AN ABSTRACT OF THE THESIS OF

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Title: The Effect of Hamstring Temperature Reduction on Quadricep's Torque.

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Ice massage is commonly applied to muscles of athletes or physical therapy patients immediately prior to participation in various types of activity. This study was designed to determine the effect of cold application (ice massage) to the hamstring (leg flexors/antagonist) and the influence upon the contraction of the opposite non-chilled quadricep ( leg extensors/agonist).

Twelve male and twelve female subjects (mean = 22.5 yrs.) with no history of lower limb injury were tested on a Biodex® isokinetic dynamometer at velocities of 180, 360, and 450 degrees per second. The testing paradigm was consistent with isokinetic protocols.

Prior to administration of the pre-test, the subject's percent body fat (PBF) and the posterior thigh's skin fold thickness (PSK) at the treatment site was recorded. The subjects were randomly assigned a limb to be tested, treated by ten minutes of ice massage, and re-tested. The mean peak torque (PT) for five maximal contractions of the leg extensors and the mean range of motion (ROM) at which the PT occurred was determined for each velocity. Differences between PT and ROM for the non-chilled and chilled limbs were analyzed for each velocity using a

repeated measures ANOVA. Peak torque was greater for the cold treatment ($F(1,23) = 5.84$, $p < .03$). Further analyses using a mixed repeated measures ANOVA indicated that cold significantly increased PT for subjects with less adipose tissue at the treatment site (PSK) ($F(1,16) = 9.32$, $p < .008$) and less PBF ($F(1,22) = 12.9$, $p = .002$). The ROM at which the PT occurred for the quadricep transpired sooner into leg extension for all subjects than did the non-chilled hamstring and at each velocity ($F(1,23) = 7.3$, $p < .02$).

Further research is needed to investigate other application methods of cold and/or heat to the antagonist muscle and determine the effect(s) of these therapeutic applications upon the agonist's contraction as well as the ROM occurrence. Thus, further investigation may necessitate changes in therapeutic protocols.

# THE EFFECT OF HAMSTRING TEMPERATURE REDUCTION ON QUADRICEP'S TORQUE

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# I. THE PROBLEM AND RELATED RESEARCH

## Introduction and Problem Statement

Knight (1985) cites 98 research studies that demonstrate the physiological effects of cold upon nerve conduction velocity (NVC). Thus, cold affects NCV and also facilitates therapeutic procedures by reducing pain and influencing other sensory nerve aspects, especially inhibition of muscle spindle sensitivity. These influencing therapeutic factors are also well documented by Michlovitz, S. L. (1990) with 77 citations and Knight (1985) extended the research references into several hundred. Specifically, the enhancement of muscle stretching techniques (increasing range of motion) by way of cold's proficiency to reduce pain and proprioception sensitivity is very beneficial adjunct. The superiority of cold as a supplement may have been best demonstrated by Prentice (1982), Prentice and Kooima (1986) and by Wilkerson (1985).

A common therapeutic procedure is to apply ice massage to the hamstring area (posterior thigh) prior to stretching in order to improve range-of-motion (ROM). This technique (ice massage and stretching) is used frequently in latter rehabilitation protocols for hamstring injuries to specifically reduce the pain-

spasm cycle. Often, after icing and stretching, the athlete participates at various intensity levels of exercise.

Paradoxically, the therapeutic benefits caused by the inhibitory effects of cold may also interfer with the muscle's ability to protect itself from over-stretching. During movement, a limb's velocity will slow upon approaching the terminal range of motion (ROM) of the joint in which the contracting muscles are acting. This deceleration is a protective mechanism created by inhibition of the agonist (quadriceps) and activation of the antagonist (hamstrings) and is the sequel of normal muscular contraction (Basmajian, 1985; Marsden, 1983; Smith, 1981). In other words, the antagonist muscle's sensory components inhibits the agonist contraction and contracts itself to decelerate the limb. This antagonistic contraction and neural inhibition upon the agonist began as afferent feed-back, initiated and controlled by the antagonist's proprioceptors, other neural factors, or by mechanisms within the muscle (Grabiner and Hawthorne, 1990). Of importance to this study is: (a) the agonist does contract and the antagonist does initially relax, (b) the antagonist is being stretched as the agonist is contracting in the opposite direction and in order to protect itself from over-stretching and thus damaging itself, the antagonist starts inhibiting the agonist and also initiates self-contraction, (c) the agonist reaches a maximal contraction but the momentum of the limb continues, (d) the antagonist must now contract more forcefully in order to prevent excessive stretch and prevent the joint from extending beyond functional limits. However, if cold treatment to the antagonist reduced intramuscular temperature and precipitated a slower NVC, the delay of the protective response might be of consequence. Although this delay might enhance

maximal voluntary contraction (MVC) of the agonist, it may also inhibit initiation of the deceleration phase of movement. The slower responding deceleration might reposition the range of motion at which the MVC occurred and subsequently cause the agonist to place the joint's ROM into a contraindicated position. A position which might create excessive stretch to an already injured muscle.

The type of activity allowed after the clinical application of cold was applied to facilitate stretch and reduce pain is paramount.. Because as a limb's velocity increases, peak torque and joint position will normally change and NCV delays would become more influenced at higher velocities (Osternig, Hamill, Lander, & Robertson, 1986; Osternig, Hamill, Sawhill & Bates, 1983). Thus, the muscular temperature reduction affects upon the antagonist may be more pronounced at higher limb velocities, i.e., running versus walking, and consequently dampening the normal protective mechanisms and creating a greater detriment upon the antagonist.

There is limited research as to whether cold application influences contractile potentials of muscles under dynamic conditions, especially at high limb velocities. There is also no research as to whether cold will influence the antagonist and consequently alter the agonist's contraction or range of motion at which the greatest tension is produced (peak torque). Paramount clinical importance is whether a temperature reduction to the antagonist muscles inhibits (increases response time) and ensues a delay for the initiation of protective mechanisms that would decelerate limb movement and consequently over-stretch the antagonist by the contracting agonist. Thus, an inhibition (delay of the

protective mechanism) might be facilitating re-injury if the application of cold was applied as an adjunct for therapeutic stretching of a muscle that had been previously injured and then the patient engaged in a dynamic activity, i.e., running. In other words, does cold application to an antagonistic muscle group accentuate contraction of the agonist, allow greater MVC, and does this potentially greater contraction occur closer to the joint's terminal position?

Two additional questions arise. First, ice massage and other cold applications are also applied for numerous therapeutic rationale during all phases of rehabilitation. Considering the frequent usage of cold application within a clinical setting, it would not be uncommon to apply cold (planned or unplanned) prior to the isokinetic dynamometer being used to assess strength . In this situation cold application may cause erroneous strength measurements due to the inhibitor effects of the cold on nerve.

The second question is whether cold could supplement the pre-contraction phenomena shown by Caiozzo, Barnes, Prietto, & Mc Master (1981), Caiozzo, Laird, Chow, Prieto, and Mc Master (1982), and Grabiner & Hawthorne (1990)? These researchers demonstrated that a preceding contraction of the antagonist could inhibit neural mechanism, or some the inhibitor, and that this pre-contraction resulted in a significant tension increase, 25% improvement for the agonist. These authors encouraged <u>immediate</u> clinical application in isokinetic rehabilitation based upon their findings. Thus, greater MVC may be achieved with the "coupling" of the enhancement technique and cold application.

## Objective of the Study

There were two questions to be addressed in this study: (a) does cold influence the neural mechanisms that protect the antagonist from excessive stretch? and (b) does the application of cold to the antagonist enhance the contractile potential of the agonist? Thus, the focus of this study was to investigate the effect of cold application on an antagonist group's (hamstring) to determine subsequent changes in the contractile potentials (output) of the agonist (quadriceps) group. Specifically:

1) What effect will reducing intramuscular temperature have on the hamstring's performance and subsequent torque curves of the quadriceps?

2) What effect will three different velocities have on torque of the quadriceps and the range-of-motion at which peak torque occurs after the hamstrings are cooled?

3) What effect will skin-fold thickness at the treatment site have upon agonist peak torque and the range of motion at which it occurs?

4) What effect will the percentage of body fat have upon the treatment and subsequent peak torque changes of the quadriceps and the range of motion at which it occurs?

## Hypotheses

1) There is a significant difference between the agonist (quadriceps) peak torque and velocity and treatment of non-cooled and cooled antagonist (hamstring).

2) There is a significant range of motion difference for leg extension at which the peak torque occurs between cooled and non-treated extremities at the selected velocities.

3) There is a significant difference between peak torque and range of motion and cooled and non-treated extremities at the selected velocities for leaner subjects and those with less skin-fold thickness at the treatment area than subjects with higher percent of body fat or greater skin-fold thicknesses at the treatment site.

## Related Literature

### Isokinetic Instrumentation

The development of isokinetic rehabilitation devices in the late 1960s provided tools by which the contractile properties of muscles under conditions of constant velocity *in vivo* (dynamic) could be measured (Osternig, 1986). Isokinetic dynamometers contain either an electronic servo-motor or hydraulic valves as speed control mechanisms, which, theoretically, prevent acceleration of the limb irrespective of increases in applied force once a preset speed is attained.

> According to Perrine (1968), when an individual applies maximum effort to an isokinetic exercise device, it will instantly accelerate to its set speed and then by preventing any further acceleration above that speed, it will load the dynamically *harnessed* muscle exactly proportionate to its maximum dynamic tension capacity through a full range at that speed. As the muscle's tension capacity and skeletal advantage varies through the range of movement, the resistance caused by the speed-governing action of the device will fluctuate accordingly and naturally accommodate to the muscle's force transmitting capacity at every point in the range. In this way, the device consistently loads the muscles for maximum work accomplishment with each repetition and does so without over stressing it at any point.
> By determining an appropriate exercise speed and presetting it on the isokinetic exerciser (thereby fixing the shortening speed at which the muscle will be loaded), it is possible to allow a muscle to contract at the specific shortening speeds on the tension-velocity curve where it can develop either its (1) maximum peak tension, (2) most work per repetition, (3) highest power output, (4) some sub maximal average power output per repetition for a maximal time duration, or it can be loaded at a specific joint speed corresponding to some special physical activity (p. 43).

Reliability: Mechanical and Physiological

The mechanical reliability of isokinetic dynamometer systems has been found to be as high as 0.99 (Osternig, 1986). Mechanical reliability is determined by: (a) setting the isokinetic device at a selected velocity, (b) attaching a known weight to its lever arm, (c) releasing the arm, (d) allowing it to "free fall", (e) and determine whether the dynamometer record the known weight accurately. Timm (1990) obtained the same level of mechanical reliability for the Biodex® isokinetic dynamometer at lower limb velocities of 60, 120, 180, 240, 300, and 450 degrees per second.

In the same study, Timm, conducted a test-retest of thirty-six subjects (30 male and 6 female) with exactly 48 hours between the test-retest sessions and

showed a physiological reliability (at the knee) of 0.99 for peak torque. Feiring, Ellenbecker and Derscheid (1990) conducted a test-retest reliability investigation with seven days between tests. The intraclass correlation coefficients of 19 subjects were between .95 and 97 for knee extension peak torque means of 60, 180, 240 and 300 degrees per second. Mawdsley and Croft (1982) conducted test-retest sessions at 30 degrees per second with one minute rest between tests. There was no difference between the test-retest measurements on 20 subjects.

Johnson and Siegel (1978) tested 40 female volunteers to determine the physiological reliability of the isokinetic dynamometer. Six test trials were administered at 180 degrees per second on each of three consecutive days and each trial was separated by approximately 20 seconds. Reliability coefficients ranged from .93 to .99 and were affected more by testing over days than over trials tested on the same day. Only peak torque for leg extension was measured in this study with the range of motion from $90^0$ of flexion to $0^0$ of extension

Testing Order

Wyatt and Edwards (1981) tested 100 subjects on a test-retest design comparing contralateral differences of peak torque. The researchers divided the subjects into equal groups of male and females. The two subject groups were then subdivided; thus, the original male and female group of 50 were subdivided into groups of 25 subjects. The divided-gender group controlled the velocity testing sequence. One subdivided group (25 per gender group) was tested at an initial velocity of 300 degrees per second and the other at 60 degrees per second.

The intermediate speed of 180 (there were three velocities measured per subject) remained the same for each group. Testing sequence for velocity was not significant (p.> .01).

## Contractile Changes at Different Limb Velocities

### Force-velocity Relationship

Perrine and Edgerton (1978) studied force-velocity and power-velocity relationships at various isokinetic speeds. At the lower velocities all subjects exhibited less than 15% deviation from their maximal peak torque. With higher velocities (maximum of 360 degrees per second), the force potential decreased by fifty percent of the slower velocities. Barnes (1980), Osternig and Hamill (1983), Wyatt and Edwards (1981) and other investigators have also found that force decreases as velocity increases. Perrine (1978) suggested that neural regulation mechanisms may be limiting the potentials of these subjects and that such a significant reduction may be a safety factor.

### Coactivation and Deceleration

Basmajian, (1985), Osternig and Hamill (1983), Osternig and Sawhill (1983), and Smith (1981) have shown that toward the termination of an isokinetic repetition the limb decelerates as the joint limits are approached. These studies also demonstrated that as a limb's velocity increased, the role of the antagonist

became more influential, especially near termination of the range of motion of the joint. The increasing influence of the antagonist is probably attributed to reciprocal inhibition and antagonist coactivation which was first demonstrated by Sherrington in the early 1900s. However, Smith (1981) has elaborated the conditions that favor reciprocal inhibition and/or coactivation antagonist as follows:

> Antagonist coactivation occurs most often in the following circumstances:
> (1) When muscular tension or limb position requires precise monitoring without load, the antagonists co-contract. This would occur in the initial phase of learning a new motor skill.
> (2) In high velocity limb displacements or under loaded conditions, the antagonist muscles after a short lull will contract strongly to decelerate the limb.
> (3) Isometric prehension of the hand in either precision or a power grip will require antagonist co-contraction to stabilize the wrist. This will add stiffness to the carpal, metacarpal, and phalangeal joints. (p. 736)

Basmajian (1985) summarized the findings of studies that investigated agonist/antagonistic relationships with respect to higher velocity limb displacements:

> All have shown that during rapid movement, the activity of both agonist and antagonist muscles displayed a triphasic pattern:
> (1) An initial burst of agonist activity with the antagonist silent (limb acceleration),
> (2) Next there is a reduction of agonist activity with burst of activity in the antagonist (limb deceleration),
> (3) Finally, there is a subsequent resumption of agonist and antagonist activity. (p. 225)

The accepted explanation for this triphasic sequence is that the nervous system attempts to prevent damage to the joint and the antagonist, which would result from the explosive force being generated by the agonist during ballistic contraction, by dampening limb acceleration with antagonistic activity

(Basmajian, 1985). Hallet (1975) noted that, in ballistic movements, the triphasic pattern originated from purely pre-programmed supraspinal signals with little influence from the peripheral sensory units (proprioceptors). Conversely, Basmajian (1985) citing Angel's (1977) study stated that "he did not support the notion of complete pre-programming of the agonist-antagonist triphasic pattern of activation and that response signals from the periphery provided contraction emphasis" (p. 232). Basmajian concludes "that both spinal and supraspinal control mechanisms are necessary in regulating agonist-antagonist functions" (p. 232).

In ballistic movements, the mechanism controlling limb deceleration is the phasic portion of the muscle spindle that inhibits the initial contraction of the antagonist but excites the antagonist toward the end of the joint's ROM. Thus, the muscle spindle and/or other proprioceptive receptors provide the necessary feedback information to excite the alpha motor neurons of the antagonists to initiate their contraction and prevent limb displacement (Gowitzke, 1980).

## Temperature Reduction

### Nerve Conduction Velocity

There is considerable evidence that tissue temperature reduction will affect nerve conduction velocity (Cote, 1979; Foldes, 1978; Knight, 1985; Lee, 1978; Lowdon & Moore, 1975). Wolf, Ledbetter & Basmajian (1976) demonstrated an inhibitory effect upon motor unit activity with cooling within the first minutes of

cold application. Petajan and Watts (1962) demonstrated a decrease in the stretch reflex response after cold treatment when measuring half refraction time, amplitude and total reflex time. They noted that smaller diameter nerve responses were decreased to a greater degree than those in larger diameter nerves. Mense (1978) studied the effects of warming and cooling on muscle spindle afferent fibers and demonstrated that warming enhanced afferent discharge and that cooling depressed the muscle spindle's afferent discharge. Mansfield (1981) showed that the dynamic component of the Achilles stretch reflex was diminished after the triceps surae was cooled.

Linear Relationship

Lowitzch, Hopf and Galland (1977) showed a quadratic relationship between cooling and nerve conduction velocity (NCV). As the intramuscular temperature decreased, the sensory NCV linearly decreased; yet the sensory decrease became more pronounced (progressed faster) as the temperature declined. Halar, De Lisa & Brozovich (1980) and Halar & De Lisa (1981) showed a correlation between skin temperature reduction and nerve conduction velocities - each Celsius degree change in tissue temperature resulted in a 1.7 to 1.9 meter per second alteration of NCV. Gassel and Trojaborg (1984) reported similar results for the sciatic nerve in the lower extremity.

Depth of Cooling

Waylonis (1967) measured the intramuscular temperature (IMT) of thighs (posterior) that had been ice massaged for either five or ten minutes. For the ten minute application, IMT was reduced by 12.5 degrees Celsius at 0.5 centimeters (cm) of depth, 11 degrees at 1 cm, 5.2 degrees at 2 cm, 1.4 degrees at 3 cm, and 0.1 degree Celsius decrease at 4 cm of tissue depth. Wolf & Basmajian (1973) inserted into the posterior thigh a 29 gauge needle thermistor, to a depth of 4.3 centimeters, in order to measure intramuscular temperature reduction of an area that had been exposed to a cold-plate. The contact area had been cooled to approximately 10 degrees Celsius. The recorded (intramuscular temperature) reduction ranged from 0.4 to 1.9 degrees with a mean of 1.2 degrees Celsius. Intramuscular temperature reduction showed a steady decline, particularly after the first minute of cooling, with a significant intramuscular difference noted within five minutes of cold application. Moore (1972) reported a decrease of $16.38^0$ C at a depth of 2 centimeters. Lowden and Moore (1975) reported that the first five minutes showed the most significant intramuscular temperature reduction, recorded at a depth of 2 cm (biceps brachii). After five minutes and with additional ice massage for ten and fifteen minute, there was no subsequent temperature reduction. Bugaj (1975) reported that analgesia at the calf was elicited after ten minutes of ice massage. This indicated that NVC had been significantly impaired.

Cooling and Subcutaneous Fat

Tissues that have a higher water content, such as muscle, have better conductivity than does adipose tissue. Adipose tissue acts as an insulator and provides resistance to heat flow (Michlovitz, 1990). Research demonstrates that the greatest cooling occurs in muscular subjects that have little subcutaneous adipose tissue overlying the muscles being cooled (Knowal, 1983; Waylonis, 1967; Wolf & Basmajian, 1973). Lowden and Moore (1977) reported an inverse relationship between skin-fold thickness and temperature reduction. The amount of fat may influence the degree and rate at which muscle can be cooled ( Lehmann & De Lateur, 1990).

## Summary of Literature and Proposed Study

Research indicates that cold inhibits nerve conduction velocity and that there is an inverse relationship between the effectiveness of cold upon conduction velocities and the amount of subcutaneous fat of subjects. Also knowing that reciprocal inhibition and co-activation exist, or that during normal locomotion when the quadricep (agonist) is contracting the hamstrings (antagonist) must initially relax. Thus, the quadricep is permited to develop tension and not be counteracted by the hamstring (antagonist). However, once the quadricep has developed required tension to assure movement, a requisite method to reduce the quadricep's tension as well as decelerate the momentum of the limb created by the contracting quadricep. If these responses did not occur, the momentum and

uncontrolled tension development would over-stretch the hamstring and compromise the joint being acted upon (knee would be over-extended).

There were two studies which demonstrated that manipulation of the antagonist (pre-contraction of the antagonist) can change the tension production of the agonist during a dynamic contraction. These studies moderated a pre-contraction of the antagonist immediately prior to contracting the agonist and thus effected the normal neural/ or another predominance and subsequently changed normal reciprocal inhibition and coactivation of the antagonist. This allowed a significantly greater torque production by the agonist. However, there is no research indicating that the usage of cold to the antagonist will also change the tension (torque) production of the agonist muscle group.

This study investigates the effect of cold application on the antagonist to determine whether there are contractile and range of motion changes to the agonist (quadriceps) at three different isokinetic velocities. Thus, the conclusion will help determine whether clinical application of ice massage to a muscle group, often used as an adjunct to stretching routines, may have a detrimental effect (over-stretching) upon the muscle being treated if there is activity immediately after the cold application and stretching. If there is an effect, an additional question is whether cold's influence is greater for patients that have less body fat or have little adipose tissue at the treatment site. Subcutaneous adipose tissue is an insulator. For this study it is also important to determine whether any significance maximal tension increase of the agonist's also accompanies a change in the range of motion in which the torque occurs. Or in other words, does the increase in the peak torque resulting in the application of cold to the antagonist

also subsequently change the range of motion of this maximal contraction? And will this change, over-stretch the treated muscle?

# II. METHODS AND PROCEDURES

## Subject Information

Twelve males and 12 females volunteered for this study. The subjects ranged in age from 18 to 35 years (mean = 22.5 yrs.). These volunteers were solicited from upper division.Willamette University Physical Education courses. Appendix C provides the population demographics, means, standard deviations., body fat percentage, and limb tested. An injury history was gathered for each subject to determine whether any significant impairment to the lower extremity existed that would inhibit maximal effort on the isokinetic dynamometer. No subjects indicated or demonstrated any lower extremity impairments. All subjects were variously engaged in distance running, triathlons, baseball, or regular fitness programs.

## Body Fat

Each subject had a body fat assessment using skin-fold calipers. Three measured skin-fold sites were used to determine body density as described by Jackson and Pollock (1978) for males and Jackson, Pollock and Ward (1980) for females. The Siri formula, as described by Lohman (1984) was then used to calculate body fat percentages. The mean body fat percentage for male subjects was 10.671% (SD = +/- 6.13%) and 19.54% (SD = +/- 2.60%) for the female subjects. The subjects' weight ranged from 98 to 241 pounds (both genders).

Fat is an insulator which retards the reduction in intramuscular temperature (Lowden & Moore, 1975; Lowden & Moore, 1977; Wolf & Basmajian, 1973). Enhanced cooling of the hamstrings can be accomplished by ice massage if the adipose tissue is minimal; greater IMT reduction will progressively delay NCV. Thus, an intramuscular temperature reduction in the hamstrings and subsequent delay of NCV, will delay the hamstring's deceleration response and affect the contractile potentials of the agonist.

## Posterior Thigh Skin-Fold (Treatment Area)

Each subject had an additional recorded skin-fold measurement of the posterior thigh. This skin-fold site, mid-portion of the posterior thigh (with the knee at 90 degrees and lower extremity relaxed when measured), did not determine body density. The measurement determined the skin-fold thickness for the area that would receive the treatment. The posterior thigh measurements ranged from 6 mm to 28 mm. of skin fold thickness, with a mean of 16.88 mm.

## Testing Procedure

Subjects had no previous isokinetic testing and each subject received instruction in the use of the isokinetic testing instrument. Each subject received information as to the study's purpose and, prior to any testing, an informed consent was obtained (Appendix A). The testing instrument, a Biodex® with version 2.0 software, was located at the Rebound Clinic, Albany, Oregon.

After the instrumentation was explained, skin-fold measurements, age, and weight were recorded; subsequently, the testing protocol was explained and the extremity to be treated was randomly determined (10 rights and 13 lefts). Each subject was positioned according to Biodex® (1990) specifications (Appendix B).

Testing consisted of two or three submaximal warm-ups and two-to-four (2-4) maximal unrecorded efforts followed by five measured repetitions of knee extension and flexion. This procedure is consistent with standard isokinetic testing protocol and was required by the Biodex® software to ensure stable measurements (Feiring, Ellenbecker, & Derscheid, 1990; Johnson & Siegel, 1978; Mawdsley & Croft, 1982; Mawdsley & Knapik, 1982; Osternig, 1986; Sawhill, Bates, Osternig, & Hamill, 1982; Timm, 1990). This procedure was followed for all testing conditions.

The velocity values used were selected because antagonist contraction effecting deceleration is more dramatic at higher limb velocities (Grabiner & Hawthorne, 1990; Marsden, Obeso, & Rothwell,1981; Osternig, Hamill, Lander, & Robertson, 1986) In addition, nerve conduction velocities (NVC) are extremely fast. Relevant to this study are the tibial nerve at 37-59 meters/second and sensory nerves at 70-120 meters/second (Basmajian & De Luca, 1985; Rothstein, Roy, & Wolf, 1991). Thus, a NCV delay effected by cold would not be as noticeable at slower limb displacement velocities. For example, the angular velocity of the leg rotating in a vertical plane about a horizontal axis through the knee is about 700 degrees per second as the skilled runner swings the leg forward. Thus, *in vivo* limbs are capable of high velocity and the purpose of this study was to approximate *in vivo* application. Higher velocities were used because 180

degrees is relative to walking, 360 degrees for a slow jogging cadence, and a maximal velocity of 450 degrees per second because the Biodex® limitation is at this velocity (Cooper, 1976; Gotwitzke, 1980; Wyatt & Edwards, 1981).

The research design of this study did not include counter balancing of velocity testing order to control carry-over effects as sequence order has not been found to significantly influence results in isokinetic dynamometer studies (Wyatt & Edwards, 1981).

The procedure of practicing submaximal and maximal contractions before obtaining the recorded measurements at each velocity was designed to reduce any practice effect from a previous speed. This is standard isokinetic testing protocol and assures high test-retest reliability. In addition, a practice effect would be additive and noted because of its progressive pattern (Winer, Brown, & Michels, 1991). Moreover, the difference between test velocities is so great that practice at one velocity does not carry-over to the next tested velocity (Timm, 1987). It was concluded that altering testing sequence would change the protocol established for the instrumentation.

The pretest had a minimum of one and a maximum of two minutes rest between tested velocities. Upon completion, the subject was removed from the isokinetic dynamometer and ice massage was administered to the entire hamstring group (posterior thigh compartment) for ten minutes. Cooling occurred while the subject was prone and relaxed upon a treatment table.

Immediately after the ice application, the subject performed the posttest using the same procedure as the pretest. The subject also had a cold pack placed between the cooled area (posterior thigh) and the isokinetic dynamometer seat.

This helped to retain the cooling effect during the testing procedure; the "cold pack" was at ambient temperature during the pretest.

Instructions, preliminary measurements, pretesting, cold treatment, and retesting took less than one hour. Subjects were either driving or being driven to Albany from Salem prior to testing.

## Data Analysis

An analysis of variance (ANOVA) software program by Clear Lake Research (1990), version 2.0, was used to analyze the data. All data were recorded and formatted on Microsoft Excel, Version 3.0, (1991). Computed simple main effects used a pooled error term and the computed degrees of freedom used Saiterwaite's approximation (Keppel 1973; Winer, Brown, & Michels, 1991). Appendix C presents the demographics of the subjects and the raw data. Appendix D presents the raw data for peak torque and range-of motion (ROM).

## Pre and Post Peak Torque Analysis Design

The isokinetic dynamometer produced a computer analysis for the dependent variable torque values (foot-pounds) for leg extension,. The independent variables were (a) treatment (no cold and cold) and (b) velocity (three levels: 180, 360, and 450 degrees per second). A computer video-graph, indicating torque measurements for each set of contractions (five extensions and

five flexions), allowed precise measurement of each peak torque by permitting pointer-positioning with a value read-out (at the pointer location) on the graph. The ANOVA software program analyzed an obtained mean for four of the five contractions. The initial contraction provided inadequate graphical assessment because the entire torque curve was not graphically presented and thus the pointer could not adequately assess the curve for many subjects. A complete within-subjects 2 x 3 factorial design (Figure 1) was used to analyze the data.

Figure 1. Complete Within-Subjects Two Factor Analysis: Treatments X Velocity

| NO COLD | | | COLD | | |
|---|---|---|---|---|---|
| 450 | 360 | 180 | 450 | 360 | 180 |
| DEGREES PER SECOND | | | DEGREES PER SECOND | | |

Treatment Area Thickness Influence

Nine subjects had a skin fold measurement (SKF) at the posterior thigh site of equal to or greater than nineteen millimeters (19) and nine subjects had a SKF equal to or less than fifteen (15) millimeters. The other subjects (6) had posterior thigh SKF between 16 to 18 millimeters (one at 16, one at 17, and four at 18 mm). Because a mixed design analysis of variance (Figure 2) was used to determine if a significant difference existed between the amount of subcutaneous adipose tissue (at the cold treatment site) and peak torque, a balanced analysis of nine subjects per

posterior SKF level (adipose criteria) was performed. Thus, amount of adipose tissue (=<15 and =>19 mm) was treated as a between-subjects variable, whereas treatment (no cold or cold) and the three levels of velocity were treated as within-subject variables.

Figure 2. Influence of Posterior Thigh Skin-fold Thickness and Treatment: Effects Upon Peak Torque - A Mixed Analysis Design

| ADIPOSE TISSUE | | | |
|---|---|---|---|
| EQUAL/LESS THAN 15mm | | EQUAL/GREATER THAN 19mm | |
| NO COLD | COLD | NO COLD | COLD |
| 450 360 180 | 450 360 180 | 450 360 180 | 450 360 180 |
| VELOCITY | | VELOCITY | |

Percent Body Fat Influence Upon Treatment

Twelve subjects had a body fat percentage less than 16% and an equal number of subjects had greater than 16%. Appendix C provides specific data pertaining to the subjects percent body fat. A mixed between-subjects design, identical to Figure 2, determined whether a significant difference existed between percent body fat and peak torque. Percent body fat (<16%, >16%) was treated as a between-subjects variable, whereas treatment (no cold or cold) and velocity levels were treated as within-subjects variables.

## Pre and Post ROM Change

The last analysis, an identical within-subjects design of a 2 x 3 factorial (see Figure 1) determined whether significant ROM mean differences occurred for peak torques and treatments at the three velocity levels. The independent variables were (a) no cold and cold, and (b) the three velocity levels. The dependent variable was the mean ROM at which peak torque occurred during extension. Peak torque has been shown to occur at different extension ROMs as velocity increases (Osternig, 1975; Osternig, 1986; Sawhill, et al., 1982). An alpha level of .05 was used for all analyses..

# III. RESULTS

The purpose of this study was to investigate the effects of cooling leg flexors on changes in the contractile potentials of the leg extensors.

## Treatment Effect to Hamstrings and Quadricep's Peak Torque

Table 1 presents the summary for a complète within-subjects, 2 x 3 factorial on 24 subjects. Torque was greater for the cold treatment (mean = 88.4 foot pounds) than for the no cold treatment (mean = 86.0 foot pounds) conditions ($F(1, 23) = 5.854$, $p < .03$). Torque means also varied with velocity levels, averaging 106.4, 79.6, and 75.7 for the 180, 360, and 450 degrees per second velocities, respectively ($F(2, 46) = 102.4$, $p < .0001$). However, any analysis comparing levels of velocity will be significantly different because the torque values that subjects are able to produce will change as the preset velocity is switched. The relative differences that occur are the result of the force-velocity principle and occur throughout the evaluations for this study. Thus, the peak torque differences between velocities are expected and in accord with the force-velocity principle of muscular contraction or that force is inversely proportional to velocity.

Table 1

ANOVA Summary of Peak Torque

| SOURCE of VARIATION | DF | SUM OF SQUARES | MEAN SQUARE | F | p |
|---|---|---|---|---|---|
| subjects | 23 | 134021.6 | 5827.03 | | |
| treatment (A) | 1 | 212.5 | 212.5 | 5.854 | .0239 |
| error | 23 | 834.9 | 36.3 | | |
| velocity (B) | 2 | 26755.7 | 13377.9 | 102.38 | .00001 |
| error | 46 | 6010.6 | 130.7 | | |
| A x B | 2 | 107.5 | 53.8 | 2.951 | .0623 |
| error | 46 | 838.2 | 18.2 | | |

An investigation of Figure 3, suggests that cold versus no cold treatment leads to similar torque at 450 and at the intermediate velocity of 360 almost equal torque between treatments, but at 180 degrees per second, cold leads to greater torque. This interpretation was verified through an analysis of simple effects, which showed that the 180 velocity was the source of the interaction ($F(1, 23) = 7.3$, $p < .02$) (See Table 2).

Figure 3. Graph of Treatment, Velocity, and Peak Torque

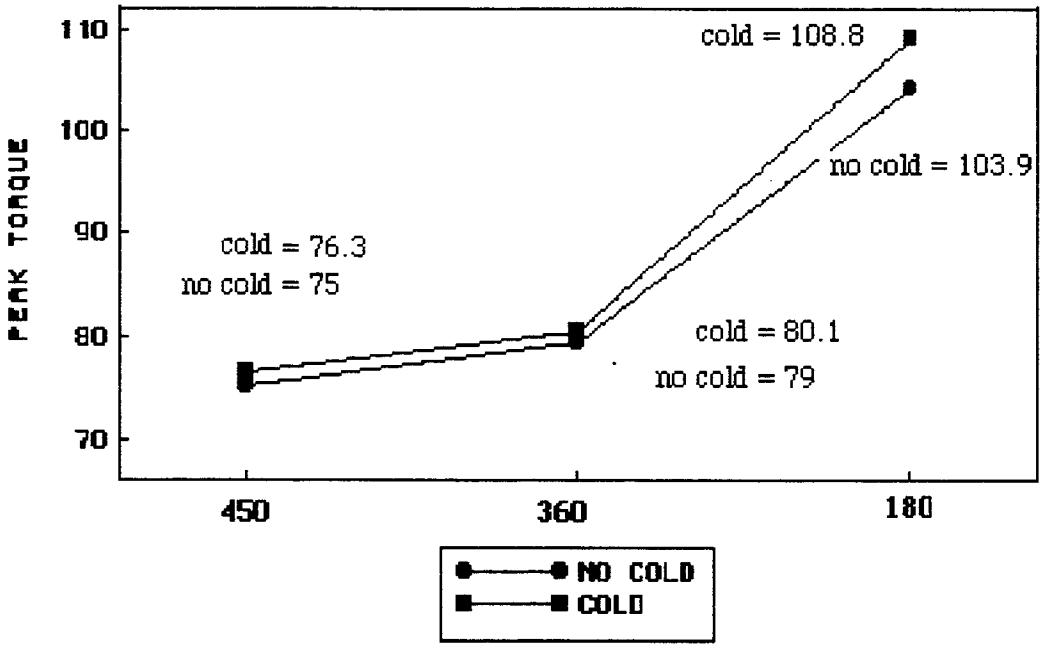


Table 2

Simple Effects of Treatment on Peak Torque

| EFFECT | MS | DF | DFe | MSe | F | p |
|---|---|---|---|---|---|---|
| Treatment @ 450 | 21.59 | 1 | 23 | 19.80 | 1.09 | .307 |
| Treatment @ 360 | 13.95 | 1 | 23 | 14.16 | .99 | .331 |
| Treatment @ 180 | 284.49 | 1 | 23 | 38.78 | 7.34 | .013 |
| Velocity @ No Cold | 5881.49 | 2 | 46 | 70.06 | 83.95 | .0001 |
| Velocity @ Cold | 755.14 | 2 | 46 | 78.83 | 95.78 | .0001 |

Cold and Treatment Area Thickness Affects to Peak Torque

The next series of Tables (3 and 4) and Figure 4 represent a mixed design, 2 x 2 x 3 factorial. This was conducted to determine whether a significant difference in peak torque existed between treatments for subject groups that differed by the amount of subcutaneous adipose tissue at the treatment site. The posterior skin-fold thickness factor had two levels (a) less than or equal to fifteen (15) millimeters and (b) greater than or equal to nineteen (19) millimeters of thickness. As with previous analyses, peak torque was measured over three velocity levels, of 450, 360, or 180 degrees per second for each treatment level.

As expected given the previous ANOVA, the main effects for treatment ($F(1,16) = 10.03$, $p < .006$) and velocity ($F(2,32) = 82.02$, $p < .0001$) were significant. The skinfold factor was significant, with a greater torque mean shown by the ≤ 15 mm group (posttest =115.74 foot-pounds) than ≥ 19mm group (posttest = 75.38) groups ($F(1,16) = 9.32$, $p < .0076$) (see Tables 3 & 4). Means for skin-fold thickness and peak torque at the three velocities are presented in Table 4. The ≥ 19mm group had a 0.21 foot-pound, 1.48 foot-pound, and 2.83 foot-pound differences at 450, 360, and 180 degrees per second, respectively; whereas, leaner subjects had treatment means of 4.62, 3.47, and 7.70 foot-pound differences for the peak torque mean of 450, 360, and 180 degrees per second, respectively. No interactions were significant.

Table 3

ANOVA Summary of Posterior Skin Fold Thickness to Peak Torque

| SOURCE of VARIATION | DF | SUM of SQUARES | MEAN SQUARES | F | p |
|---|---|---|---|---|---|
| SKF (A) | 1 | 39982.0 | 39982.0 | 9.32 | .0076 |
| error | 16 | 68637.5 | 4289.8 | | |
| treatment (B) | 1 | 309.6 | 309.6 | 10.03 | .0060 |
| A x B | 1 | 95.1 | 95.1 | 3.08 | .0983 |
| error | 16 | 493.9 | 30.9 | | |
| velocity (C) | 2 | 22924.4 | 11462.2 | 82.02 | .0001 |
| A x C | 2 | 647.9 | 323.93 | 2.32 | .1148 |
| error | 32 | 4471.7 | 139.7 | | |
| B x C | 2 | 47.7 | 23.8 | 1.33 | .2801 |
| A x B x C | 2 | 10.8 | 5.4 | .30 | .7440 |
| error | 32 | 576.2 | 18.0 | | |

Table 4

Mean Summary for Posterior Thigh Skin-Folds

| SKF | TREATMENT | VELOCITY | MEAN |
|---|---|---|---|
| ≤ 15mm | no cold | 450 | 95.26 |
| | | 360 | 103.45 |
| | | 180 | 132.71 |
| | cold | 450 | 99.89 |
| | | 360 | 106.92 |
| | | 180 | 140.41 |
| ≥ 19mm | no cold | 450 | 64.92 |
| | | 360 | 66.09 |
| | | 180 | 90.60 |
| | cold | 450 | 65.14 |
| | | 360 | 67.57 |
| | | 180 | 93.44 |

## Peak Torque to Body Fat

Table 5 presents a mixed 2x2x3 factorial, within-subjects analysis with 24 subjects. The percent total body fat for the two groups was (a) less than, or (b) greater than 16 percent. Treatment and levels were as previously discussed.

Fat percentage and velocity had a significant interaction. This interaction was ordinal but only at the velocities of 450 and 360 degrees per second. There was no interaction at 360 and 180 degrees per second. There was also a significant ordinal interaction ($F(1,22) = 5.82$, $p < .025$), see Table 5, for fat percentage and treatment with the leaner group increasing in peak torqure for the cold treatment but the greater than 16 % group made no progress between treatments. Torque did not depend on cold treatment for the > 16% group, but for the < 16% group torque is greater for the cold treatment. Simple effects analysis (see Table 6) supports this interpretation, as does plotting, showing an effect of treatment only for the < 16% group ($F(1,22) = 12.9$, $p < .002$). The > 16% group had a 0.23 foot pound difference between treatments; whereas, the < 16% group had a 4.64 foot pound increase for the cold treatment.

There was a significant difference ($F(1,22) = 7.43$, $p < .02$) between the two adipose groups The difference between groups was expected because of planned design. Treatment was also significant ($F(1,22) = 7.08$, $p < .02$) but as previously discussed, see simple analysis Table 6, only for the < 16% group. Velocity was significant, ($F(2,22) = 103.7$, $p < 00001$), as expected given previous analyses.

Table 7 presents mean torques used in a mixed 2 x 2 x 3 factorial, within-subjects analysis with 24 subjects. The percent total body fat for the two groups was (a) less than, or (b) greater than 16 percent.

Table 5

ANOVA Summary of Mixed Design for Adipose Percentage

| SOURCE of VARIATION | DF | SUM of SQUARES | MEAN SQUARES | F | p |
|---|---|---|---|---|---|
| Fat % (A) | 1 | 33830.3 | 33830.3 | 7.43 | .0123 |
| error | 22 | 100191.2 | 4554.1 | | |
| Treatment (B) | 1 | 212.5 | 212.5 | 7.08 | .0143 |
| A x B | 1 | 174.6 | 174.6 | 5.82 | .0246 |
| error | 22 | 660.3 | 30.0 | | |
| Velocity (C) | 2 | 26755.7 | 13377.9 | 103.72 | .0001 |
| A x C | 2 | 335.6 | 167.8 | 1.30 | .2825 |
| error | 44 | 5674.9 | 129.0 | | |
| B x C | 2 | 107.5 | 53.8 | 3.03 | .0586 |
| A x B x C | 2 | 56.8 | 28.4 | 1.60 | .2134 |
| error | 44 | 781.4 | 17.8 | | |

Table 6

Simple Effects of Peak Torque to Percent Fat & Treatments

| EFFECT | MSn | DFn | DFe | MSe | F | p |
|---|---|---|---|---|---|---|
| Percent fat @ no cold | 14571.9 | 1 | 22 | 2292.1 | 6.357 | .019 |
| Percent fat @ cold | 19433.1 | 1 | 22 | 2292.1 | 8.478 | .008 |
| Treatments @ <16% | 386.2 | 1 | 22 | 30.0 | 12.868 | .002 |
| Treatments @ >16% | .9 | 1 | 22 | 30.0 | .031 | .862 |

Table 7

Peak Torque Means to Percent of Body Fat: Three Velocity Levels

| FAT % | VELOCITY | NO COLD | COLD | DIFFERENCE |
|---|---|---|---|---|
| <16% | 450 | 86.79 | 91.53 | 4.74 |
| | 360 | 94.07 | 95.61 | 1.54 |
| | 180 | 119.78 | 127.4 | 7.62 |
| >16% | 450 | 63.18 | 61.13 | -2.054 |
| | 360 | 64.06 | 64.67 | 0.613 |
| | 180 | 88.04 | 90.16 | 2.12 |

Effect of Cold to Quadricep's ROM

Table 8 presents a complete within-subject's 2 x 3 factorial analysis on all (24) subject's ROM means at which peak torque occurred for (a) non-treated and (b) cold-treated extremities over the three levels of velocity. Cold treatment was significant, ($F(1, 23) = 7.3$, $p < .02$). The mean summary presented in Table 9 indicates that peak torque occurred earlier in leg extension (overall mean = 53.6 degrees) following cold than occurred in non-treated (overall mean = 50.7 degrees) limb. Full extension is at 0 degrees. There were no interaction effects.

Table 8

ANOVA Summary of ROM at Peak Torque

| SOURCE of VARIATION | DF | SUM of SQUARES | MEAN SQUARE | F | p |
|---|---|---|---|---|---|
| Subjects | 23 | 6981.73 | 303.55 | | |
| Treatment (A) | 1 | 299.72 | 299.72 | 7.31 | .0127 |
| error | 23 | 942.79 | 40.99 | | |
| Velocity(B) | 2 | 8361.99 | 4180.10 | 28.40 | .0001 |
| error | 46 | 6771.97 | 147.22 | | |
| A x B | 2 | 29.84 | 14.92 | .42 | .6585 |
| error | 46 | 1625.10 | 35.35 | | |

Table 9

Means for ROM for Treatment and Velocity

| TREATMENT | VELOCITY | DEGREES |
|---|---|---|
| no cold | 450 | 39.78 |
| | 360 | 54.73 |
| | 180 | 57.61 |
| cold | 450 | 43.42 |
| | 360 | 56.33 |
| | 180 | 61.03 |

# IV. DISCUSSION AND SUMMARY OF RESULTS

## Discussion

The objective of this study was to determine whether an application of cold to an antagonist muscle group (hamstrings) would influence an agonist muscle group (quadriceps). Clinically, cold applications to a body part have been shown to be effective in pain reduction, in creating a reduction of intramuscular temperature, in decreasing nerve conduction velocity (NVC), and in the rate of firingof motor units. These cold effects are of therapeutic benefit but may also mask certain protective mechanisms. Thus, icing a muscle group prior to an athletic event to facilitate a therapeutic procedure and/or reduce pain may distort the muscle's ability to protect itself from being over-stretched during the contraction of the opposite muscle group.

Specifically for this study, was the concern that the cold may lower the sensitivity of the muscle spindles. These protective mechanisms (muscle spindles) protect a muscle from over-stretching by causing the muscle being stretched to progressively increase contractile tension as well as decrease contraction (inhibition) of the opposite (agonist) muscles. If cold reduces the effectiveness of the chilled muscle's protective mechanism, it might increase contractile potentials of the opposite (agonist) unchilled muscle group and also the range of motion (ROM) at which this maximal agonist contraction would occur. Thus, the chilled muscle group might not progressively contract itself and not inhibit the contracting muscle until it is farther into the opposite direction and consequently over-stretched. This

would be contraindicated for an injured muscle and/or provide erroneous evaluation measurements if using an isokinetic dynamometer.

Therefore, the study was specifically designed to determine whether an application of cold to the antagonist would influence the agonist muscle(s) during a dynamic contraction and at relatively high limb velocities. The tested speed anticipated what a limb would encounter during an activity from a slow walk to a light jog. The speeds were 180, 360, and 450 degrees per second of limb movement at the knee. The contraction of the quadriceps was recorded as peak torque by an isokinetic dynamometer. The ROM at which this maximal output occurred was also recorded.

This study found that a significant difference, at the .05 level of significance, existed for the peak torque and ROM between treatments at the three different limb velocities. The cold treatment to the hamstring caused the subjects' quadriceps to produce a stronger contraction. This maximal contraction also occurred farther from complete leg extension and was reverse of the subjects' non-chilled hamstrings. Thus, when the hamstrings were not chilled by ten minutes of ice massage the quadricep's maximal torque was less and the ROM was closer to terminal knee extension.

Treatment significance upon peak torque increased when the subjects were separated into groups according to fat percentage and skin fold thickness at the treatment site (ice massage application area). The subjects who had less overall percent body fat (<16%) and were leaner (less fat between the skin and muscle) at the treatment site (posterior thigh) produced a significantly greater quadricep's contraction when the opposite muscles (antagonists) were chilled. The higher

overall percent body fat subjects also increased in peak torque but the difference was not as dramatic. There was no ROM difference between groups and all had the ROM occur earlier into extension after icing the hamstrings.

## Cold Effect Upon Peak Torque and Velocity

Cold treatment to the antagonist increased the peak torque of the agonist. Thus, intramuscular temperature reduction of the hamstrings did influence the quadriceps. The average peak torque increase over all velocities was 2.4 foot pounds. Further comparison of the treatment effects, at each velocity level, indicated that cold did not significantly influence peak torque except at the initial velocity level (180 degrees per second), $p = .013$. The initial velocity demonstrated a 4.9 foot pound increase. The other velocities of 360 and 450 degrees per second increased only 1.3 and 1.1 foot pounds, respectively.

There may be several reasons for significance, in a complete analysis design, only occurring at the initial velocity for all subjects (twenty-four). One explanation is a testing order effect. The velocity testing sequence was 180 initially, 360 intermediate, and 450 degrees per second for the last speed tested. Thus, there may have been more effort and less fatigue in the initial velocity setting and a learning effect that carried-over into the second testing session, the cold application. However, the testing procedure (see Appendix B) is standard protocol and research indicates that testing sessions on the same day are more reliable than over different days, and testing order (fast to slow or the reverse) will not confound

the results (Feiring, Ellenbecker, & Derscheid, 1990; Mawdsley & Knapik, 1982; Timm, 1990; Johnson, & Siegel, 1978, and Wyatt & Edwards, 1981).

The isokinetic dynamometer will always indicate greater torque production at a slower velocity than a higher velocity. This fact (a slower velocity produces higher peak torques) will cause the peak torques at each velocity level to be significantly different throughout this study. Thus, greater variation between the treatment velocities will exist. Therefore, greater potential for improvement at the initial speed and change may enhance the treatment variance.

There may also be a gender factor confounding the results. There was an equal number of male and female subjects. Further analysis and discussion will indicate that subjects separated contingent upon adipose tissue thickness at the treatment site and higher percent body fat produced different peak torque means for treatments at each velocity level. Females, almost exclusively, fell within the higher percent fat and skinfold thickness categories. The female grouping tendencies may also have had a significant influence upon intramuscular temperature reduction because adipose tissue inversely effects the intramuscular temperature reduction (Lowden & Moore, 1975). Females, also could not produce as much torque, especially evident at the slowest velocity, which may have resulted in foot pound deviations between the two treatments (no cold and cold). There was a smaller deviation between treatments at each of the three velocities.

These combinations, less foot pound divergence between treatments and placement in the higher adipose thickness and/or body fat percentage groups (less intramuscular temperature reduction) would account for the stable measurements between treatment means for the females. Specifically, the female group (greater

adipose densities) had an overall peak toque mean of 59.52 versus 114.52 foot pounds for male group and a difference between treatment means of 1.72 to 11.14 foot pounds, respectively. The female group also had -1.11, -0.77 and +3.6 foot pound differences for cold treatment torque means at 450, 360, and 180 velocity levels, respectively. The male group had torque improvements for all velocity levels.

The gender differences in potential contractile force, in subcutaneous fat depth at the treatment site, and in overall body fat may also explain the two interactions of the mixed design analyses:

1. Skin fold measurements of the treatment area interacted with velocity but not cold. A graph of velocity levels for greater and lesser skin fold thickness groups showed an ordinal interaction primarily between the intermediate velocity of 360 degrees per second.

2. Body fat interacted with cold but not velocity. The cold had an effect upon the less than 16 percent body fat group. Further analyses revealed that the less adipose tissue group increased in peak torque but the greater adipose tissue group had no improvement between treatments and maintained stable measurements. Therefore, the two groups, male and female, or the higher versus the lower fat percentage and those with a thicker versus lesser tissue density at the treatment site, confounded the results and created an interaction.

## Cold Effect Upon ROM

Cold also influenced ROM for peak torque occurrence. The treatment was significant ($F(1,23) = 7.31$, $p < .02$) as the complete analysis indicated (see Table 8). As previous analyses have shown, the cold treatment resulted in increased peak torque for all subjects and were especially evident in the mixed analyses that evaluated the influence of adipose tissue thicknesses at the treatment site and overall body fat. However, additional mixed analyses comparing ROM differences between < 16% body fat and > 16% body fat showed no significant difference.

Another additional mixed analysis on posterior skin-fold categories (thickness ≤15 mm or ≥19 mm) showed significant differences between groups ($F(1,22) = 5.473$, $p < .03$) and treatment influence on ROM ($F(1,22) = 8.167$, $p < .001$). At each velocity level, the treatment caused the ROM to shift away from extension. However, both groups were consistent for this effect and further investigation only indicated that the ≥15 mm group merely peaked farther from extension at the means of 54.1 degrees for no cold and 56.4 degrees for cold treatment. Whereas, the ≥19 mm group peaked at 47.1 degrees and 50.8 degrees. The degrees of separation between treatments and velocities were consistent for both groups and without interaction.

There is no explanation as to why the quadricep muscle's peak torque increased with cold treatment to the hamstring but the ROM at which this occurred was consistently prior (occurring earlier) for chilled hamstrings. The cold treatment should have caused neural conduction velocity to diminish. Thus, agonistic (quadricep) contraction would have continued longer before the previously inhibited

antagonistic muscle group (hamstring) would progressively contract and decelerate movement and inhibit the contract of the agonist. This would have resulted in ROM with the increased peak torque occurring closer to complete extension. In other words, achieving delay of the agonist (quadriceps) peak torque by inhibiting the antagonist's deceleration effect would have increased peak torque as well as have placed the occurrence closer to full extension.

There may be a neural protective mechanism that prevents greater torque production once a preset maximum has been achieved and this neural influence is not dependent upon antagonist contraction but rather by agonist proprioceptive mechanisms or is supra spinal influenced, or internal muscle influences not attributed to neural influence. (Basmajian & De Luca, 1985; Grabiner & Hawthorne 1990; Perrine & Edgerton, 1978)

## Cold Effects Upon Velocity

The main effects of velocity were significant for all analyses. The design of the study created an effect between velocities because the three speed settings (levels) on the isokinetic dynamometer applied the force-velocity principle or velocity and force are inversely proportional. Thus, as the velocity setting on the instrument was increase, the force would diminish. The principle was demonstrated throughout the study.

There was a tendency for the peak torque (foot pounds) to diverge between treatments at the initial and highest velocities and for convergence to occur at the intermediate velocity. In other words: (a) the cold application would have greater

differences for peak torque at the initial velocity (180 degrees per second), (b) but at the intermediate velocity there was a reduction in the foot-pounds, creating less disparity between treatment peak torques, and(c) the final measured velocity (450 degrees per second), again increased in treatment differences for the peak torque. The convergence was especially apparent for the <16% body fat group. The peak torque for cold treatment increased by 7.62 foot-pounds at the initial velocity, reduced to a disparity of 1.54 foot pounds for the intermediate velocity, and finally increased disparity again to 4.74 foot pounds at fastest velocity level. The >16% body fat group continued a steady decline of 2.12, 0.61, and -2.05 from initial to fastest velocity levels respectively. Thus, an interaction was created unless the <16% group was separated from the >16% body fat percent group.

## Summary

This research concludes that:

1. There was a difference between treated hamstrings which results in greater torque production of the quadriceps for the chilled hamstring.

2. The ROM at which maximal torque occurred was further from extension for the chilled hamstring.

3. There was a significant difference between main effects of peak torque relative to the adipose tissue characteristics at the treatment site and/or the subjects' percent body fat.

4. There was no significant differences between peak torque occurrence and ROM for either adipose tissue characteristic.

## Recommendations

The conclusions indicate that cold application to the antagonist does influence the peak torque and the ROM of the agonist. Cold application in this study emulated a common clinical procedure of applying ice massage to a muscle group. The assumption is that a patient might engage in either an activity or a quantitative evaluation for contractile capacities on an isokinetic dynamometer after ice massage. Because most patients are being treated for injuries, further research would determine the intensity that injured muscles are influenced by cold application relative to healthy subjects. Considering that cold reduces NCV and would inhibit pain, there might be more consequential implications. Whether the potential inhibition is detrimental or beneficial is unknown, however this study indicates that an *activity* or a *quantitative evaluation* immediately after application would be contraindicated to healthy patients and thus probably to specific types of muscle injuries as well. Contraindications would be especially opposing for subjects of low body fat percentage and/or minimal tissue thickness at the treatment site.

Further research could investigate the influence of various methods of cold application, length of application, and time interval between application and activity. Also if cold is applied in conjunction with an antagonist pre-contraction, there may be further enhancement in the MVC of the agonist. This could have significant influences upon strengthening programs whether conducted as therapeutic protocols or as adjuncts to strength training. Other researchers and studies have already proposed using the pre-contraction to enhance contractions of the agonist

Conversely, the unexpected occurrence of cold creating the agonist muscle group to achieve maximal voluntary contraction sooner than the non-treated limb (antagonist) is of significant clinical implication. Thus, prior to activity, cold application might help protect a muscle group from excessive stretch. Clinically cold acting as an adjunct to provide additional protection from over-stretching would have greater rehabilitative importance. Therefore, further research in the ROM phenomena might be more beneficial for the clinical or sports medicine setting.

This study does not convey nor imply any methods to enhance or limit therapeutic procedures or evaluations other than the cautions and considerations that have been specified. Nor does this study imply or recommend any athletic training or coaching techniques. There must be further investigation of both the increase in MVC and the change in occurrence of ROM. It must be remembered that the study used healthy subjects that performed voluntary contractions under open kinetic chain conditions. It is not known whether cold application to an injured antagonist will influence the agonist's maximal voluntary contraction (MVC) or ROM at which MVC occurs is known. Nor is it known, how cold application to the antagonist will influence the agonist during closed kinetic chain conditions.

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# APPENDICES

# Appendix A
## Subject Consent Form

# CONSENT FORM

Title: The Effect of Hamstring Temperature Reduction on Quadricep's Torque

Investigator: **Russ Cagle**

Purpose: **To determine the effect of intramuscular temperatue reduction of the hamstrings on quadricep's torque curve at different velocities.**

I have received an oral explanation of the current study and understand it will entail the following:

All testing will be done at the Rebound Sportsmedicine Rehabilitation Center located in Albany, Oregon. This will involve private transportation to Albany. I am willing to assume responsibility for any accident and injuries due to this necessary travel should, I drive to Albany. I also will not hold the investigator or any affiliations responsible for any vehicle accident in which I elect to be transported in order to participate in this study. My involvement in the experiment will consist of one visit to the rehabilitation clinic during which it will take approximately one hour to conclude the testing.

The testing will be done on the Biodex™ isokinetic dynamometer and will involve three different velocities. At each velocity settting on the Biodex™, I will be required to perform five maximal contractions of my quadriceps and hamstring muscle groups. The accommodating resistance that the isokinetic device applies will match my muscle contractions to limit contractile velocity to a preset value. The machine does not store or apply any external load. The machine will only measure the force that I apply to the lever arm. The testing protocol will be according to the isokinetic dynamometer's criteria and is the same procedure that is used to evaluate injured limbs. I understand the minimal risk of injury associated with participating in this study. The skinfold measurements that will be conducted are familiar procedures to me and there is no risk in this aspect of the study. I will have an ice massage performed to my posterior thigh for ten minutes and am willing to receive this noninvasive cold application.

The benefits of my participation in the study include contributing to the scientific study of the effect on intramuscular temperature reduction of the hamstrings torque values for quadricep muscular contraction and obtaining knowledge concerning my untreated and cold-treated hamstrings maximal contraction torque values at the three different velocities.

I understand that the results of my participation in this study will remain confidential and that I will not be identified in any way in the presentation or publication of the findings of this investigation.

I have been completely informed of and understand the nature and purpose of this research. The researcher has offered to answer any questions that I may have. I understand that my participation in this study is completely voluntary and that I may withdraw from the study at any time without penalty. I understand compensation or medical treatment is not provided by Oregon State University, Willamette University, or Rebound Clinic if I am injured as a result of participation in the research project.

I have read the foregoing and agree to participate in this study.

_____

Subject's Signature Date

_____

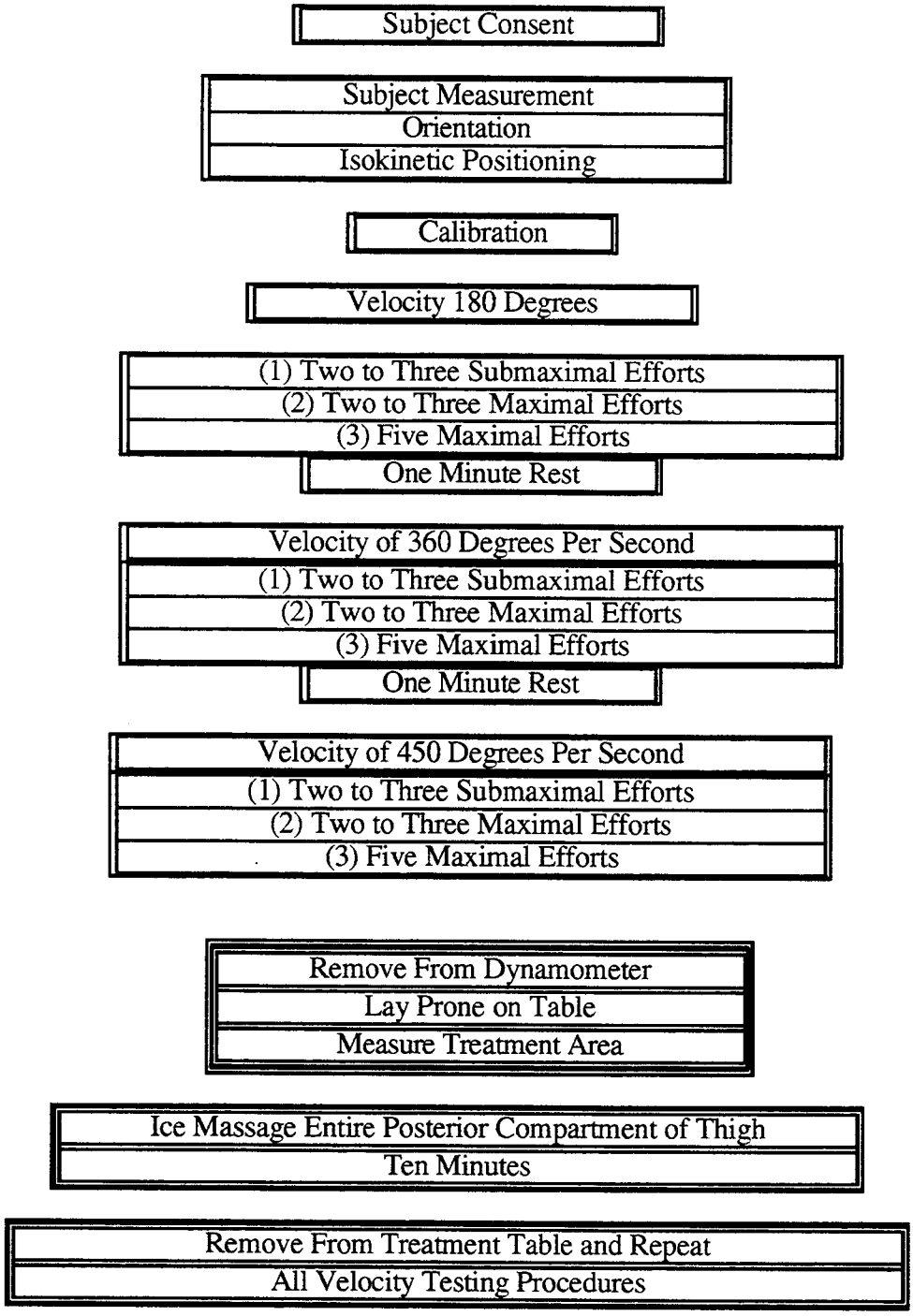
Subject's Address

_____ _____

Investigator's Signature Date

# Appendix B

## Biodex® Testing Procedure


Subject Consent
Subject Measurement
Orientation
Isokinetic Positioning
Calibration
Velocity 180 Degrees
(1) Two to Three Submaximal Efforts
(2) Two to Three Maximal Efforts
(3) Five Maximal Efforts
One Minute Rest
Velocity of 360 Degrees Per Second
(1) Two to Three Submaximal Efforts
(2) Two to Three Maximal Efforts
(3) Five Maximal Efforts
One Minute Rest
Velocity of 450 Degrees Per Second
(1) Two to Three Submaximal Efforts
(2) Two to Three Maximal Efforts
(3) Five Maximal Efforts
Remove From Dynamometer
Lay Prone on Table
Measure Treatment Area
Ice Massage Entire Posterior Compartment of Thigh
Ten Minutes
Remove From Treatment Table and Repeat
All Velocity Testing Procedures

# Appendix C

## Demographics of Subjects

| Lbs | Age | Post Thigh SKF | Post Thigh Measurements | | | | limb tested |
|---|---|---|---|---|---|---|---|
| 200 | 22 | 15 | 36 | 26 | 18 | | R |
| 184 | 21 | 12 | 33 | 17 | 13 | | L |
| 163 | 19 | 6 | 28 | 18 | 12 | | R |
| 165 | 22 | 11 | 32 | 19 | 13 | | L |
| 180 | 22 | 17 | 34 | 18 | 13 | | L |
| 158 | 22 | 8 | 34 | 18 | 12 | | L |
| 164 | 25 | 15 | 33 | 19 | 12 | | R |
| 182 | 21 | 12 | 28 | 19 | 13 | | L |
| 241 | 22 | 22 | 35 | 22 | 14 | | R |
| 165 | 21 | 12 | 29 | 17 | 12 | | R |
| 181 | 23 | 12 | 31 | 17 | 12 | | R |
| 180 | 25 | 20 | 35 | 21 | 12 | | L |
| | | | | | | | |
| 149 | 22 | 19 | 29 | 19 | 14 | | R |
| 138 | 23 | 18 | 30 | 15 | 12 | | L |
| 106 | 21 | 19 | 28 | 17 | 11 | | L |
| 122 | 22 | 22 | 32 | 16 | 12 | | L |
| 135 | 21 | 18 | 26 | 17 | 12 | | L |
| 160 | 20 | 28 | 34 | 19 | 13 | | R |
| 124 | 18 | 24 | 28 | 19 | 12 | | R |
| 134 | 23 | 19 | 32 | 15 | 11 | | L |
| 119 | 22 | 16 | 30 | 17 | 10 | | L |
| 130 | 24 | 18 | 33 | 18 | 12 | | L |
| 98 | 18 | 20 | 26 | 15 | 10 | | L |
| 120 | 35 | 22 | 29 | 21 | 12 | | R |

Population Demographics
Skin-fold sites & measurements
Percentage of body fat

| Subject | Triceps | Suprailium | Thigh | Total | Sex | | % Fat | Lbs | Age |
|---|---|---|---|---|---|---|---|---|---|
| | Chest | Abdomen | | SKF | | | | | |
| DK | 6 | 21 | 20 | 47 | M | | 13.31 | 200 | 22 |
| SY | 5 | 16 | 9 | 30 | M | | 8.07 | 184 | 21 |
| SK | 4 | 10 | 6 | 20 | M | | 4.71 | 163 | 19 |
| DM | 4 | 12 | 10 | 26 | M | | 6.94 | 165 | 22 |
| JW | 12 | 25 | 16 | 53 | M | | 15.05 | 180 | 22 |
| KA | 3 | 9 | 8 | 20 | M | | 5.04 | 158 | 22 |
| TD | 5 | 18 | 10 | 33 | M | | 9.44 | 164 | 25 |
| BK | 5 | 11 | 13 | 29 | M | | 7.76 | 182 | 21 |
| KW | 25 | 55 | 18 | 98 | M | | 26.85 | 241 | 22 |
| GW | 5 | 12 | 13 | 30 | M | | 8.07 | 165 | 21 |
| TS | 5 | 17 | 8 | 30 | M | | 8.29 | 181 | 23 |
| JA | 4 | 29 | 17 | 50 | M | | 14.52 | 180 | 25 |
| | | | | | | | | | |
| LB | 16 | 11 | 26 | 53 | F | | 21.31 | 149 | 22 |
| LC | 19 | 9 | 24 | 52 | F | | 21.03 | 138 | 23 |
| KR | 13 | 11 | 22 | 46 | F | | 18.86 | 106 | 21 |
| IB | 20 | 12 | 18 | 50 | F | | 20.29 | 122 | 22 |
| WW | 16 | 12 | 15 | 43 | F | | 17.81 | 135 | 21 |
| DO | 18 | 15 | 22 | 55 | F | | 21.85 | 160 | 20 |
| DG | 13 | 11 | 18 | 42 | F | | 17.27 | 124 | 18 |
| TA | 16 | 13 | 22 | 51 | F | | 20.70 | 134 | 23 |
| LA | 13 | 7 | 16 | 36 | F | | 15.37 | 119 | 22 |
| JB | 16 | 11 | 24 | 51 | F | | 20.46 | 130 | 24 |
| KM | 12 | 7 | 18 | 37 | F | | 15.49 | 98 | 18 |
| DC | 18 | 12 | 28 | 58 | F | | 23.79 | 120 | 35 |

Population Demographics

By Gender

| | Body Weight | % Fat | Post. Thigh |
|---|---|---|---|
| Overall mean | 154.08 | 15.11 | 16.88 |
| Overall S.D. | 33.15 | 6.46 | 5.21 |
| Male | 180.25 | 10.67 | |
| S.D. | 22.6 | 6.13 | |
| Female | 127.9 | 19.54 | |
| S.D. | 17.15 | 2.60 | |

By Groups

| | <16% Body Fat >16% | | <18 mm Post. Thigh >18 mm | |
|---|---|---|---|---|
| MEAN | 9.72 | 20.51 | 12.83 | 20.58 |
| S.D. | 3.85 | 3.00 | 3.56 | 1.62 |

Appendix D

List of Terms and Definitions

Agonist: The muscle and/or group of muscles that are in contraction as distinguished from muscles,or group, that are relaxed at the same time, antagonist, in order to allow movement (Thomas, 1989).

Antagonist: The muscle and/or group of muscles that are relaxed or in opposition to the movement of the contracting agonist (Thomas, 1989).

Co-activation: The simultaneous activation of alpha and gamma motorneurons from higher centers for purposes of initiating or perpetuating muscle contractions (Sullivan, Markos & Minor, 1982).

Co-contraction: Simultaneous muscle contractions on both sides of a joint (Sullivan, Markos & Minor, 1982).

Range-of-Motion: The amount of movement that a joint can acheive, measured in degrees; when pertaining to the knee joint, zero degrees is complete extension and ninety degrees of flexion is the leg perpendicular to the thigh (Basmajian & De Luca, 1985).

Reciprocal Inhibition: Inhibition of muscles antagonistic to those being facilitated; this is essential to coordinated movement (Basmajian & De Luca, 1985).

Velocity in-vivo: The amount of angular displacement, measured in degrees per second, that the limb can move within its ROM (Moore, 1978).