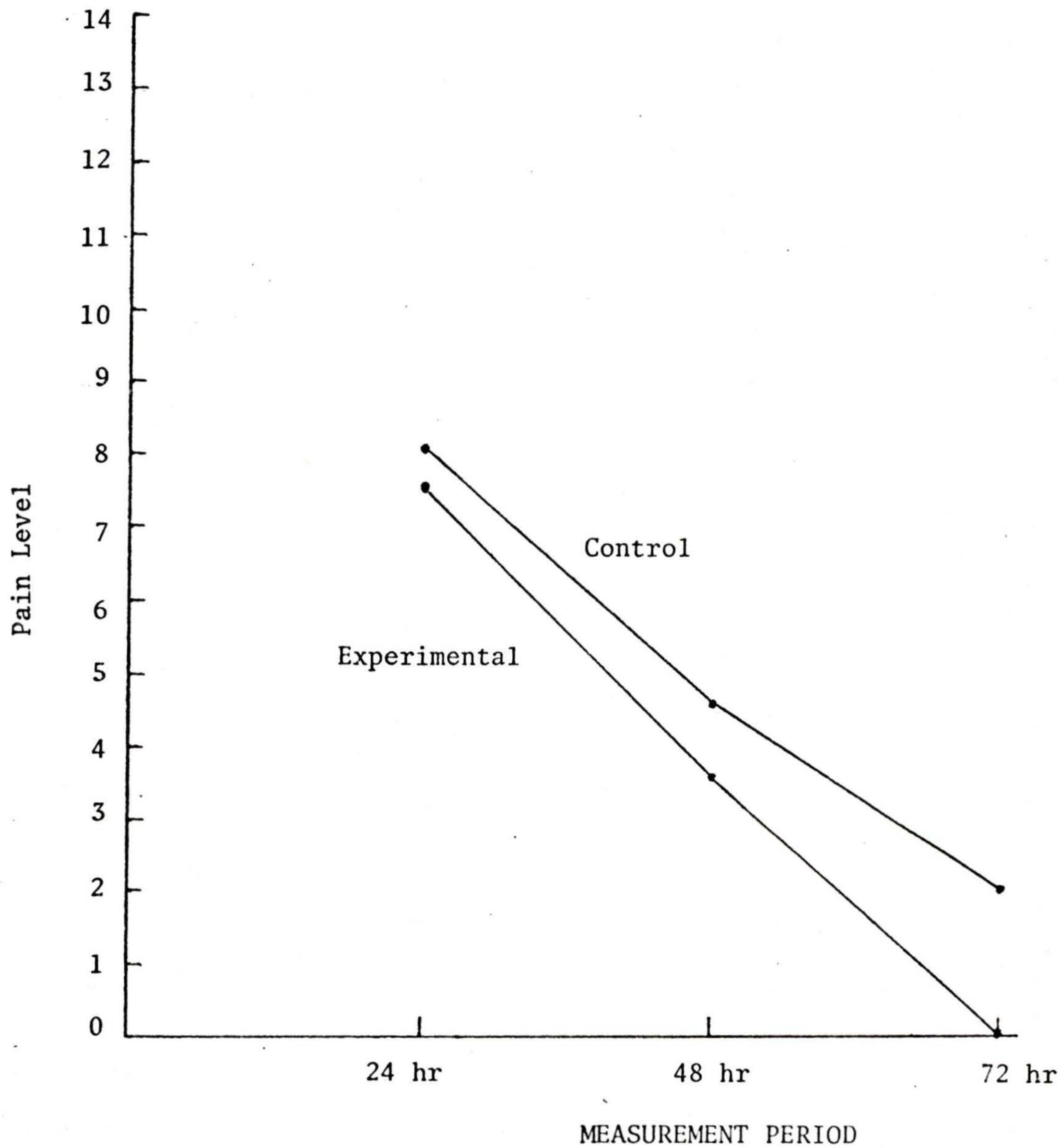
TABLE IV: COMPARISON RESULTS OF CONTROL AND EXPERIMENTAL GROUPS AT SPECIFIC OBSERVATION PERIODS

Time	Sign Test	Wilcoxon
24 hr	6/9 = .25 (N.S.)	p = N.S.
48 hr	7/8 = .03 (S)	p = N.S.
72 hr	8/9 = .01 (S)	$p < .02$ (S)

At the 48 hour measurement, some significance was indicated by the sign test. The Wilcoxon test however, failed to verify this result. This possibly suggests that although the majority of the significance between the control and experimental groups as a whole is present at the 72 hour measurement period, some statistical significance is starting to emerge at the 48 hour observation period. Figure III graphically portrays the difference between median scores of the experimental and control groups at each measurement period. It demonstrates clearly that at the 72 hour time period the stretch group is displaying significantly less muscle soreness than the control group. The 24 hour and 48 hour observation periods show a non-significant divergence between the groups. Possibly with a much larger sample there would be a greater difference between the experimental and control group at the 48 hour measurement period, thus making this difference highly significant.

Figure III

Comparison of Median Pain Levels Between The Experimental And Control Group at Specific Measurement Periods



Discussion of Results

The data of this study support de Vries proposal of beneficial effects of static stretching. De Vries (1961a) found a significantly lower level of pain in subjects using static stretching to prevent delayed muscle soreness. De Vries (1961a) was also able to show significantly lesser magnitudes of muscular distress in the experimental group at both the 24 and 48 hour observation periods. The results of this study did not determine precisely at what measurement period the significant difference occurred between the experimental and control groups although there is some evidence the major area of lessened muscular distress was at the 24 hour measurement period (Wilcoxon $p = .05$). This corresponded to the opinions of the majority of the subjects, who stated that when they had to stop the stretch manoeuvre, the muscle began to 'tighten up and become sorer.' For this group of subjects the effects of static stretching are 'short term.'

In the treatment section, comparisons between the results of this study and those of previous studies are difficult because of the differences in design. This study demonstrated that the experimental group that performed static stretching after the soreness developed had significantly ($p = .03$) lower levels of pain than those that did not stretch. In particular, the results showed that during the 72 hour observation period the difference between the experimental and control group was significant at the .01

level. The data in Table III indicates that six out of ten subjects in the stretch group were free from muscle soreness at this measurement period, whereas only two subjects were pain free in the control situation. This information could be of clinical importance if it were known how long beyond the 72 hour observation period the untreated muscle experienced discomfort. Some authorities have stated that delayed muscle soreness may last as long as six days (Astrand, 1977). Lesser statistical significance was apparent for comparisons of the data from the 48 hour observation period, where the sign test indicated significance at .03 but the Wilcoxon test was insignificant. This possibly indicates that the 48 hour measurement period contains some of the previously recorded significant difference between the control and experimental groups.

De Vries (1966) demonstrated a significant decrease in pain levels following static stretching. However, in his study the subjects did not begin stretching until 48 hours after the exercise. Also, pain measurements were only recorded immediately following the stretch, so that any long term effects were not measured serially. McGlynn (1979) found no significant differences in perceived pain over the three day period. His subjects performed stretching before and after soreness had developed.

The majority of the subjects in the treatment section stated that the static stretch procedure 'felt good.'

Some reported that the beneficial effects only lasted for one to two hours. This is a somewhat longer time than Abraham (1977) found in his subjects who reported relief for only one to two minutes. This discrepancy could possibly be the result of the length of time that the stretch position was maintained. Abraham's subjects did not hold the stretch for any designated period of time whereas all the subjects in the present study held the stretch for two minutes.

The consensus of the subjects in Group A and Group B was that the two minute stretch position provided some discomfort particularly in the shoulders and therefore would be less stressful if held for only one minute. De Vries (1961a, 1961b) incorporated a stretch of one minute which proved beneficial.

The present study introduced a different method for measuring delayed muscle soreness than was used in previous studies. The PRI(R) of the McGill Pain Questionnaire proved to be a useful instrument for collecting data. It was easy to administer and all subjects were able to readily select words to describe their pain. Since the subjects were not familiar with the scoring procedure, the validity of the PRI(R) was increased. The instrument was sufficiently discrete to detect changes over each three day period with the direction of the change corresponding to subjective expressions of the subjects.

The results of this study provided some supportive evidence for the usefulness of static stretching for the treatment and for the prevention of delayed muscle soreness. Problems inherent in the experimental design limit the applicability of these results. The design involved inducing delayed muscle soreness twice on each subject. All subjects expressed that they experienced less muscle distress on the second occasion. Although the majority of subjects recorded lower pain levels, some subjects recorded higher levels of muscle distress at certain observation periods. This discrepancy between the verbal feedback of the subjects and the recorded pain measurement raises some question as to the validity of the McGill Pain Questionniare. Also, the lack of consistency in recorded pain measurements between the two work bouts makes interpretation difficult.

There are several factors that may account for the differences in perceived pain between the two work bouts. The physiological status of the muscle organ may have changed during the two weeks between tests. The muscle could have become stronger during this time if the subjects were frequently engaged in strenuous labor with their arms. A motivational effect was present in some subjects as they did not put as much effort into the experimental exercise the second time. Psychologically, it was apparent that the subject's reactions to this type of muscle soreness depended somewhat on their personal situation. Therefore, on the second administration of the exercise, any particular problems or personal events may have been responsible for a different perception of pain. Also, immediately following the second work bout, several subjects anticipated extreme soreness before it had developed. Later, when the muscle experienced soreness, it was not as painful as they had expected, therefore, subjects assumed that the magnitude of distress was less than what they remembered from the first work bout. The familiarization of the subjects with the questionnaire, may have caused subjects to be more expressive the second time, thereby influencing the pain score.

Because of the limitations in design, the results of comparisons between the experimental and control group are of undetermined validity.

CHPAPTER V

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

Summary

The purpose of this study was to determine if static stretching could be used for the treatment and for the prevention of delayed muscle soreness. Twenty female subjects aged 20 to 35 years were randomly assigned to two groups: Group A for treatment and Group B for prevention. Each group served as its own experimental and control group. Muscle soreness was produced in all subjects following repetitions of concentric and eccentric contractions of the elbow flexors. The subjects in Group A during the experimental situation applied static stretching to the exercised arm after the soreness had developed. The static stretching was done at 24 hours, 34 hours, 46 hours, 48 hours, 58 hours, and 70 hours after the experimental exercise. Group B in the experimental situation applied static stretching immediately after the exercise and 2 hours, 6 hours, 20 hours and 22 hours later. Observations of perceived pain for each subject in the control and experimental situations were made at 24 hours, 48 hours, and 72 hours following the experimental exercise. When compared with the control group, each experimental group showed significantly less muscular distress. In the treatment Group A, major significance of .01 occurred at the 72 hour measurement period. In the prevention Group B, it could not be precisely determined at which measurement period the significant difference was contained. However, there was some indication that it did occur at the 24 hour observation period.

Conclusions

Under the conditions and limitations of this study, the results permit the following conclusions:

1. Static stretching of a muscle immediately following unaccustomed repetitive exercise seems to provide some measure of prevention of delayed muscle soreness, for women between the ages of 20 and 35 years. With the Wilcoxon test significant at the .05 level, the static stretch technique diminishes the pain level at the 24 hour measurement period following the exercise. Post-exercise differences recorded during the 48 hour and 72 hour observation periods were not significant.

2. Static stretching of the exercised muscle following the development of delayed muscle soreness provides some degree of relief for women between the ages of 20 and

35 years. When the stretching is initiated 24 hours after exercise, the effect is a significantly ($p = .01$) lessened degree of delayed muscle soreness at the 72 hour measurement period, with some statistical indication that this effect has begun by the 48 hour measurement period.

Recommendations

The present study has revealed issues in this area requiring further research. It is recommended that future studies consider the following suggestions.

1. There is a need for study to explore various methodological ways of examining the subjective response to pain. A larger homogeneous sample divided equally into a control group and experimental group, may be a possible solution to the problems in design encountered in this study. In this way, the experimental exercise is done only once on each subject.

2. As reported in the discussion of results for the treatment section, further study is needed to determine how long it would take for the control subjects to attain the same pain free state as the experimental subjects.

3. There is a possibility that the menstrual cycle or the edema associated with birth control pills caused some female subjects to react differently to the experimental exercise. Therefore, it is recommended that the sample should be heterosexual to determine whether or not any physiological conditions of the female population influenced the results.

REFERENCES

Abraham, W. "Factors in delayed muscle soreness." *Medicine and Science in Sport*, 9: 11-20.

----------. "Exercise-induced muscle soreness." *The Physican and Sportsmedicine*, 7: 57-60. October, 1979.

Alberta Teachers Association. "Muscle soreness can be eliminated." *Health and Physical Education Council Bulletin*, 12: 29-31. December, 1974.

Asmussen, E. "Observations on experimental muscle soreness." *Acta Rheumatolica Scandinavia*, 2: 109-116, 1956.

Astrand, O. and K. Rodahl. *Textbook of Work Physiology*. New York: McGraw-Hill, 1977.

Beecher, H.K. *Measurement of subjective responses: quantitative effects of drugs*. New York: Oxford University Press, 1959.

de Vries, H.A. "Prevention of muscular distress after exercise." *Research Quarterly*, 32: 177-185, 1961a.

----------. "Electromyographic observations of the effects of static stretching upon muscle distress." *Research Quarterly*, 32: 468-479, 1961b.

----------. "Evaluation of static stretching procedures for improvement of flexibility." *Research Quarterly*, 33: 222, 1962.

----------. *Physiology of exercise for physical education and athletics*. Iowa: Wm. C. Brown, 1974.

----------. "Quantitative electromyographic investigation of the spasm theory of muscle pain." *American Journal of Physical Medicine*, 45: 119-134, 1966.

Dorpat, T. and T. Holmes. "Mechanisms of skeletal muscle pain and fatigue." *Archives of Neurology and Psychiatry*, 74: 628-640, 1955.

Elton, D., G. Burrows, G. Stanley. "Clinical measurement of pain." *The Medical Journal of Australia,* 1: 109-111, 1979.

Hilgard, E.R. "Pain as a puzzle for psychology and physiology." *American Journal of Psychology*, 24: 103, 1969.

Hill, A.V. "Mechanisms of voluntary muscle." *Lancet*, 2: 947-951, 1951.

Hough, T. "Ergographic studies on muscle soreness." *American Journal of Physiology*, 7: 76-81, 1902.

Jensen, C. and G. Fisher. *Scientific basis of athletic conditioning*. Philadelphia: Lea and Febiger, 1979.

Karpovich, P. *Physiology of muscular activity*. Philadelphia: W.B. Saunders, 1965.

Kierns, W. Personal Interview. Victoria, British Columbia, March 11, 1980.

Komi, P. and E. Buskirk. "The effects of eccentric and concentric muscle activity on tension and electrical activity of human muscle." *Ergonomics*, 15: 417-434, 1972.

Lamb, D. *Physiology of exercise: responses and adaptations.* New York: Macmillan Publishing, 1978.

Leavitt, F. et al. "Affective and sensory dimensions of back pain." *Pain 4*: 273-281, 1978.

----------. "Low back pain in patients with and without demonstrable organic disease." *Pain 6*: 191-200, 1979.

Melzack, R. "McGill Pain Questionniare: major properties and scoring methods." *Pain 1*: 277-299, 1975.

----------. *The puzzle of pain.* New York, New York: Penguin Books, 1977.

McGlynn, G., N. Laughlin, V. Rowe. "Effect of electromyographic feedback and static stretching on artificially induced muscle soreness." *American Journal of Physical Medicine*, 58: 139-148, 1979.

Rodbard, S. "Pain in contracting muscles." *Pain: Research and Treatment*, ed. B. Crue. New York: Academic Press, 1975.

Staton, W. "The influence of ascrobic acid in minimizing post-exercise muscle soreness in young men." *Research Quarterly*, 23: 356-360, 1952.

Talag, T. "Residual muscular soreness as influenced by concentric, eccentric and static contraction." *Research Quarterly*, 44: 458-469, 1973.

APPENDIX A

Experimental Groups Time Forms

Group: A__________ Name: ______________________________

	Time	Check if done
Experimental Exercise:	________	________
First Measurement:	________	________
Static Stretch:	________	________
	________	________
	________	________
Second Measurement:	________	________
Static Stretch:	________	________
	________	________
	________	________
Third Measurement:	________	________

Instructions for Static Stretch Procedure

1. Sit in a straight back chair.
2. Grasp a towel with both hands behind the chair (palms facing upward).
3. Move hands as close together as possible on the towel while maintaining the elbows in a locked position.
4. Gently lift arms upward as far as possible. An assistant must help by grasping the wrists and aiding in the movement.
5. Hold the stretch position for *two* minutes, then rest for one minute.
6. Repeat the sequence.

Group: B ____________ Name: ______________________

	Time	Check if done
Experimental Exercise:	______	______
Static Stretch:	______	______
	______	______
	______	______
	______	______
	______	______
First Measurement:	______	______
Second Measurement:	______	______
Third Measurement:	______	______

Instructions for Static Stretch Procedure

1. Sit in a straight back chair.
2. Grasp a towel with both hands behind the chair (palms facing upward).
3. Move hands as close together as possible on the towel while maintaining the elbows in a locked position.
4. Gently lift arms upward as far as possible. An assistant must help by grasping the wrists and aiding in the movement.
5. Hold the stretch position for *two* minutes, then rest for one minute.
6. Repeat the sequence.

APPENDIX B

McGill Pain Questionnaire

Name: ______________________________ Group ____________ Experimental/Control

What Does Your Pain Feel Like?

Instructions

Some of the words on this card describe your pain. We'll go over them group by group. Please choose the *one* word in the group which best describes your pain right now. If none of the words in the group describe your pain then just leave it out.

1	2	3	4
1. flickering	1. jumping	1. pricking	1. sharp
2. quivering	2. flashing	2. boring	2. cutting
3. pulsing	3. shooting	3. drilling	3. lacerating
4. throbbing		4. stabbing	
5. beating		5. lancinating	
6. pounding			

5	6	7	8
1. pinching	1. tugging	1. hot	1. tingling
2. pressing	2. pulling	2. burning	2. itchy
3. gnawing	3. wrenching	3. scalding	3. smarting
4. cramping		4. searing	4. stinging
5. crushing			

9	10	11	12
1. dull	1. tender	1. tiring	1. sickening
2. sore	2. taut	2. exhausting	2. suffocating
3. hurting	3. rasping		
4. aching	4. splitting		
5. heavy			

13	14	15	16
1. fearful	1. punishing	1. wretched	1. annoying
2. frightful	2. gruelling	2. blinding	2. troublesome
3. terrifying	3. cruel		3. miserable
	4. vicious		4. intense
	5. killing		5. unbearable

17	18	19	20
1. spreading	1. tight	1. cool	1. nagging
2. radiating	2. numb	2. cold	2. nauseating
3. penetrating	3. drawing	3. freezing	3. agonizing
4. piercing	4. squeezing		4. dreadful
	5. tearing		5. torturing

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THE EFFECTIVENESS OF STATIC STRETCHING FOR THE PREVENTION AND THE TREATMENT OF DELAYED MUSCLE SORENESS

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September 10, 1980
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