AN ACUTE BOUT OF STATIC STRETCHING: EFFECTS ON FORCE AND JUMPING PERFORMANCE

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An Acute Bout of Static Stretching: Effects on Force and Jumping Performance

By

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ABSTRACT

Static stretching (SS) is used pervasively throughout the exercise science and sporting communities as a fundamental element of the pre-exercise warm-up procedure. While SS has been proven effective to increase range of motion (ROM), its proposed benefits are questionable and relatively unsubstantiated. Moreover, in recent years researchers have demonstrated that pre-exercise SS may in fact be detrimental to subsequent performance due to impaired force and power output. Yet the practical application of these relatively recent findings is limited due to the prolonged SS protocols utilized. In addition, timelines associated with enhanced ROM and performance decrements have not been established. Thus, the objectives of this study were to determine if a typical SS routine decreased force, activation, and power while improving ROM. Secondly, the study attempted to compare the duration of the performance decrement to the duration of the augmented ROM.

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"If you're not fifteen minutes early you're late."

CO-AUTHORSHIP STATEMENT

The following statements clearly identify my role in the thesis development, execution, and preparation:

1) **Design and identification of the research proposal**: The research proposal is a combination and extension of previous research conducted by Dr. David Behm and Dr. Warren Young. To modify and expand on the original studies Dr. Behm and I discussed and developed the methodology utilized. Dr. Behm provided a general overview of the study and identified the variables to be measured and I developed the precise procedures.
2) **Practical aspects of the research**: Raw data was collected by Michael Carroll, Farrell Cahil, and myself.
3) **Data analysis**: Under the auspices of Dr. Behm I performed all data analysis procedures.
4) **Manuscript preparation**: Under the auspices of Dr. Behm I prepared the manuscript.

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LIST OF ABBREVIATIONS AND SYMBOLS

AT – anaerobic threshold
CJ – concentric jump
cm - centimeters
CR – contract relax
CRAC – contract relax agonist contract
DJ – drop jump
DS – dynamic stretching
GTO – Golgi tendon organ
H-reflex – Hoffman reflex
iEMG – integrated electromyography
ITT – interpolated twitch technique
kg - kilograms
km/hr – kilometers per hour
min – minute
MN – motoneuron
MTU – musculotendinous unit
ms – milliseconds
MVC – maximal voluntary contraction
PEC – parallel elastic component
PF – plantar flexors
PNF – proprioceptive neuromuscular facilitation
PT- peak twitch
RE – running economy
ROM – range of motion
rpm – revolutions per minute
s – seconds
SEC – series elastic component
SS – static stretching
SSC – stretch shortening cycle
VO_{2max} – maximal aerobic power
VJ – vertical jump
WU – warm-up
α - alpha
^0C – degrees centigrade
γ - gamma

1. INTRODUCTION

1.0 Overview

Stretching and flexibility training amongst athletes (recreational or professional), rehabilitation clinicians, and scientists have traditionally been utilized as a means to increase range of motion about the joint (3;6;7;9). In addition, stretching programs are routinely implemented as a mechanism to decrease muscle soreness (10), reduce (15) or prevent (16) the risk of injury, rehabilitate following injury (11) and improve athletic performance (1).

Conversely, relatively recent studies have found no evidence to support the use of stretching for the aforementioned intentions. Furthermore, a body of evidence now exists suggesting that acute and prolonged bouts of stretching may in fact reduce the human performance variables it was once thought to enhance via an acute decrement in strength (2;4;8;12-14) and power output (5;17). This growing body of evidence is questioning the role of stretching prior to athletic events leading some researchers and practitioners to recommend against its use. However, the majority of the protocols utilized to study the neuromuscular effects of stretching are prolonged and not representative of a conventional stretching routine. Furthermore, the duration for which the decrements in strength and power output persist following a stretching routine has not been thoroughly investigated. Accordingly, additional research is required to justify the use or disuse of stretching in the aforesaid settings. Thus, the proposed study will employ a moderate stretching routine to investigate the aforementioned variables which may have direct implications for (1) the manner in which rehabilitative procedures used by occupational and physical therapists are administered in the post-injured or recovering individual (2)

the manner in which exercise scientists conduct experimental procedures involving warm-ups and stretching and their possible effects on performance variables (3) the training protocols utilized by athletes.

1.1 Statement of the Problem

The problem of the study was twofold: (1) to determine whether a moderate static stretching routine was sufficient to decrease force and power output and (2) to establish a force and power output deficit timeline if in fact performance decrements were observed.

1.2 Purpose of the Study

If a moderate static stretching routine causes acute impairments in maximal force and power output, athletes must reevaluate its use prior to athletic events. It was believed that the information provided by this study would contribute to a more complete picture regarding the effects of static stretching on human performance by determining the extent and duration of performance impairment following moderate static stretching.

1.3 Research Hypotheses

It was hypothesized that a moderate static stretching routine would result in:

(1) decreased voluntary force and activation

(2) decreased jumping performance

(3) increased range of motion

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2. REVIEW OF LITERATURE

2.0 Introduction

Since the time of the ancient Greeks when the Olympic movement originated, highly competitive sport and the need to excel has been embedded in athletes. The Greeks participated in highly organized sporting activities known as agonistics and referred to their method of physical training as gymnastics (1). It has been suggested that one of the training methods commonly employed by the Greeks to enhance acrobatic, dancing, and athletic abilities was flexibility training (141).

In today's society, over two thousand years later, stretching and flexibility training amongst athletes, rehabilitation clinicians, and sport scientists are commonly utilized to increase range of motion (ROM) about the joint (6;37;51). Stretching programs are routinely implemented as an intervention to decrease muscle soreness (54), reduce (109) and/or prevent (115) the risk of muscular injury, rehabilitate following injury (62) and improve athletic performance (141).

Nonetheless, with the exception of an increased ROM, relatively recent studies have found no evidence to support the use of stretching for the aforementioned intentions. A body of evidence now exists suggesting that acute and prolonged bouts of stretching may in fact reduce the human performance variables it was once thought to enhance via an acute decrement in strength (10;38;69;71) and power output (27;137). Studies have shown that stretching prior to exercise does not reduce the risk of injury (17;53;100) while others have suggested that pre-exercise stretching may in fact increase the risk of

injury (114). This growing body of evidence is questioning the role of stretching and flexibility training in athletic and therapeutic venues.

This relatively new and opposing evidence to the use of stretching has led some researchers and practitioners to recommend against static stretching (SS) prior to athletic or therapeutic events (138). However, the majority of the protocols utilized to study the neuromuscular effects of stretching are prolonged and not representative of a stretching routine employed by athletes (10;38).

There are many questions to be answered about stretching and its use as a performance enhancer and therapeutic intervention before it can be dismissed as more harmful than beneficial. For example, there are several areas within the realm of stretching which have not been thoroughly investigated including: (1) the optimal stretching methods necessary to increase ROM such as type of stretch, duration, repetitions, sets, and frequency (2) the amount of passive tension needed to be placed on the musculature during a stretching routine to cause a decrement in force output (3) the duration for which the decrements in strength and power output persist following a prolonged stretching routine and (4) whether a moderate stretching routine will induce a force deficit.

Accordingly, additional research is required to answer such questions in an attempt to justify the use or exclusion of stretching in rehabilitative and more specifically athletic settings. The objective of this chapter is to review the literature concerning various types of stretching as part of a warm-up (WU) and their effects on subsequent performance.

2.1 Warm-up Procedures

It is generally accepted and recommended by fitness professionals that a moderate WU prior to exercise is necessary and beneficial to exercise performance and reduces the risk of injury (41;54;62;109;115). A WU is generally divided into two main categories: passive or active, both with the common goal of increasing muscle temperature in preparation for exercise by literally warming-up the muscle to increase metabolic rate and perhaps more importantly to increase muscle extensibility. More specifically, warming-up can result in decreased muscle viscosity (113), increased oxygen uptake during subsequent exercise (58), nerve conduction velocity (118), glycolysis (117), ROM (120), anaerobic performance (120), muscle tensile strength (108), as well as augmenting an earlier sweating response during exercise, thus enhancing thermoregulation (24). However, there is considerable variation in the methods used to WU in an attempt to attain the aforementioned results.

The variation in WU procedures result from a variety of passive and active techniques readily available and commonly utilized. Passive WU's may be defined as methods of increasing peripheral or core body temperature without expending higher than resting amounts of energy. Active WU's can be divided into general or specific. General WU's increase body temperature by nonspecific body movements and specific WU's increase body temperature by movements of body parts that will be used in ensuing activities (4).

2.1.1 Passive Warm-ups

Various passive WU's used by athletes and coaches alike may include hot showers, massage, application of deep heat via ultrasound, superficial heat via hot packs, and stretching. A study by Strickler et al. (121) investigating the effects of passive warming on the musculotendinous unit (MTU) of the rabbit hindlimb suggested that passive warming increases the extensibility of the MTU and that this may reduce its susceptibility to strain injury. It is also common for a combination of passive techniques to be used concurrently to obtain an optimal WU. A study by Funk et al. (39) combined SS and moist heat application and demonstrated that this combination produced significantly greater flexibility of the hamstrings as opposed to non-combined procedures, thus greatly reducing the risk of muscular strain. Similarly, Knight et al. (68) investigated various means of warming-up on the extensibility of the plantar flexors (PF) muscle group. All methods utilized, including the application of deep heat, superficial heat, and active exercise, demonstrated a significant increase in ankle dorsiflexion passive ROM. However, the researchers suggested that the most effective means of passive WU prior to stretching might be the application of deep heat.

2.1.2 Active Warm-ups

Although a passive WU can increase ROM, possibly decreasing the risk of muscular strain, the more popular and sport specific WU procedure involves active techniques. Typical active WU's are often sport specific and aerobic in nature. Stewart and Sleivert (119) conducted a study consisting of no WU and a 15 min WU at 60, 70,

and 80% of maximal aerobic performance (VO_{2max}) on a treadmill to study its effects on ROM of the hip, knee, and ankle in addition to anaerobic performance. They found that although body temperature and ROM were significantly raised following all intensities of WU only the 60 and 70% of VO_{2max} conditions improved anaerobic performance. Martin et al. (84) concluded that an elevated muscular temperature arising from a 15 min treadmill WU at 10 km/hr was sufficient to lower lactate production in subsequent exercise by 25% suggesting that an elevated muscle temperature may improve the aerobic contribution to short exhaustive running. A WU of too high an intensity may impair performance. Bishop et al. (16) studied the effects of varying WU intensities on kayak ergometer sprint performance. They concluded that a WU intensity of 75% VO_{2max} may impair supramaximal performance by reducing the efficiency of contractile processes or decreasing the contribution of energy production via anaerobic sources. Thus, it appears that although a WU is recommended to enhance aerobic and anaerobic performance, the intensity, type, and duration is of utmost importance and must be chosen with care.

2.1.3 Conventional Warm-ups

Having viewed passive and active WU's separately, it must also be noted that a conventional WU involves a combination of both types. Possibly the most common procedure is to commence with a light aerobic activity of 5 min minimum to warm the muscles and connective tissues prior to the initiation of stretching (138). A plethora of studies have been conducted to determine the effects of actively warming-up through aerobic exercise followed by stretching of the musculature on ROM (120;130;131).

Wiktorsson-Moller et al. (130) studied the effects of warming-up and stretching on ROM of the lower extremities. Results showed that 6-15 min of massage and 15 min of WU cycling separately or in combination were not as effective as 15 min of WU cycling followed by stretching, which consisted of an "isometric contraction, followed by relaxation and a passive extension of the muscle," in increasing hip, knee, and ankle ROM. Williford et al. (131) showed that ROM increased in the shoulder, hamstrings, trunk, and ankle with two different types of WU groups: a jog then stretch and stretch only group. The results did however show that jogging then stretching significantly increased ankle ROM as compared to the stretch only group. Nevertheless the authors concluded that both methods offered possible advantages to increasing ROM. Thus, it appears that a general WU preceding a stretching routine is optimal for improving ROM.

2.2 Function of Increasing Core Temperature

One of the objectives of warming-up prior to physical activity is to improve human performance through an increase in core temperature. Numerous studies have investigated the effects of actively warming-up on subsequent performance, yielding mixed results. Although the majority of the research has demonstrated that an increase in temperature facilitates human performance (16;24;46;65;77;103;120), other studies have shown inhibitory effects (16;40;120) as well as no effect (20;40) of warming-up on subsequent performance. These conflicting results may be attributed to discrepancies in the type of exercise, intensity, duration or any combination of these variables utilized in the WU procedure. In addition, the performance variables measured differ in their

assessment of anaerobic (16;40;46;120) and/or aerobic (24) type activities. Nevertheless, the act of warming-up to facilitate performance prior to physical activity is generally accepted and practiced worldwide with a myriad of proposed underlying physiological mechanisms. As phrased by Shellock and Prentice (113), an increased core temperature acts to stimulate a variety of interrelating "temperature-dependent physiological processes," affecting the cardiovascular, thermoregulatory, neuromuscular, and metabolic systems. Although an in-depth review of the physiology of WU is beyond the scope of this paper, the following sections will briefly discuss the effects of warming-up on the cardiovascular, thermoregulatory, neuromuscular, and metabolic systems, respectively.

2.2.1 Cardiovascular / Thermoregulatory System

At rest, skeletal muscles receive approximately 15-20% of an individual's cardiac output, a value that increases to approximately 50% with light exercise, and 70% with heavy exercise (2). Thus, during exercise there is a redistribution of blood volume reducing the blood flow to the viscera and increasing the blood flow to the active skeletal muscle via vasoconstriction and vasodilation respectively (66). Vasodilation in active skeletal muscle resulting from reactive hyperemia (21;48), active hyperemia (103), autoregulation (106), and an increased release of hormones such as adrenaline (58), functions to assist in the production of energy aerobically via an increase in the transport of oxygenated blood. Moreover, vasodilation plays an integral role in the removal of metabolic by-products such as lactate, CO_2, and H^+ from the working muscles. These by-

products in addition to others have been demonstrated to diminish human performance (14;15;32;59;75;103).

Vasodilation near the surface of the skin will also assist indirectly to improve performance. During exercise, total body metabolism may increase 15-20 times the resting metabolic rate, resulting in increased metabolic heat production. This heat production may occur at a faster rate than heat dissipation, producing an increased body temperature referred to as hyperthermia (99). Thus, although the redistribution of blood flow to the working muscles is important for performance, vasodilation near the surface of the skin will occur in order to dissipate heat and assist in thermoregulation (24), reducing the possibility of hyperthermia. The importance of thermoregulation to performance has been studied in depth (35;45). MacDougall et al. (78) concluded that metabolic hyperthermia induced by heavy and prolonged exercise is detrimental to human performance (35;78). Mechanisms responsible have been attributed to an increased utilization of glycogen stores from the muscles and liver (112), elevated lactate concentrations resulting from tissue hypoxia (106), and an increased circulatory strain perceived as fatigue because the ability to transport oxygen to the muscle is reduced (106;111). A compromised muscle blood flow stemming from the dual demand for increased blood flow to the skin surface in an attempt to dissipate heat as well as to the working muscles may result in an increased concentration of waste products produced by the working muscles such as lactate, that are associated with fatigue (72).

The goal of a WU however is not to induce hyperthermia, but to increase temperature sufficiently enough to induce a cascade of events that may help improve

performance. Chwalbinska-Moneta and Hanninen (24) investigated the effects of warming-up at 40% of VO_{2max} on thermoregulatory, circulatory, and metabolic responses to incremental exercise in endurance athletes. Their results demonstrated that warming-up is effective in stimulating an early sweating response, thus increasing thermoregulation and attenuating the detrimental effects of hyperthermia. They also suggested that the slower rate of lactate accumulation following the WU resulted from faster peripheral circulation adjustment. Similarly, Martin et al. (84) showed that a WU resulted in a 25% lower lactate production than no WU, which was accompanied by faster cardiorespiratory adaptations to exercise, such as vasodilation in the skeletal musculature. Thus, an effective WU may act to increase cardiovascular efficiency in preparation for intense exercise through an increase in blood flow to the active skeletal muscle and myocardium, an increased dissociation of oxyhaemoglobin (84), and an earlier sweating response vital to thermoregulation (24). Perhaps more importantly Barnard et al. (8) demonstrated that a WU prior to intense exercise can eliminate or reduce the risk of potentially fatal heart arrhythmias arising from myocardial ischemia.

2.2.2 Metabolic System

Numerous studies have investigated the metabolic effects of an active WU on subsequent VO_{2max}. Perhaps one of the most commonly investigated and cited aspects of warming-up is its relationship between blood lactate accumulation and VO_{2max}. It is well established that intense exercise above the anaerobic threshold (AT) results in an accumulation of lactate in the active muscles causing metabolic acidosis via an increased

H^+ concentration and the ensuing decrease in pH. An increase in H^+ negatively affects muscle contractions with an inhibition of the release and uptake of Ca^{2+} and competition for binding sites on actomyosin (16). Furthermore, a decrease in pH can inhibit the activity of phosphofructokinase (PFK), an important rate-limiting enzyme in glycolysis (16) and lipolysis, an important factor for endurance type activities (61). Thus, it is the goal of athletes to avoid or delay the metabolic consequences of intense exercise with an efficient WU.

An effective WU will result in an elevated muscle temperature which may augment the production of energy via an enhanced speed of the rate-limiting enzymatic reactions involved in oxidative phosphorylation (70) and a more rapid dissociation of oxygen from hemoglobin (70). In the case of aerobic activities, this would be of great benefit, as it would allow the onset of steady state exercise to be attained relatively quickly resulting in a lower reliance on anaerobic fuel sources and a decreased oxygen deficit, thus less lactate production (24).

Genovely and Stamford (40) investigated the effects of a prolonged WU above and below the AT on maximal performance. They demonstrated an increased core temperature following both WU's, with an increase in blood lactate concentration associated only with the above AT condition. Although warming-up below the AT did not result in an increased performance, WU above the AT resulted in a decreased work output and peak blood lactate concentration. This seemingly contradictory finding (i.e. decrease in performance associated with decreased blood lactate concentrations) was attributed to probable glycogen depletion in the fast twitch muscle fibers. Glycogen

depletion would not allow a significant build up of lactate while impairing maximal anaerobic performance. Ingjer and Stromme (58) pointed to the possible effectiveness of active WU's when they demonstrated a higher oxygen uptake and a lower lactate concentration and blood pH during exercise when followed by an active WU as compared to passive or no WU. Possible mechanisms to explain the decrease in lactate production include a faster adjustment of the peripheral circulation resulting in improved thermoregulation (24) and an increased rate of lactate clearance through reuptake by the skeletal muscle (46). Lower lactate production following WU has also been found by other researchers (84;103) and may be attributed to an increased AT (24).

2.2.3 Neuromuscular System

The efficiency of the neuromuscular system is improved in numerous ways with an increase in temperature. An increased temperature can result in a decreased sensitivity to stretch (74) and muscle viscosity (109), as well as faster contraction (30) and nerve conduction velocity (118), all of which may aid in performance. Kulund and Tottossy (74) noted that a warmed muscle is more relaxed, meaning that the activity of the gamma (γ) nerve fibers and the sensitivity of the muscle spindles to stretch are reduced. This suggests that a warm muscle would be capable of an increased ROM, reducing the risk of muscular injury. As stated in a review by Safran and colleagues (109) muscle viscosity is also reduced leading to smoother muscle contractions following warming of the muscle, indicating the potential importance of a WU on various sporting activities where precision of movement is of utmost importance.

Additional studies have investigated the effects of temperature on muscular force and power output. Davies and Young (30) investigated the effects of heating and cooling on the contractile properties of the triceps surae. They reported that an increase of 3.1°C in muscle temperature increased contraction velocity as illustrated by a decreased time to peak twitch and half relaxation time with no effect on maximal voluntary contraction (MVC). Conversely, cooling of the muscle by 8.4 °C resulted in prolonged temporal characteristics of evoked contractile properties. The cold-induced decrease in contraction velocity may be attributed to a reduction in the activity of actomyosin adenosine triphosphatase (7), thus slowing cross-bridge cycling (13). Correspondingly, Stegeman and De Weerd (118) showed that a decreased muscle temperature attenuated conduction velocity and increased compound action potential duration, while the reverse was evident with increased muscle temperature. The relative importance of contraction velocity and the rapid transmission of neurological signals are essential to any movement where speed or power is vital to performance. An augmented conduction velocity could result in an increased contraction velocity and possibly contribute to an increased power output, thereby accentuating the importance of warming the muscle prior to performing powerful movements.

Although an increased muscular temperature appears to increase velocity-dependent dynamic force (13;110), studies have shown no enhancement of isometric force (13;30) following warming of the muscle. Conversely, cooling of the muscle has resulted in a decreased MVC (29;31;102), possibly resulting from interference of the metabolic and maximal force production mechanisms, although the exact mechanisms

have yet to be elucidated. An interesting study by Sargeant (110) showed that passive warming of the legs in a 44°C water bath resulted in an increase of approximately 11% in maximal dynamic force and power output as compared to control subjects. Conversely, cooling of the legs resulted in reductions of approximately 12% and 21% respectively. Similarly, Bergh and Ekblom (13) concluded that maximal dynamic force was reduced at low muscle temperatures and enhanced at temperatures above normal. In practical terms they noted that jumping and sprinting performance were attenuated via low muscle temperature and enhanced via an increased muscle temperature.

It appears the ability to produce maximal force is likely temperature independent while rate process dependent entities such as dynamic strength and power output are temperature dependent. Thus, as power is the product of force and velocity it seems logical to warm the muscle prior to explosive, dynamic exercises such as sprinting and jumping or at the very least preventing a decrease in muscle temperature sufficiently enough to impair performance.

2.3 Stretching

Stretching is commonly used as a means to increase ROM about the joint by professional and recreational athletes, rehabilitation clinicians, and sport scientists (6;33;37;51). The term 'stretching' however is an imprecise term encompassing a multiplicity of techniques, fitting into three general categories (i.e. SS, dynamic (DS), and proprioceptive neuromuscular facilitation (PNF)) each of which includes several sub-domains. For example, stretching in general can be broken down into active or passive, or

a combination of both as is the case in PNF stretches. To complicate matters even further, there are differences between passive (static) and active (dynamic) ROM. Passive ROM refers to "the degree to which a joint may be passively moved to the end-points in the ROM" (113) and active ROM refers to "the degree which a joint can be moved as a result of a muscle contraction usually through the midrange of movement" (113). The following sections will serve to review research pertaining to each type of stretching (SS, DS, and PNF).

2.3.1 Static Stretching

SS involves placing the muscle to be stretched (commonly referred to as the antagonist (4;99)) into a position of maximal or near maximal stretch (99). This position of near maximal stretch is subjectively described as the position of the onset of pain or mild discomfort. SS can further be broken down into active or passive stretching. An active stretch occurs when the individual that is stretching applies their own force via voluntary contraction of the agonist, whereas a passive stretch occurs when a partner applies the necessary force for the stretch of the antagonist (4). Further complications arise by combining active and passive techniques referred to as passive-active stretching and active-assisted stretching (1). Passive-active stretching involves an outside force placing the muscle into a position of maximal stretch followed by the active contraction of the agonist to hold the position. Active-assisted stretching involves the contraction of the agonistic muscle group followed by the placement of an external force to assist in further stretch of the antagonist (1).

From the various definitions and techniques it is of no surprise discrepancies in the literature exist regarding the most effective means of SS to increase ROM.

2.3.2.1 Static Stretching Effectiveness

Numerous studies have concluded that SS is effective in increasing ROM (6;22;36;104), while few studies have found no increase in ROM (140). However, the optimal method by which an increase in ROM is achievable is the subject of much debate. Even one of the most rudimentary aspects of stretching, such as the duration of the stretch has been relatively undetermined. Studies have investigated a broad assortment of sustained SS durations as well as various SS programs on ROM. Although support exists to hold stretches for a relatively long duration (i.e. $\geq$ 30 seconds (s)) (6;22;36;36;36), there is also support for shorter duration stretches (i.e. $\leq$ 20 s) (36;104;107) to increase ROM. The lack of consistency amongst the methods employed makes it difficult to ascertain an optimal stretching routine.

Animal studies have provided some insight into optimal techniques. In a study by Taylor et al. (123), rabbit MTU's were stretched to determine the duration and number of stretches necessary to induce length gains. They demonstrated that the amount of stress relaxation following the initial 12-18 s of the stretch appeared to be much less significant than the changes evident in the initial 12-18 s. They also concluded that the greatest length changes in the MTU occurred within the first four stretches. Hence, one could speculate that a stretch duration of approximately 15 s performed four times may be sufficient to induce MTU length increases, thus increasing ROM. However, human to

animal (rabbit) differences in muscle volume, fiber type homogeneity, and other variables may not permit a precise extrapolation to humans.

In humans, several studies (104;140) have investigated the effects of 15 s SS's on ROM yielding somewhat opposing results. Zito et al. (140), studied the immediate effects of one bout of two 15 s passive stretches on ankle dorsiflexion and concluded that there was no significant gains in active ROM. Conversely, Madding et al. (79) demonstrated that one bout of 15, 45, or 120 s of passive stretching significantly increased hip abduction active ROM measures immediately post-stretch. Furthermore, 15 s proved just as effective as 45 or 120 s. Differences between the studies may in part be due to the muscles studied and the amount of passive tension elicited during the stretch. Zito et al. (140) stretched the triceps surae and told the subjects to stretch to the point of slight discomfort while Madding et al. (79) stretched the hip adductors and used a dynamometer to ensure the subjects stretched to the point of a calculated stretch force. From these studies a concrete conclusion cannot be drawn as to the effects of 15 s SS's on ROM.

Furthering the use of 15 s SS's, Roberts and Wilson (104) demonstrated that 15 s of active SS was effective for increasing active and passive ROM as compared to a 5 s stretch and control group. There was no significant difference between stretching duration and passive ROM, however, the 15 s stretches showed significantly greater improvements in active ROM than the 5 s stretches. However, a comparison between the effects of 15 s SS's on ROM between this study and the above studies would be inappropriate. The protocol used by Roberts and Wilson (104) involved a five week training program and three stretching sessions per week with the total amount of time

spent in the stretched position equal between both groups, whereas the studies by Zito et al. (140) and Madding et al. (79) involved single bouts of stretching. Thus, there would be a discrepancy between chronic and acute changes in ROM respectively. Similarly, Borms et al. (18) compared the effects of 10, 20, and 30 s active SS's of the hamstrings over ten weeks and concluded that 10 s was sufficient to increase active ROM. However due to the relative vagueness of the methodology, it is difficult to compare the two studies. For example, Borms et al. (18) state that each of two stretching sessions per week lasted 50 min but fail to mention what the 50 min entailed.

In disagreement with the effectiveness of 15 s SS's, Bandy and Irion (5) investigated the effects of duration of passive SS on passive ROM of the hamstrings. They compared stretching programs whereby a SS was held for 15, 30, or 60 s five times a week for six weeks. It was concluded that holding a SS for 30 or 60 s was more effective in improving hamstring ROM than 15 s or no stretch. There was no significant difference in ROM gained between 30 and 60 s stretch durations suggesting that stretching for 30 s was sufficient. Bandy et al. (6) furthered their investigations by examining the frequency and duration of passive SS on passive hamstrings ROM. They again compared stretches of 30 and 60 s durations and compared them to three 30 s and three 60 s stretches. Total stretch times for each group were 180 s, 90 s, 60 s, 30 s, and no stretch. They concluded that one, 30 s passive SS per day was an effective amount of time to increase hamstrings passive ROM. There was no significant difference in ROM with a longer duration or more frequent SS routine.

In yet another variation, Feland et al. (36) concluded that 60 s of passive SS performed four times a day, five times a week over six weeks was more effective than 15 or 30 s to increase the passive ROM of the hamstrings. They did note however that the different findings between their study and that of Bandy and colleagues (6) may have been due to the difference in the subjects chosen. Whereas the average age of the subjects for the study by Bandy et al. (6) was 26.5 years, the average age for Feland et al. (36) was 84.7 years. They concluded that stretches of longer duration may be necessary in the aged population to overcome increased muscle stiffness and collagen deposition. Another difference within the study of Feland et al. (36) may also be due to lack of control over total time in the stretched position. If you compare the total time spent in the stretched position in groups one (control), two (15 s), three (30 s), and four (60 s) over the 6 weeks they are 0, 30, 60, and 120 min respectively. Thus, their conclusion seems somewhat unwarranted in that it is unclear as to whether stretches of longer duration or stretches of shorter duration performed more frequently are necessary to increase flexibility.

2.3.3 Dynamic Stretching

The terms ballistic stretching (BS) and dynamic stretching (DS) are often used interchangeably (this paper will use the term DS) and are characterized by an action-reaction bouncing motion in which the muscle to be stretched (antagonist) is subjected to an extreme ROM due to fast, active contractions of the agonist muscle groups (99).

2.3.3.1 Dynamic Stretching Effectiveness

There has been a limited amount of research directed towards DS when compared to SS due in large part to its quick, jerky, and forceful nature. Many researchers and practitioners fear that this type of movement may increase the likelihood of muscle or tendon injury (9;52) if the force generated by the bouncing motion exceeds the tissues extensibility threshold (113). Nevertheless, one must question the use of SS, the most widely accepted means to increase ROM (1;5) when human movement is dynamic in nature. This use of SS to increase dynamic ROM would oppose the training principle of specificity as it applies to other various training stimuli. As illustrated by Hardy and Jones (50), the measurement of dynamic not static flexibility, may give more insight into the potential benefits in speed-related sport performance. Although DS is commonly executed in track and field sports, its adoption has been slow in other sports (52).

Research has demonstrated DS to be an effective (76;93;129) as well as an ineffective (107) method to increase ROM. Sady et al. (107) compared DS, SS, and PNF stretching techniques on the ROM of the shoulder, trunk, and hamstrings muscles. In the DS group, subjects repeated a full ROM rapidly 20 times once a day, three days a week for six weeks. They concluded that the DS method used in their study was insufficient to enhance active ROM. Lucas and Koslow (76) compared SS, DS, and PNF stretching on the ROM of the hamstrings-gastrocnemius muscles. The DS group participated in approximately 5 min of DS three times per week for seven weeks. The DS group incorporated a 20 s "gentle bobbing motion in lieu of the slow, sustained method implemented by the SS group." They concluded that this treatment resulted in a

significant increase in active ROM as measured by the sit and reach test. Similar to these findings, Hardy and Jones (50) concluded that DS was an effective means to increase short term active ROM of the hip extensors. In the first part of their study, subjects performed one of two DS techniques, the first stressing speed (speed-dynamic) and the second stressing range (flexi-dynamic). Both groups performed one experimental treatment daily for seven days consisting of three trials of 30 s each interspersed with 30 s rest. Results indicated that only the flexi-dynamic group increased ROM. The authors thus concluded that flexi-dynamic exercises might be superior for pre-performance WU's. They suggested that the superiority of the flexi-dynamic group compared to the speed-dynamic group may be due to a more effective combination of flexibility and velocity.

Differences within these studies are most likely due to the inherent difficulty in investigating the effects of DS on ROM. In addition to the similar problems experienced when comparing SS studies (e.g. number of repetitions), the added variable of the velocity of DS and the manner in which differences are detected (active or passive) increases the difficulty of interpretation substantially. Thus, drawing conclusions on the effectiveness of DS on ROM is problematical.

2.3.5 Proprioceptive Neuromuscular Facilitation

PNF stretching techniques were initially developed for use by physical therapists (63), but have since been adopted and modified by researchers and practitioners. Perhaps the most common PNF stretching routines employed are the passive contract-relax (CR)

and active contract-relax-agonist-contract (CRAC), both of which require a partner. The CR and CRAC techniques both begin with the antagonist placed in a position of maximal stretch followed by a maximal (usually) isometric contraction and relaxation of the antagonist. This step is immediately followed by passive elongation of the antagonist by a partner during the CR technique and concentric contraction of the agonist to assist in antagonist elongation during the CRAC technique (99).

Although the CR and CRAC are the most common terms used to describe PNF techniques, various terms are used. For example, the CR method is sometimes referred to as hold-relax (HR) while the CRAC method is called the agonist contract-relax (ACR). Furthermore, some studies do not distinguish between SS and PNF, referring to SS as a type of PNF stretch (37). Different terms used to describe the same procedures often makes comparisons between the techniques difficult as will be discussed in the following section.

2.3.6.1 Proprioceptive Neuromuscular Facilitation Effectiveness

Research regarding PNF stretching although not as extensive as SS has received considerably more attention than DS. Although researchers have continuously demonstrated an increased ROM following PNF stretching (33;37;87;97;107;122;125), ambiguity remains with respect to an optimal PNF stretching technique. Is the CR or CRAC more effective in producing increased ROM? How long should the isometric antagonist contraction during the CR and CRAC methods be held? How long of a rest period should be given between the isometric contraction and further passive stretch

during the CR method or the contractions of the antagonist and the agonist during the CRAC method? Is it necessary to perform a MVC or would a sub-maximal contraction suffice? These questions and more have received attention since the inception of PNF stretching, yet incongruity remains.

Research has continually indicated that the CRAC technique is superior to the CR technique in increasing ROM (33;37;89) although in one study no difference has been reported (26). Ferber et al. (37) recently tested knee extension ROM during their investigation on the effects of various stretching techniques on knee flexor EMG. Their results indicated that the CRAC method produced a significantly greater increase (29%) in ROM than the CR method. Similar results were reported by Etnyre et al. (33) when they compared ankle dorsiflexion ROM following CRAC, CR, and SS techniques performed on the soleus.

The question is not whether PNF stretching is efficient, but how should it be implemented? Researchers have used various time frames to perform each of the steps necessary to perform PNF stretches. The initial phase whereby the antagonist is placed in the position of maximal stretch is perhaps the most underreported aspect of PNF stretching with the majority of studies failing to describe this process adequately. As is the case of SS, the majority of studies simply state that the muscle was stretched to the point of discomfort (86), to the point whereby tension in the muscle was felt (122), or to the limits of motion (107) without mentioning how long the muscle was kept in this position. Although the duration for which the isometric contractions of the antagonist were performed is often reported, the optimal duration is unknown. The use of 3 (26), 4

(87), 5 (37), 6 (26;87;107), 7 (122;125), 8 (125), and 10 s (95;101) isometric contractions have been reported. Similarly, various relaxation intervals between the antagonist contraction and subsequent passive (CR) or concentric contraction (CRAC) have ranged from immediate (97) to 5 s (25;37;49;125). Furthermore, the time of the second passive stretch (CR) has varied from no mention (97;107) to 15 s (37;49) with one study using a 50 s duration (25). Finally, various times of concentric contraction during the CRAC method have been reported as high as 15 s (76). Thus, it is apparent that the optimal duration to perform each of the stages during PNF techniques have not been clearly specified.

An interesting study by Nelson et al. (95) addressed the question of how long to hold an isometric contraction by examining the impact of three isometric contraction durations (3, 6, and 10 s) during a method similar to the CRAC on shoulder joint ROM. Their results demonstrated that all three contraction durations significantly increased ROM with no significant difference between them. They concluded that the 3 s duration would be most beneficial for the clinician due to its shorter time and effectiveness. Likewise, Cornelius and Hinson (26) examined the impact of three isometric contraction durations (0, 3, and 6 s) on passive ROM of the hip extensors during two different PNF techniques; CR and CRAC. Thus, there was a total of 6 experimental groups referred to as 0-PI, 0-PIC, 3-PI, 3-PIC, 6-PI, 6-PIC, with abbreviations representing the passive maneuver (P), isometric contraction of the antagonist (I), and concentric contraction of the agonist (C). Each stretch was performed 3 times with a 2 min rest between repetitions. Results demonstrated that the only inferior method of stretching used was the

0-PI. All other methods including 0-PIC significantly increased passive ROM suggesting that a passive stretch followed by a concentric contraction of the agonist was just as effective in increasing ROM as the PI and PIC methods with isometric contractions.

Research has also investigated the optimal duration of relaxation post-antagonist contraction in addition to the percentage of MVC necessary to elicit gains in ROM. Gollhofer et al. (44) concluded that the fast recovery of the stretch response to control (< 400 ms) contradicts the common practice of PNF stretching. Thus, any gains in ROM will most likely not be associated with suppressed reflex sensitivity, unless the passive stretch is performed immediately upon relaxation from the antagonist contraction. A similar study by Moore and Kukulka (90) concluded that contraction intensities of 65-75% MVC was sufficient to cause motoneuron (MN) inhibition as measured by the Hoffman reflex (H-reflex) for upwards of 5 s. They concluded that PNF stretching techniques might be useful if performed relatively quickly following the antagonist isometric contraction.

2.3.7 Summary

Suffice it to say, the techniques and their various sub-domains discussed have generated much controversy in the literature and left many questions unanswered. Questions such as which method is superior in achieving ROM without incurring injury, the number of repetitions, sets, intensity, and duration of the stretch needed to elicit optimal gains in ROM, the most effective combination of these variables to design a stretching program, and the transferability of active and passive stretching to increase

static and dynamic ROM. For example, do gains in ROM resulting from passive stretching translate into increased ROM during dynamic movements? This would seem to be a fundamental question due to the possible implications for sporting events and everyday movements. How beneficial would increasing passive ROM be, if there is no transfer to active ROM, arguably the more practical of the two? Thus, it is apparent that there still remain discrepancies as to the most efficient manner to increase ROM, albeit general recommendations can be made. One important recommendation is that stretching once per day will increase ROM for most individuals. Further recommendations include using the CRAC PNF stretching technique to achieve optimal gains in ROM. This method should be conducted by contracting the antagonist for approximately 6 s followed quickly by stretching of the antagonist via an agonist contraction. However, if SS is used, the stretches should last approximately 30 s and be performed 4 times and if DS is utilized, more emphasis should be placed on ROM than on speed.

2.4 Mechanisms of Increased ROM

The notion that the muscle is the main target area during stretching is common (86). However, this statement is vague due to the complex nature of the muscle and its structural components. Increases in ROM following stretching have been attributed to decreases in both active and passive resistance to stretch (86). Passive resistance is attributable to the collagen inherent in connective tissues (mechanical) and at rest is attributable to the parallel elastic component (PEC) while resistance during movement is generated via the series elastic component (SEC) and the contractile components of the

muscle (1). Active resistance on the other hand arises from voluntary and involuntary contractions of the muscle (neurophysiological) (86). While the literature attributes long-term improvements in ROM to alterations in the passive properties of the MTU, short-term changes are often attributed to neurophysiological events (80). Still other research has pointed to an increased tolerance to stretch as the mechanism for acute improvements in ROM with stretching (82). These conclusions however have not been totally warranted and the precise mechanisms for acute and chronic changes in ROM remain obscure.

2.4.1 Passive Resistance

Connective tissue composes up to 30% of total muscle mass (1), thus providing a substantial amount of passive resistance. Collagen, a component of various connective tissues has several important properties including elasticity (the ability of a substance to return to normal length following elongation), plasticity (the ability of a substance to undergo permanent length change following elongation), and a combination of both referred to as viscoelasticity (the ability of a substance to resist change of shape but the inability to completely return to its original shape) (56). As stated, muscle-tendon elasticity consists of a SEC and a PEC. The principle component of the PEC is the perimysium whose function is to distribute forces evenly over the muscle, thus preventing overstretching (80;83). Endomysium however, the main component of the SEC, functions to "transfer force from the contractile component to the tendon and bone in series" (83). Viscoelastic resistance to deformation is dependent on the duration and rate at which the forces are applied (85). Based on these principles, it has been suggested

that high-force, short duration stretching is optimal for elastic changes (acute) while low-force, long duration stretching enhances plastic changes (chronic) (128).

To investigate the mechanisms of increased ROM following stretching, Taylor et al. (123) studied the biomechanical properties of the rabbit extensor digitorum longus and tibialis anterior MTU's. They showed that four SS's were sufficient to alter the viscoelastic properties of the MTU as shown by a decreased peak tension with no further change due to additional stretches. In humans, Fowles et al. (38) stretched the PF for 30 min and demonstrated using B-mode ultrasound an 8 mm increase in fascicle length of the soleus and lateral gastrocnemius. Magnusson et al. (83) concluded that a SS duration of 90 s repeated 5 times was adequate to decrease muscle passive resistance (torque). Similarly, Toft et al. (124) utilized a CR stretching method to demonstrate a decreased passive resistance (up to 18%) of the triceps surae muscle. They also showed that the decrease persisted as a short-term effect lasting for 90 min and long-term effect demonstrated after 3 weeks of twice-daily training.

In opposition to the aforementioned studies, Magnusson et al. (81) investigated the effects of a more 'conventional' SS routine (45 s / 3 repetitions) on the passive properties of the MTU. They found that each 45 s stretch had no effect on the passive resistance to subsequent stretches and thus the method employed had no short-term effect on the viscoelastic properties of the muscle. They further suggested that the increased ROM evident following stretching routines is a result of increased stretch tolerance.

2.4.2 Active Resistance

The acute response to stretching has been attributed not only to mechanical factors but also to neurophysiological components (80). The neurophysiological rationale suggests that the mechanical properties of the muscle are secondary to the reflex activity of the muscle. Thus, according to this model, the increases in ROM evident following stretching are attributable to a "decrease in active resistance produced reflexively or volitionally-induced inhibition or both to motoneurons of the muscles stretched (25)." Thus, if a muscle being stretched is experiencing a decrease in activation due to various physiological mechanisms, an increase in muscle elongation would occur. Assuming this were correct, it would be directly measurable via electromyography (EMG) recording electrodes. Before further explanation however, a brief, basic description of the proposed physiological mechanisms that occur during different stretching procedures is necessary.

2.5 Neurophysiological Basis of Stretching

Muscles contain receptors of various types which if stimulated serve to inform the central nervous system of events at the muscle. The two main types of stretch receptors in the muscle are the spindles and Golgi tendon organs (GTO's). In general, the muscle spindles are capable of detecting both static (length) and dynamic (rate of length change) changes that occur to the muscle (99). More specifically, nuclear bag and chain fibers are located within the muscle spindles (intrafusal) and lie parallel to the extrafusal fiber. These intrafusal fibers are innervated by primary (Ia) and secondary (II) neurons. The Ia neurons are sensitive to static and dynamic alterations in the muscle while the II neurons

are solely static (64). The GTO's are located at the junction between the muscle and the tendon and thus lie in series with the muscle. These receptors are innervated by Ib afferents and are more sensitive to changes in the tension in the muscle than length changes (99).

When a muscle is stretched, the muscle spindles are also stretched which send signals back to the spinal cord via the Ia afferents. In the spinal cord the impulse synapses with an α-MN and an association neuron. The α-MN leaves the spinal cord and causes a reflex contraction of the stretched muscle, a process known as the myotatic (stretch) reflex (1;99). The association neuron in turn synapses with an efferent neuron innervating the antagonist, resulting in a reflex relaxation (reciprocal inhibition). The GTO's when activated however will through the same turn of events function to reflexively relax the contracting muscle (autogenic inhibition) and excite the antagonist. Thus, the muscle that was contracting is now relaxed (inverse myotatic reflex) (1;99).

2.5.1 Static Stretching

Theoretically, as the lengthening of the muscle ceases, the dynamic portion of the muscle spindles stop relaying information to the central nervous system resulting in only the static portion of the muscle spindles remaining active (64). If the stretch is maintained with sufficient tension for at least 6 s, the GTO's will activate resulting in a relaxation of the stretched muscle (113). The combined efforts of decreased excitation via the muscle spindles and increased inhibition of the GTO's on the motoneurons (MN) of the stretched muscle has been proposed to relax the muscle resulting in elongation.

2.5.2 Dynamic Stretching

Ballistic stretching is often not recommended due to the increased likelihood of muscle injury (113) resulting from the initiation of the stretch reflex described above. The stretch reflex is proportional to the amount and rate of stretch of the muscle, hence if a muscle is stretched forcibly and rapidly, a strong reflex contraction will ensue, again increasing the possibility of muscle injury (9;52). Having said this, SS has been shown to increase muscle soreness to the same extent and even more than a similar bout of DS as indicated by increases in creatine kinase (116).

2.5.3 Proprioceptive Neuromuscular Facilitation

The PNF method attempts to take advantage of both autogenic inhibition and reciprocal inhibition to elicit gains in ROM. The initial phases of the CR and CRAC methods begin with a slow stretch of the antagonist, thus attempting to bypass some of the dynamic response of the muscle spindles and attenuating the rate-induced stretch reflex. The ensuing isometric contraction of the antagonist in the CR method attempts to elicit the inverse myotatic reflex, thus producing autogenic inhibition that theoretically will have a lingering effect during the subsequent passive elongation of the antagonist, allowing a further ROM. During the CRAC method the initial two phases are similar to that of the CR method except instead of passive elongation following the isometric contraction of the antagonists, the agonists concentrically contract, producing the added effect of reciprocal inhibition. The combined effects of autogenic inhibition and

reciprocal inhibition theoretically decrease motoneuron pool excitability of the antagonist resulting in an even greater ROM.

2.5.4 Neurophysiological Mechanisms

As previously mentioned, a minimal change or decrease in the EMG signal recorded during stretching would imply muscle relaxation, thus decreasing active resistance to stretch and allowing a greater ROM (86). Mohr et al. (86) compared the EMG firing patterns during SS's in various muscles. They observed low EMG values during all stretches and suggested that either the tendon and not the muscle was being stretched or that the tension produced by the stretch was inadequate to produce a reflexive contraction as a means to protect the muscle. However a paradox has arisen in the literature in which the methods of stretching that produce the greatest gains in ROM are also associated with the highest amounts of EMG (37;89). Thus, the contribution and role of EMG activity of a muscle undergoing stretch remains unknown.

A study by Ferber et al. (37) investigated three stretching techniques (SS, CR, CRAC) on muscle EMG. They reported that the CRAC method resulted in 29-34% more ROM and 65-119% more EMG than the CR and SS techniques respectively. Osternig et al. (97) reported similar results and postulated that the greater EMG was a result of increased co-contraction resulting from increased tension on the joint limits. Furthermore, Condon and Hutton (25) reported a larger increase in EMG and decreased H-reflex amplitude during the CR and CRAC techniques as compared to SS techniques, with no significant difference in ROM. A decreased H-reflex would suggest decreased afferent

excitation of the MN pool to the antagonist resulting from a combination of reciprocal inhibition and autogenic inhibition. However, an increased EMG would imply an increased level of muscle activation. This apparent contradiction was explained by postulating that tonic EMG levels from other neural pathways acting on the alpha MN may have masked the observed effect, resulting in the observed higher EMG levels during the CR and CRAC techniques (25).

As mentioned above, measuring EMG is not the only means of monitoring muscle activation. By monitoring the H-reflex during stretching maneuvers, information regarding the afferent excitation of the MN pool of the stretched muscle may be obtained. Passive stretching has been associated with a decreased H-reflex which in turn has been shown to be positively related to the magnitude of the stretch (47). Gollhofer et al. (44) suggested that stimulation of the PF following isometric contractions resulted in a very fast recovery of the stretch response. The fast recovery (< 400 ms) of the H-reflex would indicate that the afferent excitation of the MN pool would not be suppressed following the isometric contraction thus contradicting the proposed physiological mechanisms responsible for increased ROM during PNF stretching. However, it has been reported that GTO threshold is very high during passive movements (57) implying that submaximal contractions may not produce the same amount of autogenic inhibition as a MVC. In opposition, Guissard and colleagues (47) reported that passive stretching of the muscle following agonist or antagonist contractions produced a greater ROM and greater H-reflex inhibition when compared to the SS alone. Etnyre and Abraham (34) showed similar results with decreases in H-reflex amplitude increasing from SS, CR, and CRAC.

The inhibition was greatly reduced during the CR method with similar values to the SS following 2 s of contraction. The CRAC method however produced a decrease in H-reflex amplitude with only a slight increase after 2 s. Moore and Kukulka (90) reported a depression of the H-reflex amplitude following a brief contraction that reached maximal depression between 0.1 and 1 s and was 90% recovered by 10.05 s, suggesting the potential benefits of PNF stretching techniques. Thus, according to these studies PNF techniques are superior to SS techniques for inducing muscle relaxation and increasing ROM.

An increased ROM following both chronic and acute bouts of stretching have been attributed to passive (1) and active (86) mechanisms. Although controversy remains regarding the exact mechanism responsible for increased ROM, common mechanisms proposed include increased MTU compliance (27;123), decreased neural activation of the stretched muscle (25;90;113) and an increase in stretch tolerance (81).

2.6 Stretching and Performance

Stretching to increase ROM has been accepted as an integral part of health and sport paradigms regardless of fitness or competition levels (i.e. recreational, amateur, elite). Perhaps the main reason for stretching in the general population stems from the apparent relationship between ROM and overall health. Research for example, has demonstrated a positive correlation between ROM and back health (98) indicating the potential importance of stretching to increase the ease of everyday activities. In therapeutic and sporting realms however, stretching is employed to decrease muscle

soreness following activity (54), reduce (109) or prevent (115) the risk of injury and as a method of rehabilitation following injury (62). Moreover, many athletes incorporate stretching routines into their training and WU's in an attempt to improve sport performance (141) albeit there is little scientific evidence to support its benefits (41;67).

In recent years however, the notion that stretching for increased ROM prior to athletic events to reduce the risk of injury or enhance athletic performance has undergone considerable scrutiny. A review by Shrier (114) evaluated the clinical and scientific evidence regarding the use of stretching prior to exercise and concluded that stretching prior to exercise is more likely to cause injury than to prevent it with similar findings reported by various researchers (17;53;100). Additional studies have shown that various stretching routines are sufficient to cause a significant decrease in strength (3;10;38;69;71;105) and power output (27;137). The possibility of an increased risk of injury and decreased strength and power following stretching would thus suggest that stretching prior to exercise may be more detrimental than beneficial to human performance.

2.6.1 Stretching and Running Economy

Research has indicated that there is an inverse relationship between running economy (RE) and ROM of the lower limbs (28;42;60) while the effects of stretching have also indicated increased RE (43) as well as no effect (94). The majority of these findings suggest that an increased flexibility will lead to a decreased RE. Gleim et al. (42) examined the relationship between lower limb ROM and walking and jogging economy

(relative VO_{2max}). They reported that once treadmill speeds exceeded normal walking pace, individuals who were the least flexible in 11 static ROM tests were up to 12% more economical ($r = 0.43$). They concluded by stating "nonpathological musculoskeletal tightness was associated with a decreased steady-state VO_{2max} for treadmill walking and jogging." Similarly, Craib et al. (28) investigated the association between nine measures of limb and trunk flexibility with oxygen costs at two different treadmill running speeds. Results indicated significant correlations between dorsiflexion ($r = 0.65$) and standing hip rotation ($r = 0.53$) with the mean oxygen cost of running (less flexible runners more economical). Proposed mechanisms to explain the negative correlation between flexibility and RE include a greater energy return due to efficient elastic recoil from the previous stride (42) and less active contraction of stabilizing musculature reducing energy costs (28). While these studies point to a negative correlation between ROM and RE, does this necessarily mean that acute increases in ROM resulting from stretching will negatively affect RE?

Godges et al. (43) provided a group of recreationally active males with a SS program and measured RE at 40, 60, and 80% VO_{2max} immediately post-stretch. The stretches resulted in an increased static ROM of the hip flexors and extensors as well as a reduction in the aerobic energy cost of running at the three submaximal speeds. They suggested that the increased RE may have been due to improved co-ordination, greater pelvic symmetry, or a reduced resistance to limb movement. In disagreement with Godges et al. (43), Nelson and colleagues (94) had subjects perform 15 SS's, 3 times a week over 10 weeks. Although the program resulted in an increased static ROM, there

was no effect on the aerobic demand of submaximal running, suggesting that a chronic SS program does not necessarily influence RE. Based on the assumption that one of the mechanisms of an increased ROM is a more compliant MTU, this data would oppose the negative relationship between MTU compliance and RE as suggested by Gleim et al. (42) and Craib et al. (28). However, Magnusson (80) indicated a lack of evidence linking chronic changes in ROM to the passive properties of the MTU. Thus, an increased ROM does not necessarily mean an increased MTU compliance that would negatively affect RE.

2.6.2 Stretching and Jumping Performance

The effect of stretching on jumping performance has received considerable attention in recent years due to the notion that stretching may inhibit maximal force production (23;27;137), which would have major implications within the sporting milieu. Researchers have investigated the effects of DS (93), SS (23;69;137) and PNF stretching (23;137) as part of a WU on jumping performance yielding mixed results. Young and Elliott (137) investigated the effects of static and PNF stretching as well as MVC's on jumping performance. They demonstrated that SS significantly decreased drop jump (DJ) performance while PNF stretching and MVC's had no significant effects. They suggested that the negative influence of SS might result from increased compliance of the MTU, which may be important for fast stretch shortening cycle (SSC) movements. A more compliant MTU would be more inept at transferring muscular forces to the bone from the eccentric to concentric phase during the DJ. Given the fact that PNF stretching is often

considered superior to SS to increase ROM (37;89;107;122), it seemed unlikely that SS and not PNF stretching would decrease force output. Insignificant decreases in DJ performance following PNF stretching were attributed to a countering effect of a lingering facilitation on the activation of the MU's due to muscular contractions and a lack of increase in MTU compliance.

In opposition, Church et al. (23) investigated varying WU procedures on vertical jump (VJ) performance. Subjects performed three different WU methods including a general WU, a general WU with SS, and a general WU with PNF (i.e. CRAC) stretching. Their results indicated that PNF stretching significantly decreased VJ performance although there was no significant difference in ROM between the three groups. The authors attributed the lack of difference in VJ following SS to inadequate time under tension and suggested that the decrease seen with PNF stretching may have resulted from increased autogenic inhibition of MU's. However, the former is difficult to quantify as the researchers failed to adequately depict the SS methods. Although the names of the stretches are specified, the main question to be asked is whether there was equal time spent in the stretched position between the two groups? This may help explain the difference between the findings of this study and the study by Young and Elliott (137) when comparing the SS methodology. In agreement with Church et al. (23), an insignificant decrease in VJ performance (3%) following SS was also found by Knudson et al. (69) who concluded that there was no difference in the biomechanics of the VJ performance that would suggest a more compliant MTU. These results are confounded however by the finding that the majority of the subjects (55%) decreased VJ performance

on average by 7.5%. The difference between the two studies with PNF stretches (non significant versus significant decrease in force output respectively) may be due to the type of PNF stretch used. Young and Elliott (137) used a CR-PNF stretch whereas Church et al. (23) used a CRAC-PNF stretch. The CRAC method is often recommended as a superior method to increase ROM than the CR technique (37;89;97) and thus may have produced a larger increase in ROM due to a more compliant MTU, decreased neural inhibition, or a combination of both.

A recent study by Young and Behm (139) is perhaps one of the most practical investigations of various WU procedures on subsequent performance. They investigated the effects of submaximal running, SS, and practice jumps on jumping performance in an attempt to elucidate an optimal WU procedure. Based on comparisons of concentric jump (CJ) height, their results demonstrated that 4 min of running was better than a control WU or a 4 min run plus SS. In addition, the 4 min run plus SS plus practice jumps were significantly better than a 4 min run plus SS. Thus, SS produced a negative effect while submaximal running and practice jumps produced positive effects on CJ performance. It is also interesting to note that the SS method applied had a relatively low volume that was more representative of a realistic stretching routine. It consisted of two SS's each for the PF and quadriceps consisting of two repetitions of 30 s per exercise for each of the four exercises.

Although it appears as though stretching decreases power output, a direct conclusion based on the results of various studies would be inconclusive and unjustified due to the complexity of research methods. Discrepancies such as the type of stretching

used (SS, DS, or PNF) and their effects may vary according to which type of jumping movement is used. As mentioned in a previous section, stretching alone provides a very complex set of variables that when combined differently may yield conflicting results on factors such as ROM. The most common jumping techniques (countermovement jump, DJ, and CJ) may also prove to be problematical to control. These jumps can be varied based on being unilateral or bilateral, the height from which the drop occurs (DJ), the angle at which the jump begins (CJ), and whether the jump is performed for maximal height, low ground contact time, or a combination of both. Moreover, whether or not an aerobic WU or practice jumps are used in combination with the stretches may result in different effects on the mechanical (SEC, contractile components, and PEC) and neurological (reflexes) properties of the muscle and the movement.

2.6.3 Stretching and Force Output

The effects of chronic stretching routines on performance have suggested that flexibility training can increase strength (132;136) and rate of force development (55). However it is not unreasonable to state that many athletes do not incorporate stretching programs into their training but merely perform moderate levels of stretching prior to athletic events. Thus, the investigation into the effects of acute bouts of stretching on force output is justified. Force deficits following acute bouts of stretching have been reported (3;10;38;71;93;105) ranging from 5 to 30% (138) for up to one hour post-stretch (38) and have been suggested to be joint-angle (91) and velocity specific (92). Kokkonen et al. (71) had subjects perform 5 different SS's consisting of 6 repetitions (3 assisted and

3 unassisted) with a 15 s hold. They reported a significant decrease in 1 repetition maximum forces for both knee flexion (7.3%) and extension (8.1%) following the SS protocol. These decreases were accompanied by significant increases in ROM (16%). They concluded that the major factor affecting force loss might have been a result in decreased MTU stiffness. Similar findings have also been reported following acute bouts of DS (93). Nelson and Kokkonen (93) employed the same method as above with the exception that the stretches were dynamic (the subjects bobbed up and down for 15 s). Similar results were found with a 9% increase in ROM accompanied by 7.5% and 5.6% decreases in knee flexion and extension forces respectively.

Potentially offering more insight into the effects of stretch-induced performance decrement, Behm et al. (10) reported a significant 12% decrease in isometric knee extension MVC force and muscle activation as measured by integrated EMG (20.2%) and the ITT (2.8%). Furthermore, an 11.7% decrease in twitch force was reported with no significant decrease in tetanic force production. These results were similar to those reported by Rosenbaum and Hennig (105) when they reported decreases in passive peak force (5%), force rise rate (8%), half relaxation time (5%), and EMG of the gastrocnemius (16%) and soleus (17%). Similarly, Fowles et al. (38) investigated the effects of passive stretching on the PF over a one hour duration. They demonstrated decreased MVC forces at 5 (21%), 15 (13%), 30 (12%), 45 (10%), and 60 (9%) min post stretching. In addition, motor unit activation and EMG were significantly decreased but recovered by 15 min.

Although it appears acute bouts of stretching induce force loss it must be noted that the stretching protocols used by Behm et al. (10) and Fowles et al. (38) are prolonged and not representative of a common stretching routine employed by athletes. For example, Behm et al. (10) used five different SS's for the quadriceps over a 20 min time frame while Fowles et al. (38) stretched the PF for a total of 30 min. The stretching program employed by Rosenbaum however was only 3 min in total and still resulted in force loss. Thus, the practice of stretching to increase performance has become somewhat of a paradox in that the very method employed to increase performance may in fact be reducing it.

2.7 Stretching and Force Loss: Potential Mechanisms

Various mechanisms have been purported to explain force loss following stretching with the majority of researchers focusing on an increased MTU compliance, neural inhibition, or a combination of both.

2.7.1 Increase MTU Compliance

Although the exact mechanisms responsible for increases in ROM following stretching are debatable, the increase is commonly attributed to decreased MTU stiffness (132;135). The effect of this decrease on sports performance however remains unclear and dependent on whether the contraction is isometric, concentric, eccentric, or a combination as with the SSC (137). Improved performance with a rigid MTU has been demonstrated to be favorable during isometric and concentric contractions (127;133).

Wilson et al. (133) reported that MTU stiffness was significantly related to isometric and concentric performance ($r = 0.57$ and 0.78 respectively). They suggested that a stiffer MTU augments force production via an improved force-velocity and length-tension relationship. A stiffer MTU would be more effective during the initial transmission of force, thus increasing rate of force development. Is the same true however during SSC type movements? Belli and Bosco (12) suggested that the work performed during SSC movements would be enhanced with a stiffer MTU during hopping movements. If MTU stiffness decreases following stretching this may help explain the decreased jump height evident following acute bouts of stretching (23;27;137).

In opposition to benefits derived from increased MTU stiffness, Wilson et al. (132) concluded that increased compliance of the MTU was beneficial. They studied the effects of flexibility training on SSC performance by comparing loads lifted during a movement incorporating the SSC (a rebound bench press) and a movement that did not use the SSC (a purely concentric bench press). Subjects performed four different SS's of the glenohumeral joint twice per week for eight weeks. Following the training, subject ROM was significantly increased along with a significant decrease in SEC stiffness. There were additional increases in loads lifted with the rebound bench press (5.4%) and purely concentric bench press (4.5%), although only the former attained statistical significance. Corresponding with additional research (134), it was suggested that a more compliant SEC increased the ability to store and release elastic energy during the rebound bench press lift. The perception of a positive correlation between force output and SEC compliance is further supported by Walshe and Wilson (126). They compared MTU

stiffness and the ability to perform SSC dynamic jumps from various heights (i.e. DJ's). Results indicated that subjects with musculature were significantly disadvantaged at higher drop heights (DJ80 cm and DJ100 cm) than their more compliant counterparts. In other words, at higher eccentric loads, a reduction in the rebound jump height was apparent. They postulated that the stiffer MTU would have a decreased ability to mitigate the high loads placed on it, thus stimulating increased inhibition during the DJ via the GTO's. This inhibition would override the facilitation effect of the stretch reflex resulting from a bias towards a protective mechanism (126) when high levels of force are placed on the muscle. Thus, incorporation of neural inhibition to reduce performance has also been postulated.

2.7.2 Increased Neural Inhibition

Forces produced can be enhanced if the muscle is first stretched to cause eccentric tension followed immediately by a concentric contraction (73). It has been proposed that the eccentric phase initiates the stretch reflex resulting in increased motor unit activation during the concentric phase, thus greater force production (19). Thus, one could speculate that a decreased force or power output might result from disruption of the stretch reflex (27;96). Mechanisms such as decreased muscle spindles sensitivity (3), and increased activation of reflex inhibitors (group III, IV, and Ib afferents) have all been discussed (3;71).

Behm et al. (10) concluded that following 20 min of passive stretching of the quadriceps, the decreased twitch force (11.7%) indicated a more compliant MTU. They

also concluded however that the lack of change in tetanic force coupled with the decrease in muscle activation levels (2.8% and 20.2%, interpolated twitch technique (ITT) and EMG respectively) suggested that the force decrease (12%) following stretching was more neurological than mechanical in nature. In somewhat a similar study, Fowles et al. (38) reported a stretch-induced force deficit for up to one hour post-stretch of the triceps surae. In addition motor unit activation and EMG were significantly depressed but recovered 15 min following SS. Fowles et al. (38) concluded by stating that the decreased strength initially resulted from impaired activation and contractile force and then by impaired contractile force for the duration of the force deficit. So what mechanisms could be responsible?

One potential mechanism stems from increased inhibitory drive to the MN through increased activation of the GTO's. During the majority of stretching protocols employed, subjects are told to stretch to the point of pain tolerance that theoretically could activate the GTO's. Activation of the GTO's would result in autogenic inhibition of the α-MN's (88). However, it has been suggested that an extremely potent stimulus is necessary to activate the GTO's (57). Furthermore, reflex inhibition following stretching maneuvers has been shown to recover fairly rapidly (ranges from < 400 ms to 10 s have been reported) (34;44;47;90). Thus, it is unlikely that a lingering effect would persist long enough to suppress MVC force and reflex sensitivity during voluntary movements that are typically tested 5-10 min post-stretch (10;38).

Another potential mechansism stems from activation of the mechano- and nociceptors. As mentioned, subjects often stretch to the point of pain tolerance that may

activate the type III and IV afferent fibers. However, Avela et al. (3) concluded that presynaptic inhibition of the Ia afferents due to the stimulation of the group III and IV afferents was doubtful do to a lack of chemical agents present in the blood (i.e. blood lactate and serum CK). In addition, as pointed to by Behm et al. (10) low correlations between pain and inactivation of 0.1 have been reported (11). Furthermore, Fowles et al. (38) noted that the discomfort of a maximal stretch subsides upon relaxation and would not be likely contributors to decreases in MVC force output that normally occurs several minutes post-stretch. Thus it is unlikely that stimulation of the group III or IV afferents contributes to decreased force and activation levels.

A potential mechanism may be elucidated from a study by Avela et al. (3). They investigated the effects of passive stretching of the triceps surae muscle on reflex sensitivity. Following one hour of stretching there were significant decreases in MVC (23.2%), EMG (19.9%), stretch reflex peak-to-peak amplitude (84.8%), and the ratio of H-reflex to muscle compound action potential (M-wave) (43.8%). Thus although neural propagation seemed unaffected (M-wave), afferent excitation of the MN pool (H-reflex) was impaired. They suggested that the decrease in the excitation of the MN pool resulted from a reduction in excitatory drive from the Ia afferents onto the α-MN's, possibly due to decreased resting discharge of the muscle spindles via increased compliance of the MTU. To summarize, stretching would damage the muscle spindles resulting in decreased fusimotor support (i.e. γ-loop support) to the α-MN's responsible for contraction.

In light of the current research it appears as though chronic stretching may serve to increase strength (132;136) and rate of force development (55). However, the effects of acute stretching may have negative implications for sport performance in terms of force or power deficits (23;27;38;137). Suggestions have been made regarding negative correlations between MTU stiffness and movements incorporating SSC performance (132) as well as positive correlations between MTU stiffness and isometric and concentric muscle performance (127). Young and Elliott (137) summarized the current research by stating, "findings indicate that a lower MTU stiffness is advantageous for SSC muscle performance but disadvantageous for isometric and concentric performance." Thus, the implications for human performance are substantial. It appears as though stretching prior to explosive movements may impede performance as well as isometric and dynamic strength.

2.8 Conclusion

The WU is accepted as an essential part of any pre-exercise routine with subsequent performance benefits arising from increased functioning of the cardiovascular, thermoregulatory, metabolic, and neuromuscular systems. Proposed benefits include a reduced risk of injury and further increases in performance via an increased ROM as a result of stretching, one of the key components of a WU. Based on the idea that many of the proposed benefits of pre-exercise stretching are speculative, inquiries regarding the proposed benefits of stretching on performance have come under intense scrutiny in recent years. Researchers for example have established that pre-

exercise stretching may reduce strength and power output, thus decreasing subsequent performance. Researchers have hypothesized both mechanical and neurological alterations to explain the 'performance deficit.'

Based on the notion that stretching may be detrimental to performance, it would seem logical to abandon its use during the WU procedure for activities involving maximal strength and power output. This however would be an unjust recommendation by researchers and practitioners due to the prolonged stretching protocols employed in the majority of these studies. Before stretching can be cast aside as more detrimental than beneficial to performance, scientists must utilize more practical bouts of stretching to investigate its effects on subsequent performance. This would provide athletes and coaches with the practical knowledge they need to develop an optimal WU procedure.

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AN ACUTE BOUT OF STATIC STRETCHING:

EFFECTS ON FORCE AND JUMPING PERFORMANCE

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3.0 Abstract

The objectives of this study were to determine if a typical static stretching (SS) routine decreased force, activation, and power while improving range of motion (ROM). Secondly, the study attempted to compare the duration of the performance decrement to the duration of the augmented ROM. Twelve subjects were tested pre- and post- (POST, 30, 60, 90, and 120 min) SS of the quadriceps and plantar flexors (PF) or a similar period of no stretch (control). Measurements during isometric contractions included maximal voluntary force (MVC), evoked contractile properties (peak twitch and tetanus), surface integrated electromyographic (iEMG) activity of the agonist and antagonistic muscle groups, and muscle inactivation as measured by the interpolated twitch technique (ITT). Vertical jump (VJ) measurements included unilateral concentric and drop jump height, flight to contact time ratio, and relative power. ROM associated with seated hip flexion, prone hip extension, and plantar flexion-dorsiflexion was also recorded. Following SS there were significant overall 9.5% and 5.4% decrements in quadriceps MVC and ITT respectively. Force remained significantly decreased for 120 min (10.4%), paralleling significant % increases in ROM (120 min). Following SS, there were no significant changes in jump performance or PF measures. The parallel duration of changes in ROM and quadriceps isometric force might suggest that force and activation decrements may be related to inhibitory influences associated with increased muscle compliance or joint capsule stress.

Key Words: activation, plantar flexors, power, quadriceps, range of motion

3.1 Introduction

It is generally accepted and recommended by fitness professionals to perform stretching routines following a light aerobic activity as part of a pre-exercise warm-up (WU) (37). Stretching has been demonstrated as an effective means to increase range of motion (ROM) about the joint (2;12) and is commonly utilized by athletes to decrease muscle soreness (15), reduce (27) or prevent (29) the risk of injury resulting from tight musculature, and rehabilitate following injury (18). Moreover, many athletes incorporate stretching routines into their training and WU's in an attempt to improve sport performance (39). In recent years however, the proposed benefits of stretching prior to exercise have undergone considerable scrutiny. It was concluded in a review by Shrier (1999) that stretching is more likely to cause injury than to prevent it with similar findings reported by various researchers (8;14;25). In addition, recent studies have shown that various stretching routines are sufficient to induce strength (1;3;13;19;20;26) and power deficits (11;36) ranging from approximately 5 to 30% (37) for up to one hour post-stretch (13) and have been suggested to be joint-angle (22) and velocity specific (23).

Thus, the practice of pre-exercise stretching to enhance performance has become somewhat of a paradox in that the very method employed to enhance performance may in fact be reducing it. Recommendations to abandon pre-exercise stretching seem premature however in light of the fact that the majority of stretching protocols utilized to investigate performance decrements were prolonged and not representative of commonly employed stretching routines. For example, Behm et al. (3) used five different static stretches (SS's) for the quadriceps over a 20 min time frame while Fowles et al. (13) stretched the plantar

flexors (PF) for a total of 30 min. Furthermore, if pre-exercise stretching were deemed necessary for sporting activities involving maximal force and power it would seem beneficial to determine the duration of stretch-induced performance decrement. Performance decrement timelines could then be employed as a means to formulate recommendations as to when pre-exercise stretching should be performed. For example, if the negative effects of stretching have subsided within 60 min of stretching, but ROM remains increased for 120 min, then one could speculate that the athlete could stretch 60 min pre-exercise without any adverse effects on performance.

Consequently, the objective of this study was twofold in nature: (1) to determine whether a moderate static stretching routine is sufficient to decrease force and power output and (2) to establish a force and power output deficit timeline if performance decrements are observed. Based on the existing literature, it is hypothesized that an acute bout of SS will adversely affect isometric force and jump performance while increasing ROM.

3.2 Methodology

Subjects: Twelve male volunteer subjects (20-44 years, 181.6 cm ± 14.8, 87.3 kg ± 15.2) were recruited from the university population. Subjects were verbally informed of the procedures, read and signed a consent form and a physical activity readiness questionnaire (PAR-Q) (9) prior to participation.

General Study Design: Subjects acted as their own control group. The study consisted five testing days: Day 1 was used for subject familiarization with the testing procedures (i.e. vertical jump techniques, isometric muscle contractions, electrical stimulus); Testing on days 2 to 5 were randomized to test the plantar flexors or the quadriceps muscle group (control and experimental). The testing days were interspersed with a minimum of 24 hours rest. For a schematic representation of the methodology refer to figure 3-1.

Warm-up: Subjects performed a 5 min submaximal WU on a cycle ergometer. The subjects were instructed to cycle at 70 rpm with a resistance of 1 kp, to increase muscle temperature, although this was not measured.

Intervention: Three muscle groups (quadriceps, hamstrings, and PF) of the dominant leg received 2 successive SS's consisting of 3 repetitions each. Each stretch was held for 45 seconds (s) followed by a 15 s relaxation period for a total stretching period of 270s/muscle. The order in which the muscle groups were stretched was randomized. All stretches were held at the position to the onset of pain. Stretches included the standing straight knee and standing bent knee (PF's), the modified hurdler and supine hip flexion (hamstrings), and the prone buttocks kick and kneeling buttocks kick (quadriceps), (figures 3-2 – 3-7 respectively).

Experimental Setup:

Knee extensors (figure 3-8): Subjects were seated on a bench with their hips and knees flexed at 90°. Restraints were placed over the quadriceps, across the hips, and around the chest to ensure consistency of joint angles (90° at hip and knee). The lower limb was inserted into a padded strap at the ankle and attached by a high tension wire to a Wheatstone bridge configuration strain gauge (Omega Engineering Inc. LCCA 250, Don Mills Ontario). Bipolar surface stimulating electrodes were secured over the inguinal space, superficial to the femoral nerve as well as the distal portion of the quadriceps immediately superior to the patella. Surface electromyography (EMG) recording electrodes (MediTrace Pellet Ag/AgCl electrodes, Graphic Controls Ltd., Buffalo, NY) were placed collar to collar (dimensions 3 x 2 cm) over the mid-belly of the vastus lateralis and long head of the biceps femoris. Ground electrodes were secured on the tibia and fibular head.

Plantar flexors (figure 3-9): Subjects were seated in a straight back chair with hips and knees at 90°. Contractions were performed or elicited with their leg secured in a modified boot apparatus (13) with their ankles at 10° of dorsiflexion; the optimal angle for plantar flexion force production (13). Bipolar surface stimulating electrodes were secured over the popliteal space and immediately superior to the gastrocnemius - soleus intersection. Surface EMG recording electrodes (same as previous) were placed over the soleus at the gastrocnemius - soleus intersection and over the mid-belly of the tibialis anterior. Ground electrodes were secured on the tibial shaft.

Electrode Measurement and Preparation: Thorough skin preparation for all recording electrodes included removal of body hair and dead epithelial cells with a razor and abrasive (sand) paper around the designated areas, respectively. This was followed by cleansing of the designated areas with an isopropyl alcohol swab. EMG activity was amplified (X 1000), filtered (10-1000 Hz), rectified (Biopac Systems Inc, Holliston MA), monitored and stored on computer (HP Pavilion N5310). The integrated electromyographic (iEMG) activity was measured over a 1s duration beginning at 250 ms after the first stimulus during the interpolated twitch technique (ITT).

Stimulating electrodes, 4-5 cm width, were constructed in the laboratory from aluminum foil, and paper coated with conduction gel (Signa Creme, Parker Laboratories, Fairfield NJ) and immersed in a saline solution. The electrode length was sufficient to wrap the width of the muscle belly. The electrodes were placed in approximately the same position for each subject.

Force Measurement: All voluntary and evoked torques were detected by strain gauges, amplified (Biopac Systems Inc. DA 100: analog-digital converter MP100WSW, Holliston MA.) and monitored on computer (HP Pavilion N5310). Data were stored at a sampling rate of 2000 Hz and analyzed with a commercially designed software program (AcqKnowledge III, Biopac Systems Inc. Holliston MA).

Evoked Contractile Properties: Peak twitch torques were evoked with electrodes connected to a high-voltage stimulator (Digitimer Stimulator Model DS7H+,

Hertfordshire UK). The amperage (10 mA-1A) and duration (50 μs) of a 100 - 150 volt square wave pulse was progressively increased until a maximum twitch torque was achieved. The average of 3 trials was used to measure twitch amplitude (PT). Tetanic stimulation (100 Hz) was administered at the same stimulus intensity as the twitch for a 300 ms duration.

Interpolated Twitch Technique (ITT) (figure 3-10): The ITT was administered, with two evoked doublets superimposed at 1.5 s intervals on two maximal voluntary contractions (MVC's) to estimate an average superimposed signal (4;5). Furthermore, a potentiated doublet was recorded 1.5 s following the voluntary contractions. Superimposed doublets rather than twitches were utilized to increase the signal-noise ratio. An IT-doublet ratio was calculated comparing the amplitudes of the superimposed stimulation with the post-contraction stimulation to estimate the extent of inactivation during a voluntary contraction (interpolated doublet amplitude / potentiated doublet amplitude x 100 = percentage of muscle inactivation) (4;5). A ratio estimating muscle inactivation rather than activation was calculated, because the superimposed or interpolated force evoked upon the voluntary contraction activates those muscle fibers "not activated" or left "inactivated" by the voluntary command. Rest periods of two minutes were provided between contractions.

Vertical Jumping Tests: All jumps were performed unilaterally with the dominant leg on a contact mat (Innervations, Muncie, IN, USA) and analyzed using a commercially available software program (Kinematics Measurement Systems, Innervations, Muncie, IN, USA). Measurement variables included: jump height, flight to contact time ratio, and relative power. Jumping tests included the concentric jump (CJ) and drop jump (DJ).

Concentric Jump (figure 3-11): The subjects initially stood on the contact mat with knee flexed to 90°. The subject then held the position for a 2 s period at which time they were instructed to jump as high and fast as possible. Subjects left the mat with the knee and ankle fully extended and landed in a similarly extended position to ensure that accurate flight time was recorded. The CJ eliminated any active pre-stretch of the musculature and thus only utilized a concentric contraction. The non-dominant leg was flexed at the knee and maintained in a neutral position throughout the jump to mitigate any potential momentum transfer.

Drop Jump (figure 3-12): The subjects performed a DJ from a 30 cm high platform. The subjects were instructed to place their hands on the hips and step off the platform with the leading leg straight to avoid any initial upward propulsion ensuring a drop height of 30 cm. They were instructed to jump for maximal height and minimum ground contact time to increase the flight to contact time ratio. The subjects were again instructed to leave the mat with knees and ankles fully extended and to land in a similarly extended position to ensure the validity of the test as the software assumes flight time up and down are equal.

Range of Motion: ROM of the hip flexors (figure 3-13), hip extensors (figure 3-14), and plantar flexors (figure 3-15) of the dominant leg were measured.

Statistical Analysis: Data were analyzed with a 2 (TREATMENT: experimental and control) x 6 (TIME: PRE-, POST, 30, 60, 90, 120 min) ANOVA with repeated measures. Subjects acted as their own controls. F ratios were considered significant at $p < 0.05$. If significant interactions were present, a LSD Post Hoc analysis was conducted. Descriptive statistics include means and standard error.

3.3 Results

Quadriceps

Isometric MVC. SS resulted in a significant ($p < 0.05$) 9.5% decrease in MVC force of the quadriceps (data collapsed over testing sessions). MVC force was significantly decreased for the 120 min testing duration (8.4% – 10.4%), thus establishing a force deficit timeline (figure 3-16). MVC did not change significantly in the control condition.

Inactivation (ITT) (figure 3-17). SS resulted in a significant overall 5.4% increase in the inactivation of the quadriceps ($p < 0.05$). There was no interaction effect between condition and time and thus a timeline associated with increased inactivation could not be established. Inactivation did not change significantly in the control condition.

Electromyography. There were no significant iEMG differences between quadriceps experimental and control conditions.

Evoked Contractile Properties. There was no significant difference between the experimental and control condition.

Jump Performance. There were no significant differences between the experimental and control condition for any of the jumping variables.

Range of Motion (figure 3-18). With data collapsed (stretching days combined) SS resulted in a significant increase in sit and reach ROM ($p < 0.05$) lasting 120 min. When compared to the control condition, ROM increased by 10% (POST), 8% (30 min), 7% (60 min), 6% (90 min), and 6% (120 min) post-stretch. Sit and reach ROM did not change significantly in the control condition. There were no significant differences in ROM during hip extension or plantar flexion between conditions.

Plantar flexors

There were no significant differences in any of the variables measured (MVC, % inactivation, EMG, evoked contractile properties, jumping performance, or ROM) between control and experimental conditions.

Reliability

Reliability measures ranged from 0.85 – 0.97 for voluntary isometric measures, 0.79 – 0.93 for vertical jump (VJ) measures, 0.72 – 0.75 for evoked contractile properties and 0.85 – 0.98 for ROM measures.

3.4 Discussion

The most significant findings in this study were that the moderate SS routine resulted in (1) significant 9.5% and 5.4% average decrements in quadriceps MVC and ITT respectively, with MVC force remaining significantly decreased at 120 min (10.4%) and (2) significantly increased hamstring ROM for 120 min (6%). To our knowledge, this is the first study to use a moderate SS routine to demonstrate a force deficit timeline of the quadriceps. The decreased force and activation of various muscle groups following bouts of SS is consistent with other research (3;13). The fundamental difference between the aforementioned studies and the present study however is the duration of the applied SS. Whereas the total SS duration in the present study was 4.5 min per muscle group, Behm et al. (3) stretched the quadriceps over a 20 min time frame while Fowles et al. (13) stretched the PF for a total of 30 min. Thus, even a more moderate SS routine can result in quadriceps isometric force and activation decrements.

Force and Activation

The decrease in MVC force was associated with a significant decrease in ITT, indicating the possibility of a neurological deficit. This was further supported by the

absence of significant changes in PT or tetanus forces, which if decreased would indicate a peripheral impairment.

The stretches used in the current study placed the knee in a position of maximal flexion. When combined with the added pressure of supporting the body weight on the patella of the stretched limb (figure 3-7) there may have been a significant amount of intra-articular knee pressure (16). Further increases in intra-articular pressure may result from dislocating torques during the stretches. As stated by Behm et al. (3), "dislocating torques would be placed upon the tibial portion of the knee joint, by forces pulling or pushing the distal potion of the tibia toward the pelvis." Since substantial forces were placed on the distal portion of the tibia (a rigid structure), a similar dislocating torque would be resisted at the proximal tibia by musculotendinous and ligamentous (i.e. anterior cruciate ligament) structures. Significant stress on the ligaments and joint capsule might contribute to inhibitory actions on the motoneuron (MN). Although a far more severe situation, Behm and St. Pierre (6) demonstrated significant activation deficits in the plantar flexors of patients who had recovered from ankle fractures. They speculated that the lingering inactivation months after the fracture may have been related to joint capsule and ligamentous damage. Whereas there was no measure of damage to the connective tissue in this study, the stress placed on the tissues around the knee may have caused a transient inhibitory effect on the quadriceps.

Surprisingly, there was no significant force or activation decrement detected with the PF. In contrast to the tibia, the foot is composed of multiple bones that could help dissipate some of the dislocating torque effects. Furthermore, the medial and lateral

longitudinal arches would flatten in response to the stretch further buffering the stress on the ankle joint capsule. In addition, the SS's employed for the PF were active whereas the stretches for the quadriceps were passive. Thus, although the subjects were told to stretch to the point of mild discomfort, the tester was unable to determine the relative tension placed on the muscle. It is possible therefore that while both muscle groups were stretched for the same duration, the quadriceps may have been subjected to greater tension. Furthermore, differences between the PF and quadriceps may have resulted from differing fiber compositions. As stated in a review by Smith (29), it has been suggested that slow twitch fibers are more pliable than fast twitch fibers (28). The PF have been documented to possess a higher % of slow twitch fibers than the quadriceps (17;21). Hence, it could be postulated that longer duration or higher intensity SS's may be necessary to incur force deficits in the more pliable PF (higher % of type I fibers).

Stretching may induce a plastic deformation of the connective tissues (i.e. increased musculotendinous unit (MTU) compliance) and/or an altered length-tension relationship. Taylor et al. (30) demonstrated in rabbits that four SS's were sufficient to alter the viscoelastic properties of the MTU as shown by a decreased PT, while Toft et al. (31) demonstrated a decreased passive resistance (up to 18%) of the triceps surae muscle for up to 90 min. In humans, Fowles et al. (13) demonstrated an 8 mm increase in fascicle length of the soleus and lateral gastrocnemius associated with decreased MVC force. This suggested that the decrease in MVC force resulted from an altered length-tension relationship. More specifically, the lengthened fascicles would be at a less than optimal position in the length-tension relationship when retested at the same angle following SS.

If this were the case, an increased MTU compliance would thus be expected to be evident in PT and tetanus values.

Avela et al. (1) investigated the effects of passive stretching of the triceps surae muscle on reflex sensitivity, providing a potential mechanism for decreased MVC force. Following one hour of stretching there were significant decreases in MVC (23.2%), EMG (19.9%), stretch reflex peak-to-peak amplitude (84.8%), and the ratio of H-reflex to muscle compound action potential (M-wave) (43.8%). They suggested that the decrease in H-reflex amplitude resulted from a reduction in excitatory drive from the Ia afferents onto the α-MN's, possibly due to decreased resting discharge of the muscle spindles via increased compliance of the MTU. To summarize, stretching would damage the muscle spindles resulting in decreased fusimotor support (i.e. γ-loop support) to the α-MN's responsible for contraction.

Behm et al. (3) demonstrated that SS of the quadriceps decreased twitch force by 11.7% indicating MTU compliance. They concluded however, that the lack of change in tetanic force coupled with the decrease in muscle activation levels suggested that the force decrease following stretching was more neurological than mechanical in nature. Similarly, Fowles et al. (13) reported a 10% decrease in PT following SS of the PF coupled with a decrease in ITT. They stated that even though full activation of the PF (ITT) was evident by 15 min of recovery, MVC remained decreased for up to 1 hour post-stretch. Thus, they suggested that the early phase of decreased MVC resulted from impaired activation and contractile force, and by impaired contractile force for the force deficit duration (1 hour). As ROM returns to pre-stretch values it would be postulated

that MTU compliance would also return to 'normal,' thus mitigating any decrease in force resulting from increased MTU compliance. This mechanism was unable to be determined however as the protocol lasted 120 min during which force decreases and increased ROM were still present.

Improved performance with a rigid MTU has been demonstrated to be favorable during isometric and concentric contractions (33;34). Wilson et al. (34) reported that MTU stiffness was significantly related to isometric and concentric performance ($r = 0.57$ and 0.78 respectively). They suggested that a stiffer MTU augments force production via an improved force-velocity and length-tension relationship. A stiffer MTU would be more effective during the initial transmission of force, thus increasing rate of force development. Kokkonen et al. (20) reported a significant decrease in 1 repetition maximum forces for both knee flexion (7.3%) and extension (8.1%) following a SS protocol accompanied by significant increases in sit and reach ROM (16%). They concluded that a major factor affecting force loss might have been a result of decreased MTU stiffness.

Thus, although the exact mechanism(s) responsible for the decrease in MVC force are unable to be determined, it is suggested that changes in muscle compliance or stiffness and possibly joint connective tissues may elicit inhibitory influences upon the MN.

Jumping Performance

A seemingly perplexing result of the current study was that in light of decreased force and activation of the quadriceps following SS, VJ variables remained unaffected. Researchers have investigated the effects of dynamic stretching (DS) (24), SS (10;19;36) and proprioceptive neuromuscular facilitation (PNF) stretching (10;36) on jumping performance yielding mixed results. Church et al. (10) investigated varying WU procedures on VJ performance and demonstrated that PNF stretching significantly decreased VJ performance while SS had no effect. Correspondingly, an insignificant decrease in VJ performance (3%) following SS was also found by Knudson et al. (19) who concluded that there was no difference in the biomechanics of the VJ performance that would suggest a more compliant MTU. These results are confounded however by the finding that the majority of the subjects (55%) decreased VJ performance by 7.5%.

In disagreement with the above studies, a recent study by Young and Behm (37) investigated the effects of submaximal running, SS, and practice jumps on jumping performance in an attempt to elucidate an optimal WU procedure. They concluded that SS produced a negative effect on CJ performance. It is also of interest that the SS method applied had a relatively low volume composed of two SS's each for the PF and quadriceps, consisting of two repetitions of 30 s per exercise for each of the four exercises. In support of these findings, Young and Elliott (36) demonstrated that SS significantly decreased DJ performance and suggested that the negative influence of SS might result from increased compliance of the MTU, which may be important for fast stretch shortening cycle (SSC) movements. Belli and Bosco (7) suggested that the work

performed during SSC movements would be enhanced by a stiffer MTU during hopping movements. If MTU stiffness decreases following stretching this may help explain the decreased jump height in other studies (10;11;36).

In opposition to performance benefits derived from MTU stiffness, Wilson et al. (32) concluded that increased compliance of the MTU was beneficial. They had subjects perform flexibility training for eight weeks resulting in increased ROM and decreased series elastic component (SEC) stiffness. Subjects showed increases in loads lifted with the rebound bench press (5.4%) and purely concentric bench press (4.5%), although only the former attained statistical significance. Corresponding with additional research (35), it was suggested that a more compliant SEC increased the ability to store and release elastic energy during the rebound bench press lift. The perception of a positive correlation between force output and SEC compliance is further supported by Walshe and Wilson (1997). They compared MTU stiffness and the ability to perform SSC DJ's from various heights. Results indicated that stiff subjects were significantly disadvantaged at higher drop heights (DJ80 cm and DJ100 cm) than their more compliant counterparts. They postulated that the stiffer MTU would have a decreased ability to mitigate the high loads placed on it, thus stimulating increased inhibition during the DJ via the Golgi tendon organs. This inhibition would override the facilitation effect of the stretch reflex resulting from a bias towards a protective mechanism (32) when high levels of force are placed on the muscle. This may help explain the lack of difference in VJ variables evident in the present study. VJ testing is usually performed using a bilateral model (32;36;38). In the current study however, subjects stretched and performed VJ's unilaterally. Thus, the DJ

height of 30 cm in the current study performed unilaterally would exert significantly greater loads than bilaterally which would benefit from a more compliant MTU. The increased MTU compliance may have the high loads decreasing the inhibitory effect on VJ performance.

Range of Motion

In the present study there was no significant increase in the ROM of the hip flexors although sit and reach ROM increased significantly immediately post-stretch (10%) and remained increased for 120 min (6%). The lack of change in hip flexor ROM could be attributed to the fact that it involved active contraction of the hip extensors. If the SS routine caused a similar decrease in hip extensor force as it did in the hip flexors (quadriceps), it is possible that the hip extensors would be unable to fully support or raise the weight of the stretched leg to a position of maximal hip extension (figure 3-13). While the sit and reach measures the ROM of the hip extensors, similar studies have used the sit and reach as an indicator of increased ROM of muscle groups other than the hip extensors (i.e. hip flexors) (20).

3.5 Conclusion

Moderate SS of the quadriceps resulted in a significant decrease in MVC force output paralleled by significantly increased hamstring ROM (both lasting 120 min) while jumping performance was unaffected. Mechanisms responsible are hypothesized to be an interaction of neuorological and mechanical factors. It is suggested that the insignificant

change in PF MVC force following a similar bout of SS may be related to dissipation of dislocating torques, subjectivity of the stretching protocol, and predominant slow twitch fiber composition.

These findings suggest that a pre-exercise WU incorporating a moderate SS routine impairs force production for up to 120 min. Thus, for activities involving maximal force/power output, it is suggested that SS such as the methods utilized in the current study be avoided at least 120 min pre-performance. Further research is needed to determine if submaximal SS intensities are sufficient to increase ROM without impairing force production.

3.6 Figure Legend

Figure 3-1: Methodological Overview: A schematic representation of the methodology employed. The force and power measurements are indicated via bold and italic lettering respectively. Randomized variables are indicated by brackets (i.e. { }).

Figure 3-2: Standing Straight Knee: The subject is standing against a wall bending one leg at the knee and keeping the other leg extended behind the body. The heel of the back leg remains in contact with the floor.

Figure 3-3: Standing Bent Knee: Similar to figure 3-2 except the knee of the leg to be stretched is placed in a bent position.

Figure 3-4: Modified Hurdler: Referred to as the 'modified hurdle stretch.' The subject is bending their non-stretched leg at the knee, curling the sole of the foot to the inside of the opposite thigh. The subject then leans forward to stretch the hamstrings of the extended leg.

Figure 3-5: Supine Hip Flexion: With the subject lying supine on the floor, the tester holds the leg by the ankle and knee (to ensure full extension) and pushes the leg back towards the subjects upper torso.

Figure 3-6: Prone Buttocks Kick: With the subject lying prone on the floor, the tester holds the leg by the ankle and pushes the ankle back towards the buttocks.

Figure 3-7: Kneeling Buttocks Kick: With the subject kneeling on the floor, the back leg is held by the ankle and pushed towards the buttocks.

Figure 3-8: Knee Extension Apparatus and Setup: Subject is seated on a bench with the hips and knees at 90^0. Restraints as indicated, were placed across the quadriceps, hips, and chest to minimize movement. During contraction, the subject grasps the handrails and maintains contact with the surface of the table at all times. The ankle cuff was secured and attacked via a high-tension wire to the force transducer.

Figure 3-9: Plantar Flexion Apparatus and Setup: Subject is seated with the hip and knee at 90^0. During contraction, the subject grasps the upper prongs on the boot apparatus while maintaining an upright posture.

Figure 3-10: Interpolated Twitch Technique: An interpolated doublet ratio was used to estimate the extent of muscle inactivation during a MVC. % Inactivation was calculated by dividing the superimposed stimuli (SI) during the MVC by the post-contraction potentiated twitch (PT). MVC was calculated by a peak-to-peak measurement (the difference between baseline and the highest point of voluntary force recorded) (4).

Figure 3-11: Concentric Jump: The subject is standing on the contact mat with the knee at 90^0 and the hands placed on the hips. The subject then jumped as high as possible (without any pre –jump dipping motion) ensuring that the knee and ankle were fully extended during lift-off and landing phases. The non-dominant leg remains flexed for the test duration.

Figure 3-12: Drop Jump: Subject is standing on a 30 cm high platform with the hands on the hips. The subject then steps off of the platform to the contact mat and performs an explosive jumping movement.

Figure 3-13: Hip Flexor Range of Motion: The subject is lying prone on the floor with the head resting on the forearms. The stretched leg is then raised in an arc up and back toward the head as far as possible, ensuring a fully extended knee and an ankle position of 90^0. The distance from the toe to the floor was measured.

Figure 3-14: Hip Extensor Range of Motion: Subject is sitting in a modified hurdler position placing the sole of the foot of the stretched limb against the flexometer. With the knee fully extended and palms facing down, the subject bends and reaches forward as far as possible, holding the point of maximal reach for 2 s. The distance between the big toe and the floor is then measured.

Figure 3-15: Plantar Flexor Range of Motion: Subject is sitting on a bench with the foot relaxed. The goniometer is then placed over the specified landmarks (tip of long arm aligned with the head of the fibula; the short arm with the proximal portion of the fifth metatarsal; the axis of rotation with the mid portion of the lateral malleolus). The subject was then asked to perform maximal dorsiflexion at which time the ROM in degrees was measured.

Figure 3-16: Effect of Static Stretching on Quadriceps MVC: Columns represent the maximal voluntary contractions of the dominant quadriceps for the stretch and control conditions. Asterisks indicate significant differences ($p < 0.05$) between stretch and control conditions. Vertical bars represent the standard errors.

Figure 3-17: Effect of Static Stretching on Quadriceps % Inactivation: Columns represent the % inactivation of the dominant quadriceps for the stretch and control conditions. Vertical bars represent the standard errors.

Figure 3-18: Effect of Static Stretching on Hamstrings Flexibility: Columns represent range of motion (sit and reach test) of the hamstrings for the stretch and control conditions. Asterisks indicate significant differences ($p < 0.05$) between stretch and control conditions. Vertical bars represent the standard errors.

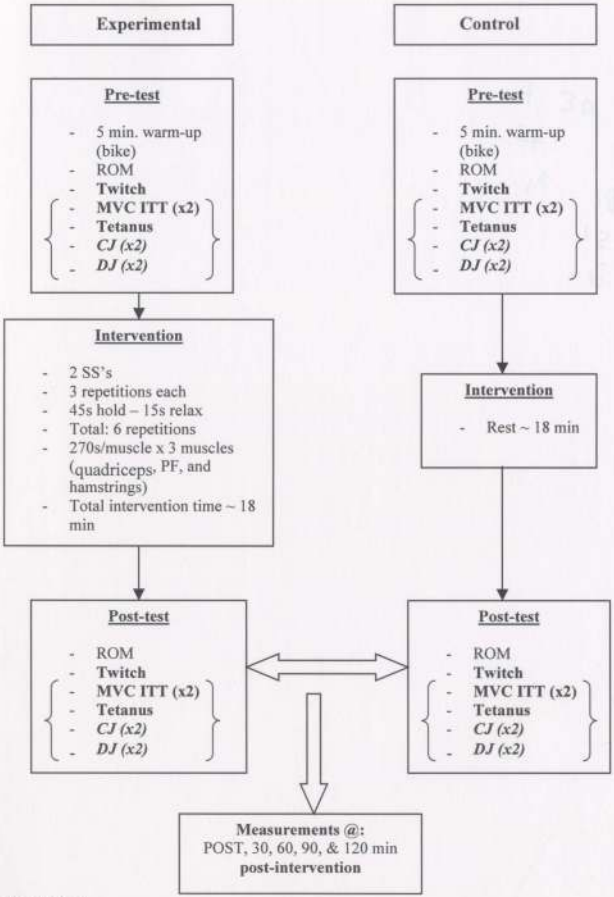

Experimental
Control
Pre-test
- 5 min. warm-up (bike)
- ROM
- Twitch
- MVC ITT (x2)
- Tetanus
- CJ (x2)
- DJ (x2)
Pre-test
- 5 min. warm-up (bike)
- ROM
- Twitch
- MVC ITT (x2)
- Tetanus
- CJ (x2)
- DJ (x2)
Intervention
- 2 SS's
- 3 repetitions each
- 45s hold – 15s relax
- Total: 6 repetitions
- 270s/muscle x 3 muscles (quadriceps, PF, and hamstrings)
- Total intervention time ~ 18 min
Intervention
- Rest ~ 18 min
Post-test
- ROM
- Twitch
- MVC ITT (x2)
- Tetanus
- CJ (x2)
- DJ (x2)
Post-test
- ROM
- Twitch
- MVC ITT (x2)
- Tetanus
- CJ (x2)
- DJ (x2)
Measurements @:
POST, 30, 60, 90, & 120 min
post-intervention

Figure: 3-1

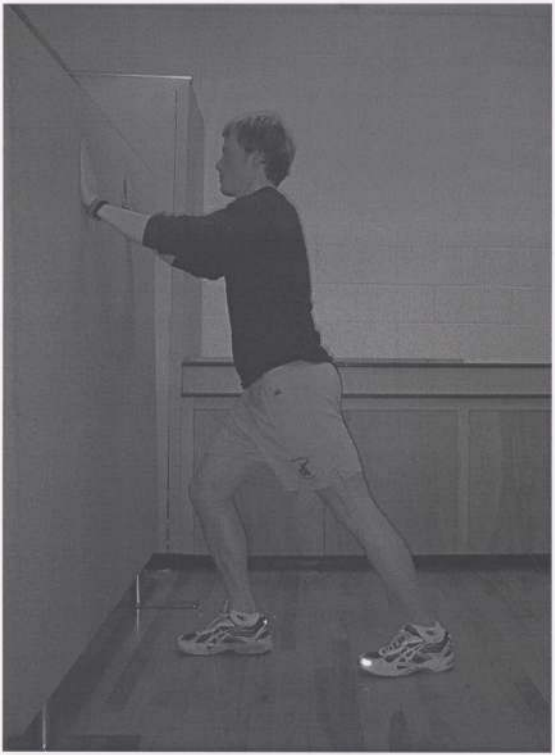


Figure 3-2

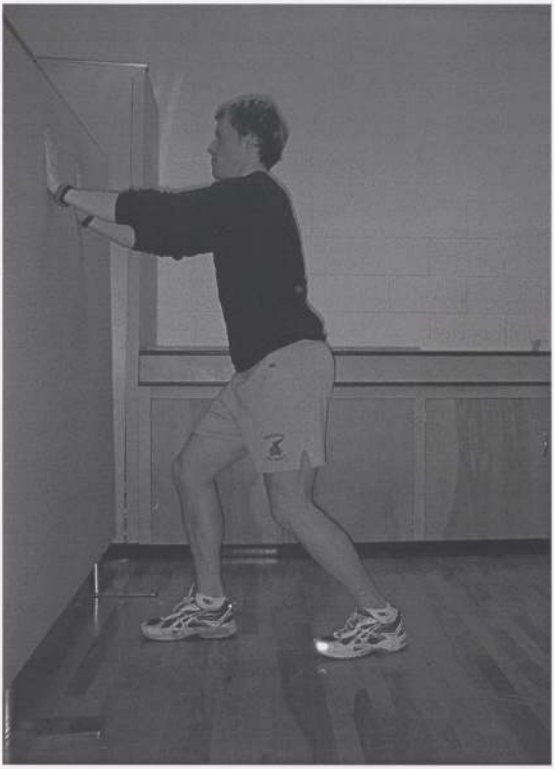
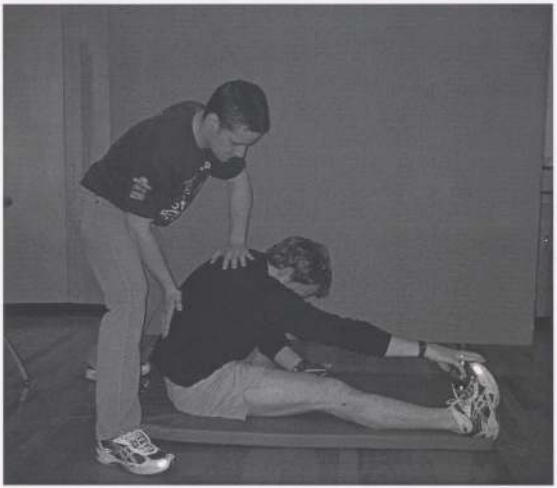


Figure 3-3



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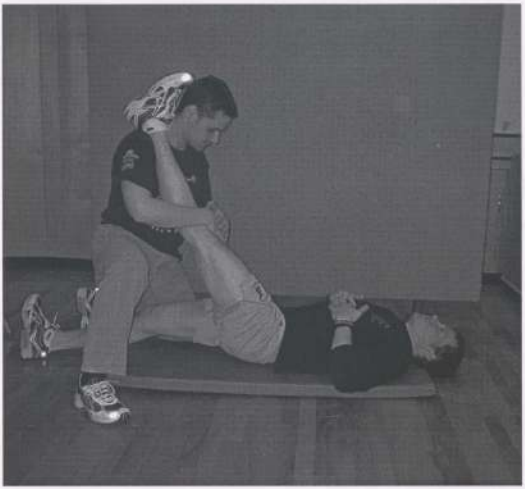

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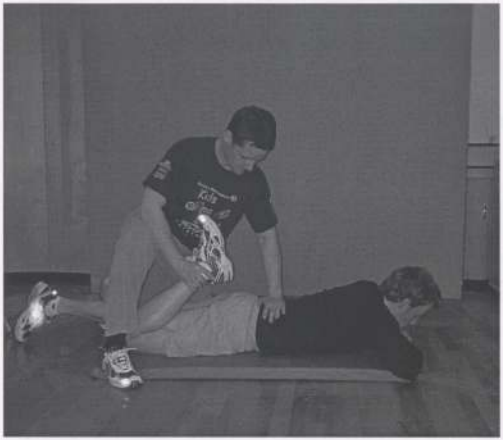

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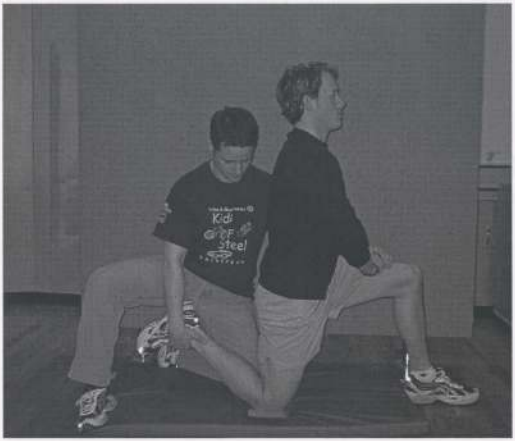

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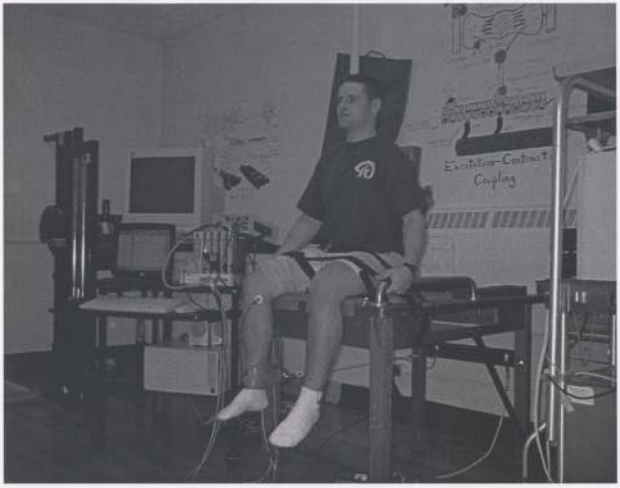


Figure 3-8

Figure 3-9

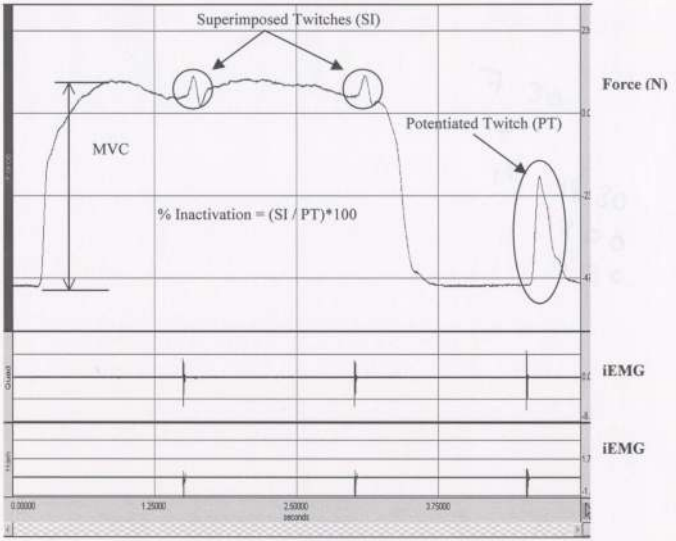
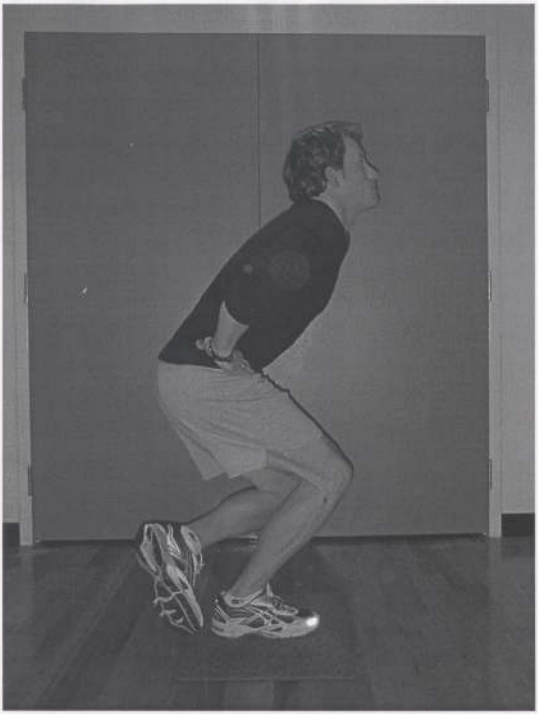

Superimposed Twitches (SI)
Force (N)
Potentiated Twitch (PT)
MVC
% Inactivation = (SI / PT)*100
iEMG
iEMG
0.00000
1.25000
2.50000
3.75000
seconds

Figure 3-10



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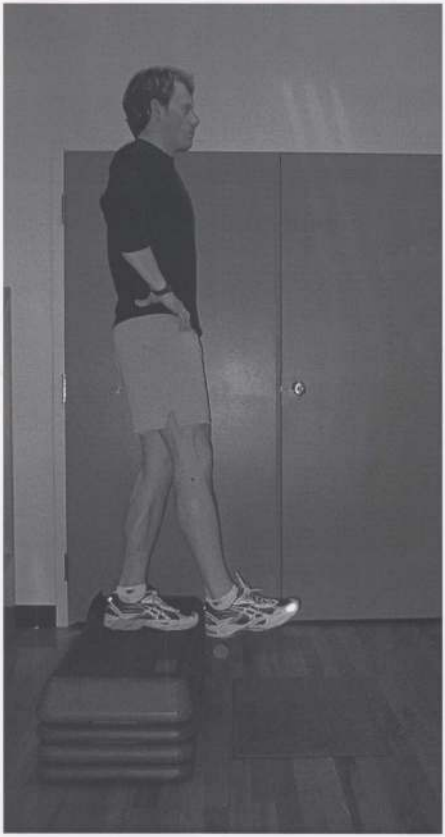

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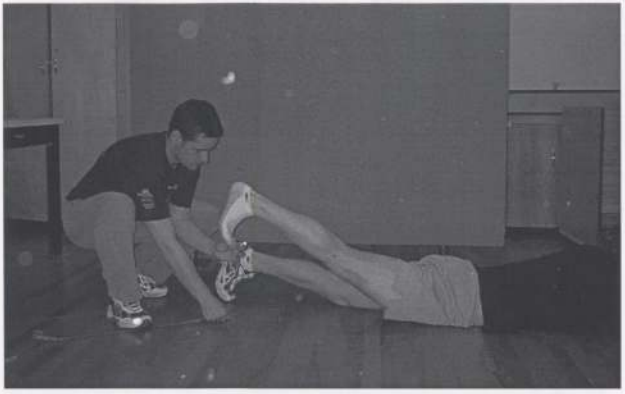


Figure 3-13

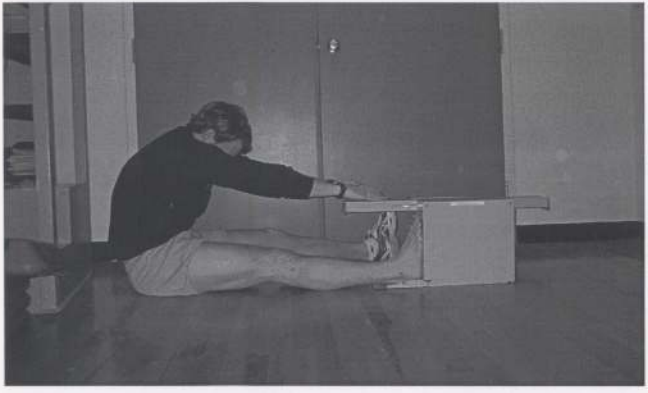
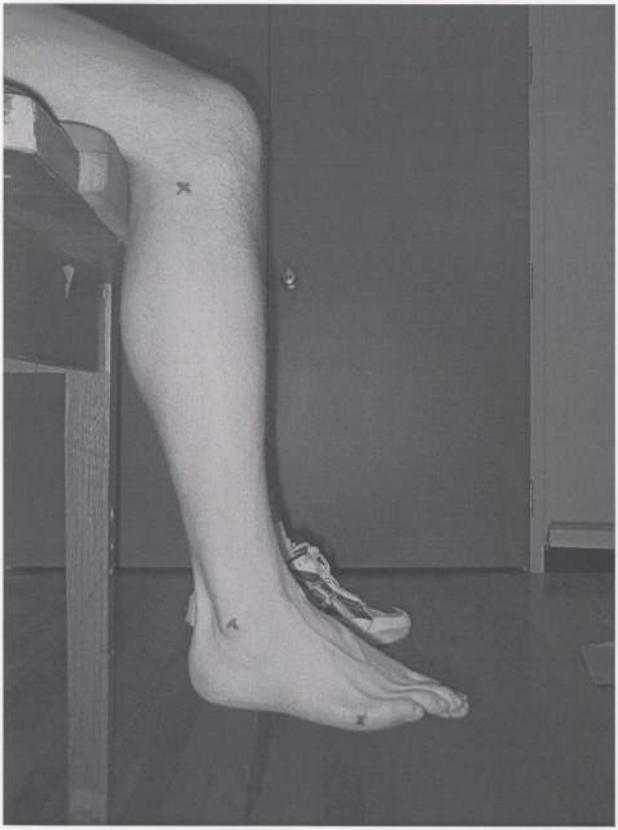


Figure 3-14



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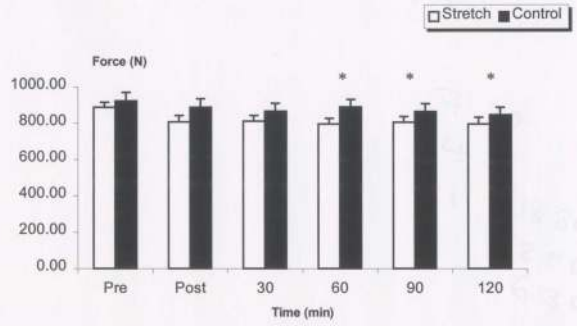

Figure 3-16

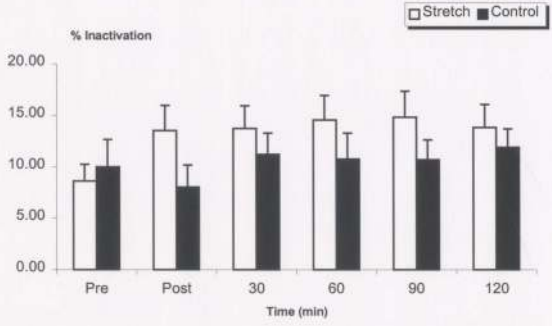

Figure 3-17

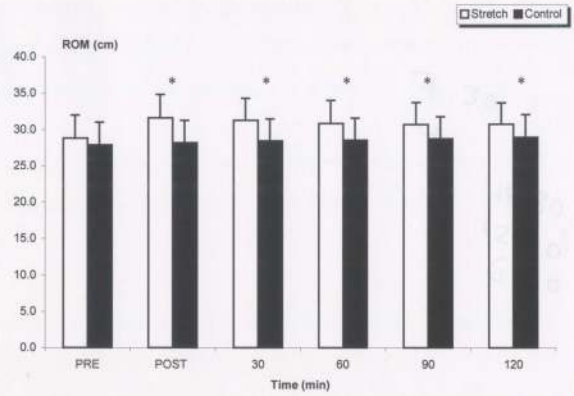
Stretch
Control
ROM (cm)
40.0
35.0
30.0
25.0
20.0
15.0
10.0
5.0
0.0
*
*
*
*
*
PRE
POST
30
60
90
120
Time (min)

Figure 3-18

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