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Hamstrings Contractile Timing in Anterior Cruciate Deficient Subjects

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Advisor: Dr. Tony Bauer, PhD.
Lakehead University

Committee: Jan Perkins, M.Sc.
Health Sciences North-McMaster University

Dr. Moira McPherson
Lakehead University

Paul Stratford
McMaster University

By

© Michael Poling, H.B.P.E., M.Sc.(Cand.), P.F.L.C., C.K.

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CONTENTS

TABLES AND FIGURES

LIST OF TABLES

LIST OF FIGURES

APPENDICES

CHAPTER 1
INTRODUCTION

Anterior cruciate injuries are extremely debilitating injuries for active individuals. It is estimated that 60 to 70 % of all serious knee injuries involve damage to the anterior cruciate ligament (ACL) (Brant, 1989). One of the functional roles of the hamstrings is to prevent the anterior translation of the tibia, absorbing a portion of the anterior shear forces on the ACL (Brant, 1989; Nordin & Frankel, 1989). Timing of the hamstrings contraction during gait provides an important counter force to stabilize the knee. However, if the force occurs too early or too late, stabilization is compromised. Electromyographical (EMG) research analyses demonstrates that the hamstrings increase in activation just before heelstrike (Gauffin & Tropp, 1992; Gronley & Perry, 1984; Phyllis, 1993; Johnstone, 1993). This response may be due to training of the muscle to respond to spatial and temporal cues, causing a compensatory contraction of the hamstrings (Schmidt, 1988; Magill, 1989).

Strengthening the hamstrings may increase the tensile strength and supportive capabilities of the muscle tissue and may help support an unstable joint, but for the support to be effective, the contraction must occur just prior to the anterior shear force acting on the joint. The supportive force caused by hamstrings contraction occurs prior to the anterior shear force caused by heelstrike. In the stable knee, the anterior shear force is countered by the contracting hamstrings action and internal joint translation is minimized (Nordin & Frankel, 1989). Intrinsic changes in knee position and angle can damage the ACL without the addition of external forces, such as a direct blow to the leg. During the swing phase of the gait cycle, an anterior shear force

is produced by the quadriceps acting on the tibial head to extend the leg in preparation for heelstrike. The resulting anterior shear force on the ACL is significant (Tibone & Antich, 1992) and the anterior quadriceps shear force may trigger a stretch reflex, resulting in a hamstrings contraction (Beard et al., 1993; Beard et al., 1994; Schultz et al., 1984; Schmidt, 1988). An increased risk of re-injury to the ACL may be due to a failure in contractile timing of the hamstrings, rather than lack of hamstring or quadriceps strengthening which is traditionally administered during rehabilitation of the ACL deficient knee. (Baratta et.al., 1988; Kålund et.al, 1990)

The majority (65-70%) of ACL injuries result from cutting motions, or motions involving a rapid change in direction of the centre of mass (Brant, 1989; Moore and Wade, 1989). This movement causes a rotational tension as well as anterior shear stress on the ligament fibres without the benefit of muscle tensile forces to compensate during a sudden movement. The parallel nature of the ligament fibres reduces the effectiveness of the ACL to accommodate the joint stress, resulting in possible damage (Van DeGraaff & Fox, 1989). In people with a history of ACL injury, the timing of hamstring muscle contraction is thought to be accentuated, contracting earlier in the uninjured leg, possibly to aid compensation for injury prevention. (Beard et al., 1993)

Another injury mechanism to the ACL is a direct blow to the anterior aspect of the femur. This creates a tensile force on the anterior cruciate ligament. The blow can rupture ligament fibres through extensive bone translation or directly snap them due to the ligament's inability to withstand the tension. (Nordin & Frankel, 1989)

Treatment in cases where subjects plan to continue an active lifestyle may include

surgical intervention. A successful reconstruction usually requires a replacement using either synthetic ligaments or collagenous tissue from another body part such as the patellar tendon. Although surgical reconstruction of a torn ACL can increase stability, limitations exist following surgery such as decreased range of motion, pain and swelling, atrophy of the muscles in the region and persistent joint instability. In most cases, use of the joint post-surgically is rarely rated at the pre-injury level (Tibone & Antich, 1988). Additionally, research has shown that delaying surgical repair may result in faster return to function, possibly due to adaptations by the joint receptors and strengthening of surrounding tissues (Shelbourne & Foulk, 1995). These findings indicate that conservative methods such as gait adaptation and muscle timing correction may be used to augment surgical techniques. In many cases where surgery is not elected, function has been rated low.(Tibone et al., 1986; Arms et al., 1984) Recent research, supporting a non-surgical early mobilization style of treatment, continues to challenge the conventional treatment of arthroscopic examination and reconstruction (Moore & Wade, 1989; Kalund et al., 1990; Beard et al., 1994; Osternig et al., 1995).

PURPOSE

The purpose of the study was to analyze electromyographical (EMG) timing patterns for hamstrings contraction in anterior cruciate ligament (ACL) deficient knees during walking gait patterns.

A secondary purpose is to relate functional measures on a Lysholm scoring scale to adaptations in the knee. Lower functional scores with an injured leg adaptation may indicate a non-desirable effect due to injury or a failed attempt to compensate. Higher functional scores with the adaptation may indicate a desirable effect due to a stabilizing effect.

LIMITATIONS

Subjects in the sample population differ in the mechanism of injury to the ACL. All subjects sustained their injury more than two years and less than six years prior to testing (Timoney et al., 1993; Kalund et al., 1990). This was to ensure that any alterations are not the effect of a recent injury that was still healing, or the result of secondary arthritic changes. Due to the subject inclusion guidelines, the number of subjects for this study was 10. This number of subjects may not be representative of the true ACL deficient population. The ages of the subjects varied between 18 and 35 years old.

Other factors may have affected comparison of results such as the subjects' previous and current activity level, degree of injury and injuries to related structures, such as the hip or ankle. Gait changes to the injured leg may have affected the uninjured leg through compensation, including a change in the muscle activation pattern of the hamstrings prior to heelstrike. To account for this, several testing conditions including a range of speeds and inclines, were used to maximize the gait variations and determine if a significant difference exists between injured and uninjured legs.

The anatomical location of ACL rupture was not determined at the time of injury. This may affect the specific type of instability the subject displays. An ACL rupture at the antero-medial band may result in instability in 90 degrees of flexion while a tear of the posterolateral band may result in instability at 0 degrees of flexion. (Arnheim, 1989; Adrian & Cooper, 1989; Van DeGraff & Fox, 1989)

DEFINITIONS

Accelerometer-an electronic device used to measure motion in any of the three planes of motion.

Anterior cruciate ligament-one of two cruciate ligaments in the knee joint responsible for preventing anterior translation of the tibia.

Artifact-information within EMG data that does not represent muscle activity. Sources include electrical devices, skin movement, electrode movement, cable movement, etc.

Collagenous tissue-tissues, usually connective, that utilize collagen as a building matrix. Collagen is a structural protein used by the body and is flexible, yet extremely strong.

Dynamometer-a device used to directly measure the amount of force exerted by an object.

Electromyography (EMG)-the process of recording electrical changes that occur in a muscle during contraction.

Extra articular-outside the joint capsule

Heelstrike-the point at which the foot first contacts the ground in a gait cycle

Intra articular-see Intra capsular.

Intra capsular-within the joint capsule. The joint capsule surrounds articulating surfaces of the knee and retains the synovial fluid within the joint.

Mechanoreceptors-sensors within a joint that provide information to the central nervous system about the position and movement of a joint, eg. Pacinian corpuscles.

Motor Unit-a unit consisting of a motor neuron, motor end plate, muscle innervation and myofibril within the muscle tissue. The force generated by a muscle is directly affected by the number of motor units involved.

Muscle Action Potentials (MAP)-a series of electrical impulses sent via nerve tissue to the

neuromuscular junction. These "action potentials" cause electrochemical changes in the muscle tissue, initiating a contraction.

Normalized-force or time values expressed as a percentage of the maximum or as a ratio to the maximum. This is used to compare data between subjects.

Pacinian corpuscles-fast adapting mechanoreceptors, providing nearly instant stimulus to changes in tissue condition.

Parallel fibred-tissue -when the fibres within a structure are aligned in one direction.

Proprioceptors -sensory organs that respond to pressure and position.

Reflex arc-a path of electrical stimulus from a sensory nerve to the spinal cord and back to the antagonist muscle. The arc is designed to provide much faster response to potentially dangerous stimuli (ie. heat) than a path to the brain for processing.

Ruffini's organs-slow adapting mechanoreceptors, providing generalized stimulus over a period of time.

Shear force-A loading mode in which a load is applied parallel to the surface of the structure.

Temporal cues-time based cues upon which the body relies for sensory information

CHAPTER 2

REVIEW OF LITERATURE

Related literature was reviewed to include; specific anatomy and physiology of the knee, mechanics and components of gait, and muscle strengthening rehabilitation for Anterior Cruciate Ligament (ACL) deficiency. Electromyographical (EMG) research related to ACL deficiency is the focus of the review.

Anterior cruciate ligament injuries affect gait pattern mechanics, due to the function of the ACL, a dense protein fibre band composed of collagen. The origin of the ACL is the distal, posterior intercondylar aspect of the femur and the insertion, the proximal anterior aspect of the tibial plateau. According to Brant (1989), the ACL is responsible for 85% of the resistive forces that prevent anterior tibial translation. When the ligament is damaged, the role of other support stabilizers, such as the hamstrings, becomes crucial for knee support. Both the origin and insertion of the ligament are intra capsular. Besides the prevention of anterior translation of the proximal head of the tibia, the ligament aids in preventing abnormal rotary motion due to the twisted structure of the ligament fibres. The ACL is divided into three components: the anteromedial, intermediate and posterolateral sections. When the knee is in full extension, as in heelstrike, the anteromedial section of the ligament is in tension. As the knee joint moves into flexion, such as the swing through phase of gait, these fibres relax and the posterolateral fibres of the ligament tighten.(Arnheim, 1989)

Since 1988, there has been an increase in research dealing with the function of surrounding muscle tissue and the support structures for the knee joint. Studies have investigated muscular deficiency and its relationship to injury occurrence and many findings

support retraining the strength of the upper leg musculature. increasing the emphasis on hamstrings retraining in ACL rehabilitation to provide a more stable knee. (Baratta et al.. 1988: Arnheim. 1989; Kannus et al., 1992; Seto et al., 1988)

The quadriceps muscle complex, consisting of the vastus lateralis. rectus femoris. vastus medialis and vastus intermedialis, is responsible for knee extension. (Van De Graaff & Fox, 1989) These muscles originate along the ilium and upper portion of the femoral shaft and insert on the patella and anterior tibial tuberosity via the patellar tendon and ligament. A concentric contraction of the quadriceps creates an anterior shear force during the swing phase of gait on the ACL due to anterior translation of the tibial head. This motion is controlled by the anterior cruciate ligament and the hamstrings. which limit anterior translation through production of a posterior translation force at the tibial head.

The patellar tendon and patellar ligament are the primary attachments of the quadriceps to the tibial tuberosity and act as a pulley system for the quadriceps to attach to the tibia. The patella improves the mechanical torque of the quadriceps tension force by increasing the perpendicular distance between the axis of rotation and point of application on the tibia (Nordin & Frankel. 1989).

The hamstrings muscle group. the knee flexors, consist of the biceps femoris, semitendinosus and semimembranosus. These muscles originate predominately on the ischial tuberosity and insert on both the medial and lateral condyles of the proximal heads of the fibula and tibia. The contracting hamstrings produce a posterior shear force on the head of the tibia during normal walking gait and aid in compensating for anterior shear forces at the knee.(Brant. 1989; Nordin & Frankel. 1989; Moore & Wade. 1989; Gauffin & Tropp. 1992; Staubli & Jakob.

1991)

The gastrocnemius, a two joint muscle functioning at both knee and ankle, originates on the medial and lateral epicondyles of the femur and inserts on the calcaneus. The gastrocnemius plays a minor role in knee function, since the insertion point on the femur creates a small torque arm. It may, however, play a role in knee stabilization during the gait cycle.

Adductor magnus, the only adductor to attach near the knee, originates on the pubic rami and inserts on the linea aspera of the femur and the medial epicondyle of the proximal head of the femur. Adductor magnus is responsible for internal rotation and adduction of the femur. Although the adductors do not insert on the tibia, this muscle group may affect the position of the femur through internal rotation and adduction, and, therefore, may have an indirect effect on the mechanism of injury (Van DeGraaff & Fox, 1989; Arnheim, 1989).

The gracilis muscle originates from the symphysis pubis, inserting on the medial portion of the anterior tibial tuberosity and functions to flex and internally rotate the lower leg as well as adducting the thigh. Although the gracilis can produce posterior shear forces, its effect on knee stabilization is not clear (Van DeGraaff & Fox, 1989; Tokuhiro et al., 1985).

NEUROLOGICAL MECHANISMS

Various sensory and motor structures control stability at the knee joint. Most include some type of proprioceptor. (Magill, 1989; Schmidt, 1988; Beard et al., 1993)

The muscles surrounding the joint, the hamstrings, gastrocnemius and quadriceps, all contain muscle spindles and Golgi tendon organs. These mechanoreceptors function to report

neural feedback for muscle stretch and tension (Schmidt, 1988; Stener, 1959; Zimney, Schutte & Dabezies, 1986). The ligaments of the knee, specifically the anterior and posterior cruciate ligaments, contain Ruffini organs (Ruffini endings) and Pacinian corpuscles. The joint capsule also contains these mechanoreceptors and, with the ligaments, provides essential neural feedback to maintain stability of the joint during the application of external forces and torques (Zimney et al., 1986, Schmidt, 1988). The information provided by these mechanoreceptors aids in protection of the ACL by triggering protective reflexes within the hamstrings. The

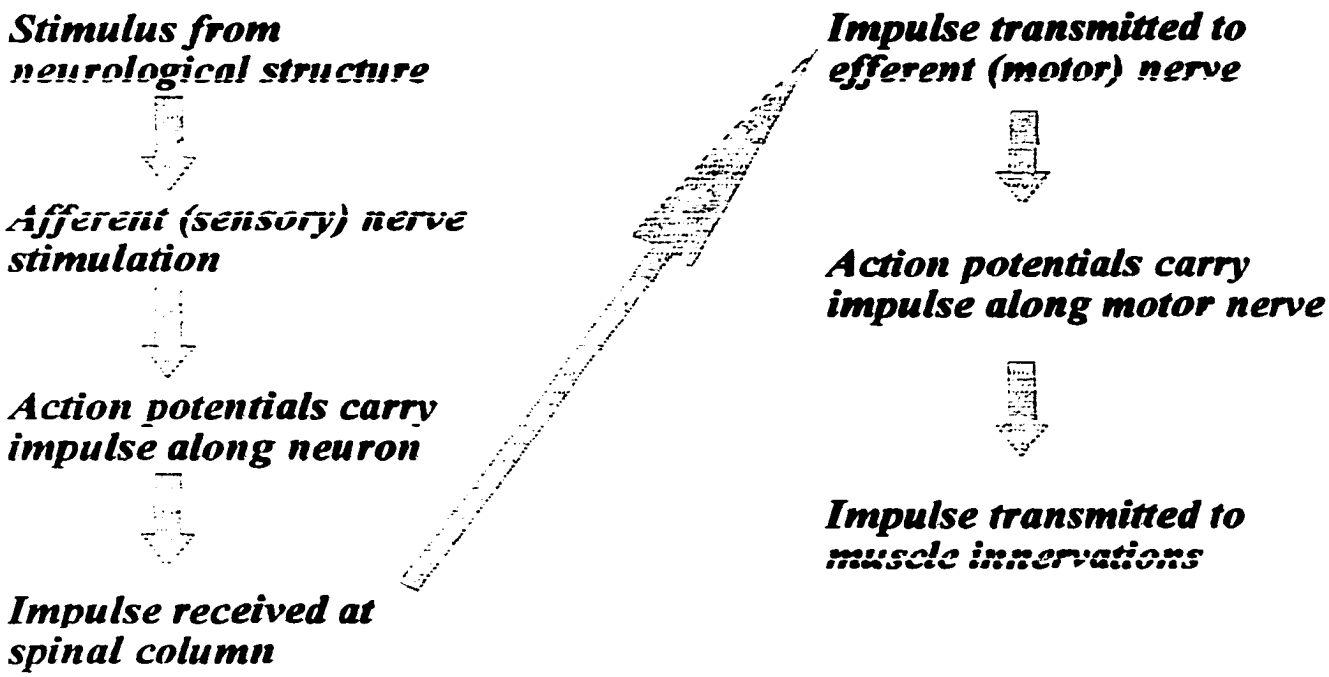

Figure 1: Propagation of a Myotatic Reflex **Modified from Magill(1989)**

propagation of the reflex is reported by Magill (1989) and Schmidt (1988) in Figure 1.

A myotatic reflex, described above, is the simplest reaction and requires 30 to 50 milliseconds (msec) to complete the loop from afferent neuron to muscular contraction. In the knee, the impulse propagation includes a further step within the spinal cord. The impulse is sent

to higher nerve centres for organization and requires a longer period before reaching muscle innervations. This type of reflex is termed "a long loop" and requires approximately fifty to eighty milliseconds (msec.). (Schmidt, 1988; Magill, 1989) These reflexes do not work independently of each other, but in sequence. The initial myotatic reflex does not produce much force in comparison with the long loop reflex. The long loop reflex is also modifiable by instructions, and may be trained. Muscle activation patterns, being long loop reflexes, may respond to training, compensating more efficiently for the forces produced during gait. (Schmidt, 1988; Magill, 1989)

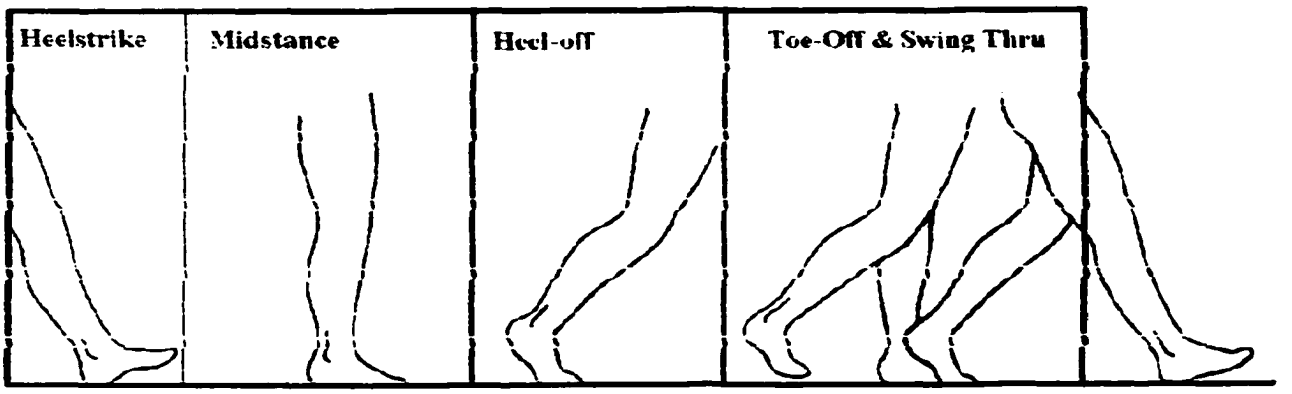

Figure 2: Phases of Gait **Arnheim, 1989**

BIOMECHANICS DURING GAIT

Gait has been defined with various sequences and phases as shown in Figure 2. For the purposes of this study, the five main phases in the gait cycle, include; heel strike, midstance, heel-off, toe-off and swing-thru.(Arnheim, 1989; Tokuhiro et al., 1985) (See Figure 2)

Stance phase of gait includes heel strike, midstance, heel-off and toe-off and makes up about 65% of the total time for one gait cycle. Heel strike is defined as the instant at which the heel first contacts the surface and constitutes 25% of the total stance time. Generally, it is accepted that heel strike indicates the beginning of the gait cycle, though some researchers have used toe-off as the start point.

Shear forces on the ACL at the moment of heel strike are approximately three times that of bodyweight.(Nordin & Frankel, 1989) This force must be controlled or dissipated by the surrounding tissues of the knee and includes the hamstrings muscles. Without this compensation, the reaction force on impact would translate into a greater joint shearing force, allowing anterior translation of the tibia. The proximal end of the tibia is also incapable of compensatory motion unless the ACL was damaged and the strong fixture between the femur and the tibia was broken. Due to the length of the tibial lever, and the strong bony support

structures of the ankle. most of the joint reaction forces during gait are compensated for at the knee joint, rather than the ankle joint.(Nordin & Frankel, 1989; Arnheim, 1989; See Appendix I, Figure 6)

Midstance is defined as the point at which the foot is flat on the ground and force transmission is centred through the knee joint axis. This phase constitutes 40% of the stance phase and knee joint reaction forces are compensated through bony articulation and soft tissue support. Body weight is transferred down the shaft of the femur through the cartilage and articulating surface of the tibia and down the leg. As the femur rests almost entirely on the tibia, little muscle or ligament intervention is required for stability (See Appendix I, Figure 7). Toe-off is defined as the instant the toe leaves the ground. This phase includes 35% of the stance portion of gait and is responsible for forward propulsion. During this phase, a posterior shear force acts on the tibia, due to the position of the femur anterior to the tibia.(Tokuhiro et al., 1985; Hirokawa et al., 1992) During the swing phase period, the majority of the quadriceps contractions occur when the foot is not in contact with the ground. The knee is free of compression forces, though anterior shearing forces remain and may affect hamstrings pre-contraction in preparation for heelstrike, especially if the quadriceps have been strengthened extensively, as in many current knee rehabilitation programs.

KNEE MUSCULATURE RESEARCH

There is an increased emphasis on muscular strengthening techniques for the prevention or treatment of knee injury. Moore & Wade (1989) examined a variety of college level athletes to determine the strength goals necessary to prevent anterior cruciate injuries in athletes. Results indicated that more female athletes than male sustained ACL injuries (20 females injured versus

2 males injured). No difference in joint ligament laxity between men and women was found when measured with a KT-1000 Arthrometer. The study concluded that the cause of the increased frequency of female injury rates was due to inadequate conditioning, or more specifically, strength in the hamstrings and quadriceps. A sample of 50 male and 30 female athletes were tested for bilateral hamstrings and quadriceps strength using a Cybex II Isokinetic Dynamometer. The sample included football, basketball, soccer, track and skiing athletes. The purpose was to define strength goals for various athletes based on bodyweight. Mean relative strength was measured and converted to foot pounds relative to bodyweight. The normalized male and female scores were then compared. The findings indicated that female quadriceps strength rated only 70% of male levels and hamstrings strength was only 63% of the male level. More importantly, the hamstrings/quadriceps strength ratio was significantly less for women, 0.58 and 0.66 for men. Since the hamstrings contribute to joint stabilization, these results were determined to be a major factor contributing to ACL injuries. Specifically, the higher hamstrings/quadricep ratio, the lower the incidence of ACL injury. It was concluded that this was due to increased hamstrings strength counteracting the anterior shearing force produced by the quadriceps.

A study by Seto et al. (1988) examined quadriceps/hamstrings strength, objective ligament stability and functional activity levels five years post surgery in subjects with ACL reconstruction. Twenty-five subjects, with a mean age of 31 years, were recruited and a battery of tests performed. The tests included; a functional activity level survey, an objective ligament laxity assessment involving valgus and varus, anterior drawer and rotational stress testing, and a quadriceps/hamstrings strength test utilizing the Cybex II dynamometer. Those subjects who

had received either intra articular or extra articular ligament reconstructions were noted to have a significantly lower scores in quadriceps strength than their non-operated leg. Those who had intra-articular reconstruction showed significantly lower hamstrings strength scores as compared to the non-operated leg. Subjects' scores on the strength tests were positively correlated with the level of function the subjects had attained five years post surgery. Function was measured by a functional rating scale based on the Lysholm and Gillquist functional rating scale (Tegner & Lysholm, 1984; See Appendix II, Table 2).

Kannus et al. (1992) studied quadriceps and hamstrings involvement in 37 knees with anterior cruciate injuries eight years post injury. The findings indicated that muscle strength between the injured and uninjured knee was not significantly different at slow speeds. However, significant relative strength deficits of 10% were seen between injured and uninjured legs when the speed of movement increased. The authors state that this may be due to atrophy of type II fast twitch muscle fibres. This finding was confirmed by McNair and Wood (1993) in a study of 17 subjects to compare quadriceps strength in ACL deficient and uninjured knees when tested for knee extension at 180 degrees per second on an isokinetic KinCom knee dynamometer. Results showed a significant difference between limbs in the strength of the quadriceps muscle. ACL deficient limb strength was significantly lower compared to the uninjured limb. The authors concluded that type II muscle fibre atrophy had occurred.

A study by Arms et al. (1984) demonstrated significant strain in the ACL in vivo, due to quadriceps contraction between 0 and 45 degrees of knee flexion. Researchers used a strain transducer attached directly to the anterior cruciate ligament to monitor ACL tension and strain during resisted knee extension while the femur was stationary. Results indicated that no

significant strain to the ACL occurred beyond 60 degrees of flexion, but that strain was maximal between 0° and 45° of flexion. Heelstrike occurs with nearly full extension of the knee, with high tension levels on the ACL prior to heelstrike. Heelstrike provides an additional shear force. This anterior shear force produced by heelstrike increases the translation of the tibia and must be compensated for by the ACL and hamstrings. The authors stated that quadriceps contraction can produce significant strain on the ACL by placing an anterior shear force on the tibia, drawing it forward during quadriceps contraction. It was concluded that quadriceps force can affect joint stability adversely in 0 to 45 degrees of knee flexion, hampering the function of the hamstrings in limiting anterior tibial translation.

Hirokawa et al. (1992) demonstrated, on 12 cadavers, that quadriceps contraction results in significant anterior translation of the tibia from 0° to 80° of flexion. A 12 kg load on the quadriceps muscle caused a 6.3 mm anterior displacement of the tibia at 30° of flexion. The range of anterior translation was between 15° and 45° of flexion and quadriceps contraction was found to also produce approximately 7° of internal rotation of the tibia between 0° and 90° of flexion. As internal rotation also places additional stress on the medial fibres of the ACL, there is evidence for a potentially high combination of forces resulting from the quadriceps contractile force (Hirokawa et al., 1992; Mercer & Gleeson, 1995). The fact that the study was performed on cadavers limits the extrapolations that can be made with regards to the effects on live anterior cruciate ligaments. This study shows a discrepancy from the Arms et.al. (1984) study in the strain produced beyond 60° of knee flexion. This may be due to a hamstrings co-contraction in the Arms et.al. (1984) study, as it was in vivo. Effects of tissue preservation and length of time since death may both affect the tension, loading mechanics and force absorption capabilities of

tissue (Guyton, 1991; Van DeGraaf & Fox, 1989).

The results from these studies support the idea that a focus on quadriceps strengthening, often used in clinical treatment of ACL subjects, may be contraindicated and hamstrings strengthening the primary focus.

Researchers have also reported a decrease in quadriceps strength in ACL deficient knees and have hypothesized that this may be due to a stabilization reaction to minimize quadriceps shear force and increase hamstrings stability relative to quadriceps strength.(Tibone et al., 1986) Stabilization, therefore, may not be due simply to increasing hamstrings strength and adjusting timing, but may also depend on adaptations by the quadriceps.(Hirokawa et al., 1992; Tibone et al., 1986; Berchuk et al., 1990)

O'Conner (1993) studied a computer based model of the knee joint and found that co-contraction of the hamstrings and quadriceps resulted in an exact balancing of forces only at an angle of 22° of flexion. At angles lower than this critical flexion angle, the forward pull of the patellar tendon and quadriceps was greater than the backward pull of the hamstrings, thus producing an anterior shear force leading to anterior translation of the tibia. At flexion angles higher than 22°, O'Conner found that the backwards pull of the hamstrings was greater than the anterior shear force of the quadriceps. In addition, results indicated that simultaneous contraction of quadriceps, hamstrings and gastrocnemius can completely unload the cruciate ligaments at flexion angles higher than 22°. The author suggests that since muscle support can completely unload the cruciate ligaments, hamstrings strength training could be used as a non-surgical method of treatment to help stabilize the knee at higher flexion angles. There may be a question of the validity of O'Conner's computer based model, given the extreme stresses and

wide variety of stress combinations placed on the knee during daily activities. Simple flexion and extension is not adequate to accurately simulate normal knee range of motion. Internal and external rotation stresses as well as compressive and shear forces are placed on the knee in an assortment of instances and combinations.(Brant, 1989; Staubli & Jakob, 1991) O'Conner's study is limited in that it does not quantify these forces nor the external loads on the knee, with the exception of body weight. The musculature is identified in this study as providing a significant support mechanism for the cruciate ligaments.

The literature suggests that there are significant effects of muscular interaction with the knee joint in lowering the amount of strain on the anterior cruciate ligament, especially at angles less than 45 degrees of flexion, such as during heelstrike. The following section examines the literature in regards to the timing of this musculature during the gait process.

ELECTROMYOGRAPHY

Electromyography measures the electrical activity produced during a muscle action potential (MAP) within a motor unit. Varying levels of contraction and timing of contraction can be measured using electromyography. Electrical characteristics differ between subjects, therefore, for comparative purposes, processed EMG is usually normalized or measured as a percentage of the subjects' recordings for a maximum isometric contraction.

Tibone and Antich (1988) studied 11 subjects two years post ACL reconstruction. All subjects had reconstruction utilising patellar tendon grafting from the same leg. All subjects were tested preoperatively for base-line measures and returned two years later to undergo follow-up tests. The tests consisted of a subjective assessment and functional analysis, an objective examination for ligament instability, Cybex II dynamometer testing for hamstrings and quadriceps strength, hamstrings and quadriceps EMG, radiographic assessment for degenerative changes in the joint, measurement of anterior shear force displacement of the tibia using a knee arthrometer, and force plate and film analysis while performing cutting motions in a lab setting. Both the injured and uninjured knees were tested and compared. Subjective assessment rated the operated knee at 83% of pre-injury status from 53% before reconstruction. Six out of 11 subjects were able to return to full competition with or without a brace with no further injury. EMG values were normalized and compared. Quadriceps EMG activity was 85% that of the non-injured knee, this difference being significant. Hamstrings EMG values did not significantly differ between injured and non-injured legs. The fact that hamstrings values remained the same between injured and non-injured legs and that quadriceps strength was found to be only 85% that of the non-injured leg may indicate that the increased hamstrings strength relative to quadriceps

had a significant stabilizing effect on the knee joint. This may be the reason for 6 out of 11 subjects returning to full activity with no further injury. The study did not indicate whether or not the highest hamstrings EMG values were recorded by those subjects who had returned to full sport competition.

Gauffin & Tropp (1992) used integrated electromyography (IEMG) to analyse nine subjects with old ACL injuries during a one-legged jump performance. In addition, a subjective survey of functional activity and static laxity tests were performed bilaterally using a 90 N load applied to the proximal end of the tibia in both anterior and posterior directions. Muscle strength testing utilising a Cybex II dynamometer was performed in conjunction with movement analysis using light emitting diodes placed on the subject. The subject was video taped with simultaneous EMG and Kistler force plate measurement. Subjects had not had any type of reconstruction with the exception of meniscal injuries. Arthroscopic examination showed complete ACL tears in all subjects. Results demonstrated changed patterns of muscle activation timing as well as joint movement at the knee between the injured leg and the non-injured leg that have adapted to decrease shearing forces on the ACL. Peak quadriceps Percent EMG (%EMG) was significantly reduced in the injured leg while hamstrings peak %EMG remained the same as the non-injured leg. These results indicate that the ratio of hamstrings strength to quadriceps strength increased in the injured leg. A net increase in the hamstring/quadricep ratio may more effectively control tibial translation

Stener (1959) was the first to show that articular nerves responded to mechanical stresses in cats. By looping a thread underneath the medial collateral ligament (MCL) in a feline subject, Stener demonstrated that by pulling transversely, action potentials could be elicited in the

afferent branches of the nerve servicing the MCL. Moreover, it was also demonstrated that the greater the transverse force on the ligament, the greater the number of action potentials that were elicited in the nerve.

Shultz et al. (1984) examined human cruciate ligaments by microscope and discovered small mechanoreceptors within the ligament, all of which attached to a single afferent axon that then exited the joint capsule. Their conclusions suggested a possible protective reflex arc between the anterior cruciate ligament and the hamstrings muscle group.

A more detailed analysis was performed by Zimney, Schutte and Dabezies (1986) on human cruciate ligaments obtained from cadavers. Microscopic analysis confirmed the presence of Ruffini's organs and Pacinian corpuscles within the cruciate ligaments. These structures are mechanoreceptors and their main function is to provide feedback regarding the tension within tissue. It was theorized that the cruciate ligament itself was able to provide feedback regarding it's condition rather than relying on muscle structures and joint capsules to for feedback as once thought. These histological studies are significant in that they demonstrate the potential feedback loop by which muscular support for the joint is initiated.

Solomonow et al. (1987) tested two groups of subjects during a maximal extension exercise of the knee performed on a Cybex II dynamometer, monitoring quadriceps and hamstrings EMG signals. One group consisted of six subjects with no previous history of knee injury and EMG was recorded during a maximal effort of extension and the Mean Absolute Value (MAV) was used to compare to the EMG from a slow (15°/sec) extension. Results showed a low level of hamstrings EMG activity (approximately 7% of the Mean Absolute Value) during the slow extension trial. Solomonow et al. then tested 12 subjects with

arthroscopically confirmed ACL deficiencies. During maximal extension trials, EMG output showed a significant decrease in quadriceps EMG activity and a significant increase in hamstrings EMG activity at 42° from full extension. This increased hamstrings activity was attributed to anterior translation of the tibia at 42° and a protective mechanism elicited by the mechanoreceptors in the joint capsule.

A follow up study by Baratta et al. (1988) examined the quadriceps and hamstrings EMG output in three subject groups on a Cybex II dynamometer. Group One consisted of non-athletic individuals with no prior knee injury and Group Two elite level athletes involved in jumping sports with no history of hamstrings specific exercise. Group Three consisted of elite level athletes involved in jumping sports training with a regular regimen of hamstrings specific strengthening exercises. EMG analysis of flexion and extension at 15° per second revealed decreased hamstrings muscle activation patterns in Group Two. The results are attributed to the fact that Group Two's training regimen was quadriceps dominant with little or no hamstrings training involved. Recommendations included a hamstrings strength training program to improve the antagonistic activation pattern of the hamstrings muscle group and to create better balance between anteroposterior forces at the knee.

Barrack, Skinner and Buckley (1989) studied proprioception in the knees of 11 subjects with arthroscopically confirmed ACL deficiencies showing an average of 6.6 mm of anterior translation on a KT-1000 arthrometer. Subjects were blindfolded and each leg was placed in an air splint. A motor driven system was then used to move the subject's leg from the starting position to 40° of flexion and the subjects were asked to press a button when they felt a change in their knee joint position. The study was double-blind in that the researcher was not aware

which knee was injured. Both injured and uninjured knees were tested and compared. Results showed a significant difference between control and ACL deficient knees. Control knees were able to detect 2.57° of joint movement whereas ACL deficient knees required 3.53° of movement to detect motion. These results demonstrate decreased proprioception on the ACL deficient leg. The methodology of this study raises the question: which structure is responsible for joint motion detection? Proprioceptors within the joint capsule, muscle stretch receptors in the surrounding muscle tissue, superficial nerve stimulation in the stretched skin or Golgi tendon organs within the patellar, biceps femoris and pes anserinus tendons are all capable of providing sensory feedback. In addition, sensory feedback provided by the subject is cognitive and non-reflexive, whereas hamstrings co-contraction during walking has been shown to be reflexive. The response times to anterior joint translation by the hamstrings may indicate that an antagonistic response is likely reflexive. It is questionable that the Barrack et al. (1989) study was measuring the reflex arc between the anterior cruciate ligament and the hamstrings as measured response time was much greater. This is likely due to the fact that sensory information must be processed in the brain to provide a cognitive response (Schmidt, 1989). It is likely that the processed information from the brain does not accurately represent the timing of a true reflex which is processed at the spinal cord level.

A study using EMG to examine ten subjects with unilateral ACL lesions showed a significant decrease of quadriceps and gastrocnemius activity plus an increase in medial hamstrings activity during cutting movements (Branch, Hunter & Donath, 1989). Subjects were braced with a Lennox Hill knee brace. Braced trials demonstrated decreased quadriceps and hamstrings activity when compared to unbraced trials. Branch et al. concluded that there was a

dynamic compensation of anterior tibial translation by the hamstrings and that functional bracing did not proprioceptively stimulate muscle contraction to improve joint stability while in a cutting motion. (Tibone & Antich, 1992)

Kålund et al. (1990) studied nine subjects with total isolated ACL ruptures and nine control subjects at least 2 years post-injury and who still suffered from incidents of "giving way" or knee collapse. Results showed no significant differences between control and ACL deficient subjects during horizontal walking. During treadmill walking on a 25° incline, significant differences were observed in the lateral and medial hamstrings between control and experimental groups. ACL deficient subjects were observed to have hamstrings activation significantly earlier than control subjects relative to heel strike, eliciting muscular activations 60 to 117 ms earlier than non-injured knees. Kålund et al. (1990) explained this significance as an attempt by the hamstrings muscle group to regain stability of the joint.

Kålund et al. (1990) utilised a subject pool of nine subjects with isolated total ACL ruptures who suffered from giving way during daily activities or recreational sport. Subjects experienced frequent episodes of knee collapse, two to three years after the injury, and demonstrated earlier muscular onset times in their ACL deficient knee. The authors state that the subjects are exhibiting "signs of stability", yet they have collapses during daily activities and sports. These statements are in contradiction, as successful rehabilitation should allow normal daily activities without significant instability. (Tibone and Antich, 1986)

Sinkjær & Arendt-Nielsen (1991) found onset times to be earlier in ACL-deficient subjects in all five muscles acting on the knee joint. They also found the duration of muscle involvement to be prolonged in ACL-deficient knees. The study used EMG to examine

hamstring, quadriceps and gastrocnemius contraction times relative to heelstrike. Contraction was defined as an EMG amplitude increase equal to the standard deviation during a period of non-usage of the muscles. Values were normalized to percentages, 100% representing one gait cycle.

In 1993, Beard et al. studied 30 subjects with unilateral ACL deficiencies by measuring the timing of hamstrings reflex contraction. Methods included attaching an accelerometer to the anterior aspect of the tibia to measure initial tibial movement. The posterior aspect of the lower leg was then subjected to a 100 Newton(N) shear force in the anterior direction, the direction in which the ACL would normally prevent movement. EMG was used on the hamstrings muscle to determine the time of contraction following the 100 Newton(N) force. Results indicated that the uninjured limb responded nearly twice as quickly as the injured limb. Mean time between initial tibial movement and hamstrings contraction was 52 milliseconds (ms) in the uninjured leg and 99 ms in the injured leg. In addition, a correlation ($r=0.62$, $p<0.05$) was found to exist between the onset of hamstring contraction and the frequency of "giving way" or functional instability based on subjects score on a Lysholm scale. Average Lysholm score was 65 for ACL deficient subjects and 100 for control subjects indicating a decreased perception of function when onset of hamstrings contraction was later. Beard et al. attributed this decrease in self perceived function to a loss of proprioception. This study involved only a static model of a dynamic system and is limited in its generalizability.(Gauffin & Tropp, 1992) It is well established that a static position hampers proprioception of joint receptors and muscle spindles (Guyton, 1991).

Jennings & Seedhom (1994) repeated the Beard et al. (1993) experiment but were unable

to produce a significant difference between injured and non-injured legs. Although the Jennings & Seedhom study states: "Our experiments were identical to those of Beard et al.. . .", the methods were not identical. A Cincinnati functional scoring system was used in place of the Lysholm functional scoring system which may have affected the rating of a subject's functionality. Only 11 subjects were investigated by Jennings and Seedhom, being only one-third that of the original Beard et al. (1993) study, leading to questions in generalizability. The study incorporated subjects who had been diagnosed by arthroscopy eight months to ten years post injury. The large variation in post-injury periods is a limiting factor which may influence the data. Beard et al. (1993) utilized only subjects who had obtained their injury less than eight months before arthroscopic examination. The subject pool was much more homogeneous in the Beard et al. study. Analysis of the results of Jennings' et. al. 1994 study shows that, although the researchers do not report a significant difference, the high standard deviations indicate a wide variance in scores. This may indicate erratic test results obtained between tests and introducing a reliability question.

The results of studies by both Beard et al. and Jennings et al. are questionable in that the use of a static position to elicit a reflex reaction based on proprioception principles does not allow normal mechanoreceptor stimulation. The inhibited proprioception by the static position may mask strength deficits in a joint (Guyton, 1991). Muscle spindles and joint mechanoreceptors, such as Ruffini organs and Pacinian corpuscles, require movement to provide feedback. During static positioning, motion and tissue elongation are minimal, also minimizing the amount of receptor feedback. It is likely that hamstrings coactivation preventing anterior translation of the tibia is dependent upon the same mechanoreceptors and will be affected by the

decreased stimulation and feedback.

Recently, Osternig, Caster and James investigated the coactivation patterns of the biceps femoris during maximal flexion and extension of the knee on an Orthotron isokinetic dynamometer. Two subject groups, ACL deficient subjects and uninjured subjects with no previous knee injury, were tested. All injured subjects had reconstruction of the ACL with one exception. Measurements of knee torque and quadriceps and hamstrings EMG activity were measured during the movements. Findings indicated no significant difference in torque production between the injured and uninjured groups. In addition, it was also shown that no significant difference in torque existed between normal and injured legs within the injured group. There was a significant difference in the amount of EMG activity in the hamstrings between normal and ACL injured legs within the injured group. ACL deficient knees generated approximately half of the EMG activity of the uninjured knee, although no significant differences in torque were produced. EMG values during this extension were expressed as a percentage of mean EMG activity recorded during a maximal flexion. A second group of uninjured subjects with no history of knee injury produced no significant differences in EMG activity across legs.

A lack of weight bearing causes intra capsular pressures to decrease, changing the pressure and stimulation to the mechanoreceptors and proprioceptors within the joint capsule and therefore may have an undesirable effect for application to rehabilitation. Based on these findings, it is preferable to utilize walking or jogging as a practical test for knee EMG studies to provide realistic results.(Nordin & Frankel, 1989; McArdle et.al., 1991)

Wojtys (1994) tested 40 normal subjects and 100 ACL-deficient subjects and found a

significant difference between injured and uninjured leg muscle recruitment patterns. In addition, he found a significant correlation with subjective functional parameters obtained from patients.

A study of onset timing of vastus medialis and vastus lateralis muscles in patients with patellofemoral pain was conducted in 1995. The method to define muscular contraction onset was to utilize the first bipolar contraction that exceeded one Standard Deviation (S.D.) of the mean resting baseline amplitude during the first 15 milliseconds of the sampling window. This allowed artifact noise to be eliminated from consideration when recording onset times.(Karst & Willet, 1995)

The literature discussed demonstrates a question and need for more research on the issue of hamstrings contraction timing and tension to support alterations in ACL deficient individuals. A demonstrated pattern exists for the ACL deficient subject displaying several alterations, including decreased hamstrings strength (Osternig et al., 1995, McNair & Wood, 1993), and changes in the timing of hamstrings co-contraction prior to heelstrike. (Beard et al., 1993; Beard et al., 1994, Sinkjaer & Arendt-Neilsen, 1991; Kalund et al., 1990)

CHAPTER 3

METHODS AND PROCEDURES

The purpose of the study was to analyze electromyographical (EMG) timing patterns for hamstrings contraction in anterior cruciate ligament (ACL) deficient knees during walking gait patterns.

A secondary purpose is to relate functional measures on a Lysholm scoring scale to adaptations in the knee.

METHODS

Subjects included 10 male and female volunteers between the ages of 18 and 40 years, the range which includes the highest rate of ACL injury (Tibone & Antich, 1988; Seto et al., 1988; Moore & Wade, 1989).

Subject selection included ACL deficient subjects whose ACL was not surgically reconstructed. Subjects were a minimum of two years post injury due to the average length of most rehabilitation programs and recovery time.(Guyton, 1991; McArdle, Katch & Katch, 1991). By the end of a two year period post injury, most neurological changes have already occurred (Gauffin & Tropp, 1992). Subjects were able to walk without a visible limp, as judged by the examiner. Subjects were recruited from physiotherapy clinics in the Thunder Bay region based on consultation with the subjects, their doctors or their rehabilitation therapists.

All subjects signed a consent form prior to taking part in the study (See Appendix V). A detailed injury history was completed through an interview, including injury history, activity levels, age and maturation factors which may affect the internal validity of the results. Events such as additional joint trauma, the rehabilitation program and daily activity levels which

occurred between the time of injury and the time of testing were not controlled, but were described using an injury history (See Appendix IV). All subjects had been examined arthroscopically, however, no repair had been performed on either the ACL or meniscus.

The interview process included a history of previous injuries to the knee, hip, ankle or foot. Length of time since injury, treatment methods, progress of recovery, functional activity levels and a description of activity levels in the past 3 months were recorded. Activity levels include athletic endeavours, work ability, recreational activity and activities during daily living. Subjects completed a Lysholm Knee Scoring Scale (Tegner & Lysholm, 1985; Noyes et al., 1984; Noyes, 1977; See Appendix II) to determine knee functional levels. Subjects then outlined the most recent knee injury with regards to the mechanism of injury and treatment methods. (See Appendix IV)

Clinical testing began with a visual examination of the injured and uninjured legs. Straight leg raise testing was performed passively to determine hamstrings tightness as well as Ober's test for iliotibial band tightness. Valgus and varus pressures were applied to test knee ligament laxity. Finally, two standard tests of ACL laxity were performed. To gauge the degree of ACL deficiency, the anterior drawer test and the Lachman drawer test were performed. The purpose of clinical testing was to ensure that an ACL deficiency exists and that anterior tibial translation in the ACL deficient knee was greater than in the uninjured knee. Subjects who did not display positive signs for ACL deficiency were removed from the sample population. These ligament laxity tests were not used as data, but simply to ensure a homogeneous subject group. Subjects which tested positively on tests other than the anterior drawer and Lachman's tests of ACL instability were eliminated from the study and referred to a physician for further

investigation.

In order to assess the level of functional joint stability, subjects were assessed using the Lysholm scoring scale. The Lysholm scoring scale has been found to be a reliable method of determining self perceived daily function (Tegner and Lysholm, 1984; Noyes et al., 1984; Noyes, 1977; See Appendix II). The scoring scale provides information regarding the subject's knee function, during daily activities and work tasks. Questions were answered by the subject and tallied to form a percentile score. These scores were used to assess whether a relationship exists between injured and uninjured knees and functional stability using a Pearson statistical correlation. As all subjects had a unilateral ACL injury, a score of 100% was assumed for the uninjured leg.

Muscle action potentials (MAP) were recorded using Silver Chloride (Ag Cl) surface electrodes placed over the semimembranosus and semitendinosis muscles and a ground electrode over the tibial tuberosity. Active electrodes were placed over the muscle belly, 2 cm either side of the muscle action centre (Kalund et al., 1990). Hamstrings electrode placements were landmarked by measuring the distance between the centre of the knee at the joint line and the centre of the buttock at the level of the greater femoral trochanter. Both legs were prepared in an identical manner and monitored simultaneously. Foot switches to record heelstrike were taped directly to the heel. The foot sensors functioned as toggle switches to record a signal at heel contact. The location of the foot switches was at the most posterior and plantar aspect of the heel to record any contact with the ground and the size of the foot switches covered 75% of the heel, to ensure heelstrike capture.

A Bortec Electronics Inc. AMT-8 eight channel EMG system amplified the MAP (muscle

action potential) signal using a small pre-amp attached on the subject's waist. The signal was then transmitted along a 50 foot shielded cable to the main amplifier.

A shielded cable connected the pre-amplifier to both the electrodes and to the main amplifier. Signals were analyzed using the Global Labs signal processing package running on a 486 DX2/66 PC equipped with an analog to digital conversion board (Global Labs, 1993). Raw signals were analyzed without processing the signal further. Three trials of five seconds each were recorded, each containing several heelstrike cycles. Each trial was preceeded by collection of 15 msec of baseline EMG with no muscle activity in a standing position. Each heelstrike cycle was analyzed independently for hamstring timing. The time values were obtained using the first heelstrike marker as zero, the second heelstrike marker as 100 percent of the gait cycle. The onset of hamstring EMG occurred in between these two markers. This was expressed as a percentage of the total gait cycle, as explained below. Muscle contraction was defined as the first bipolar increase in amplitude more than one standard deviation above 15 msec of baseline EMG during a period of inactivity. This varied between subjects from 0.6 to 1.5 volts, depending on amplification and was calculated by SPSS by segregating the 15 msec of baseline EMG in standing. The first bipolar increase above this level for each subject was marked at the instant of occurance via a time marker within the dataset in SPSS. Muscle onset occurred at the end of the swing phase of gait, prior to second heelstrike. The timing data was normalized by representing hamstring contraction onsets as a percentage of the total gait cycle, so that data could be compared between subjects. The total gait cycle (100%) is the time from one heelstrike to the next heelstrike of the same leg. Normalized timing values were exported to Microsoft Excel to provide graphical analysis and to SPSS for Windows version 6.1 to provide

statistical analysis.

Reliability of the electromyography readings was tested using one test subject on a flat treadmill surface at 3 km/hr utilising the EMG methods outlined. The subject had no previous knee, hip or ankle injury and was prepared using the same methods and procedures as outlined for the sample population. The subject performed three trials and was retested on three separate occasions. On each occasion electrodes were placed according to the methods outlined. See Appendix VI for results of reliability statistics.

Subjects were asked to refrain from any strenuous exercise for 12 hours prior to the testing procedure to control for muscular fatigue. In addition, subjects were instructed to refrain from ingesting caffeine or any other stimulant for 2 hours prior to the test, as these have been found to increase neuronal excitability by lowering the action potential threshold and may affect EMG results (CSEP, 1986; Guyton, 1991).

Subjects were prepared for data collection in five consecutive steps:

1. An interview detailing injury history, activity level and injury mechanism;
2. A clinical examination using standardized objective testing;
3. A warmup (See Appendix III);
4. EMG preparation and electrode placement;

The interview and clinical examination were carried out as described in the Methods section above.

The next phase of subject preparation was a warm-up, familiarization procedure. The purpose of the warm-up was to control for pre-test activity and familiarize the subject with the treadmill. The warm-up/familiarization procedure consisted of a light stretching routine for both

the upper and lower body followed by treadmill walking for 2 minutes at 3 km/h and 1 minute at 5 km/h.(See Appendix III) The subject were then prepared for EMG analysis.

Electromyographical analysis preparation was completed as described in Electromyography in the Methods section.

Utilising the Bortec system and Global Lab signal analysis package, compression activated foot switches were inserted into the shoes of each subject and provided a precise measurement for heel strike timing. The switches provided a spike during heelstrike and a flat baseline readout when no weight was on the heel. Foot switches were tested to ensure that footwear and placement did not trigger false heel contact signals. Data was processed, collected and stored for analysis by the computer.

Before the main testing procedure was initiated, a pre-test, consisting of a series of knee extensions and flexions was completed to ensure a clear signal was recorded and that the heel strike sensor was functioning. A 15 msec baseline EMG measure was recorded to detect noise or artifact while the subject stood equally weighted on both legs and to establish a standard deviation to define muscular contraction.

The main testing protocol for this study involved walking on a treadmill at 3 and 5 km/h:

1) On a level surface and

2) On an incline (10°) simulating uphill walking;

Beginning with the treadmill speed at 3 km/h at no incline, subjects walked normally for a period of 2 minutes at flat and 10° incline, during which time, EMG was recorded for three randomly selected 5 second intervals. The three 5 second trials were recorded to allow up to 12 full heelstrike gait cycles to be sampled from each leg (Murray et al., 1984). The trial data was

saved for later analysis.

TABLE 1: Testing protocol at various speeds and inclines

Speeds	*Treadmill Grade*	
	Flat	10° Incline
3 Km/hr	(#1) 3x 5 sec. trials One minute rest	(#3) 3x 5 sec. trials One minute rest
5 Km/hr	(#2) 3x 5 sec. trials 3 minute rest	(#4) 3x 5 sec. trials

Subjects rested approximately one minute, while the incline was readjusted. The same procedure was repeated at 5 km/hr. A total of 6 five second readings were recorded for each of the 3 km/h and 5 km/hr speeds. Subjects were allowed three minutes of rest between incline settings. Each five second sample produced three heelstrike cycles, for a maximum of 9 cycles at each speed & incline variation.

The dependant variable being measured was hamstring timing (the time between hamstrings activation and heelstrike). Knee functional activity level, based on the Lysholm scoring scale, was a second dependent variable which was correlated to hamstring timing. The independent variables were the leg (injured versus non-injured), speed of the treadmill (3 km and 5 km) and the incline of the treadmill (0 and 10°).

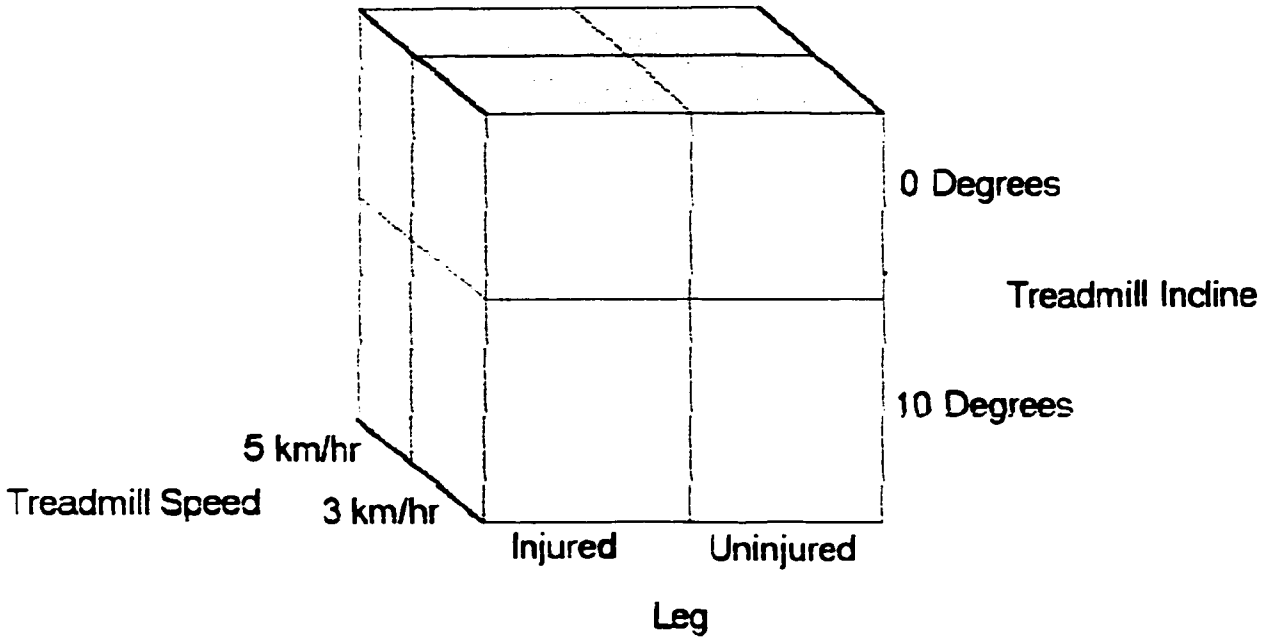

Figure 3: Factorial ANOVA Design

STUDY DESIGN

The research design consisted of a factorial repeated measures group design. An ANOVA determined if a significant difference existed for hamstring contraction times between legs. inclines and speeds while a correlation of the hamstring timing and functional scores on the Lysholm scoring scale determined if a relationship existed between knee function and hamstring timing. (See Figure 3)

Heelstrike timing was automatically recorded by the EMG system and incorporated into the data with a 0 indicating no heelstrike and a 1 indicating a heelstrike.

Hamstring onset was determined by visually examining data curves and selecting the first EMG increase above baseline prior to the second heelstrike in any given heelstrike cycle. The

hamstring EMG increase was required to be bi-polar and greater than one standard deviation above baseline EMG collected in a static standing posture with no muscle activation. It occurred following the stance and toe-off phases and was visually identifiable on EMG tracings (Tokuhiro et.al., 1985; Montgomery et.al., 1994).

Multiple heelstrike cycles were obtained for each subject at each testing condition. These were treated as a multiple samples and grouped by subject number. Analysis was performed by including each cycle as separate measurement. Each measurement was then included in the ANOVA.

STATISTICAL ANALYSIS

Two separate statistical analyses were carried out. The first was an ANOVA to determine if a significant difference existed between legs, treadmill incline level and treadmill speed. The second was a Pearson correlation between functional scores on the Lysholm knee scale and hamstring timing values.

A 2 (incline) x 2 (speed) x 2 (leg) ANOVA was performed to determine if a significant difference existed between injured and uninjured legs at 0° and 10° incline and speeds of 3 km/hr and 5 km/hr on a treadmill.

The three independent variables analysed in the ANOVA are as follows:

1) Injured Leg vs Uninjured Leg

2) 0° Incline vs 10° Incline

3) 3 km/hr Speed vs 5 km/hr Speed

The dependent variable, %ONSET, was calculated from raw hamstring timing data by the following formula:

$$\%ONSET = \frac{Heelstrike2 - HamstringOnset}{Heelstrike2 - Heelstrike1}$$

This provided a normalized %ONSET value representing the percentage of gait cycle that hamstring onset occurred prior to the final heelstrike in a particular gait cycle. The obtained %ONSET values was used as the dependent variable in a factorial ANOVA with Leg, Speed and Incline as the independent variables. (Kålund et.al., 1990)

Correlation coefficients (r. value) between Lysholm scale values and %ONSET values were calculated to indicate if a relationship existed between the functional score of Lysholm scale and the timing values for hamstrings. The correlation coefficient indicated if a relationship existed between hamstring timing values and functional activity levels. The purpose of this analysis was to determine if any demonstrated significant differences between legs were related to an increase or a decrease in self perceived function.

Reliability

Three trials of data from a normal, uninjured leg were collected using the test procedures described. An ANOVA procedure was used to test for differences between trials. The calculated F-ratio of 0.0130 fell inside the critical area of F, indicating that a significant difference between test trials does not exist, therefore the data is considered reliable.

CHAPTER 4

RESULTS

The purpose was to measure electromyographical (EMG) timing patterns for hamstrings contraction in anterior cruciate ligament (ACL) deficient knees during walking gait patterns. Electromyographic timing was correlated to knee functional scoring scales.

Timing data was collected and hamstrings contraction expressed as a percentage of each gait cycle measured, to allow for comparison between cycles. Normalization was performed by obtaining the absolute time (ms) of final heelstrike (AFH) of a given cycle and subtracting the absolute time of hamstring onset (AHO). The function of this equation was to locate hamstring onset relative to final heelstrike (RHO). This value was divided by the total time (CYCLE) from initial heelstrike to AFH, providing the percentage of the gait cycle prior to final heelstrike which hamstring onset occurred (%ONSET).

The following equation shows the procedure:

AFH - AHO=RHO (ms) *CYCLE=AFH - Initial Heelstrike Time (ms)*

RHO ÷ CYCLE =%ONSET

SPSS/PC 6.1 for Windows was used to statistically analyze data as previously described with an ANOVA procedure. Muscular contraction timing values provided represent the percentage of total gait cycle from the last heelstrike, before which the hamstring co-contraction occurs.

Analysis of variance showed two main effects, a Leg main effect ($F=893.492$, $p<0.000$) and a Speed main effect ($F=47.828$, $p<0.000$) at the 0.05 alpha level as shown in Figure 4 & 5. In addition, two interaction effects were found to be significant. Leg/Speed and Incline/Speed.

Refer to Figure 6 & 7 for group mean plots of interaction effects. A significant Leg/Speed interaction effect as shown in Figure 7, was observed with a calculated F-value of 18.05 ($p<0.000$) at a 0.05 alpha level. As demonstrated in Figure 7, hamstrings onset occurred earlier on injured legs at both speeds, however onset occurred even earlier at a higher speed. A significant Incline/Speed interaction effect as shown in Figure 6, was observed with a calculated F-value of 9.41 ($p<0.002$) at a 0.05 alpha level. Figure 6 shows hamstring onset occurring earlier as speed increases, but more so at a flat incline level.

No significance was shown in the Incline main effect ($F=1.157$, $p=>0.282$) at the 0.05 alpha level, the Incline/Leg interaction ($F=1.597$, $p=>0.207$) nor the Incline/Leg/Speed three way interaction ($F=3.053$, $p=>0.081$), each at the 0.05 alpha level.

The results of the Pearson correlation (see Table 4, Appendix VIII) indicate that a negative correlation exists between Hamstring Latency and Lysholm Functional Scales scores. A negative correlation indicates that as hamstring onset times occurred earlier, Lysholm scores decreased and vice versa. A Pearson correlation coefficient of -0.60 was calculated between Onset values and Lysholm scores.

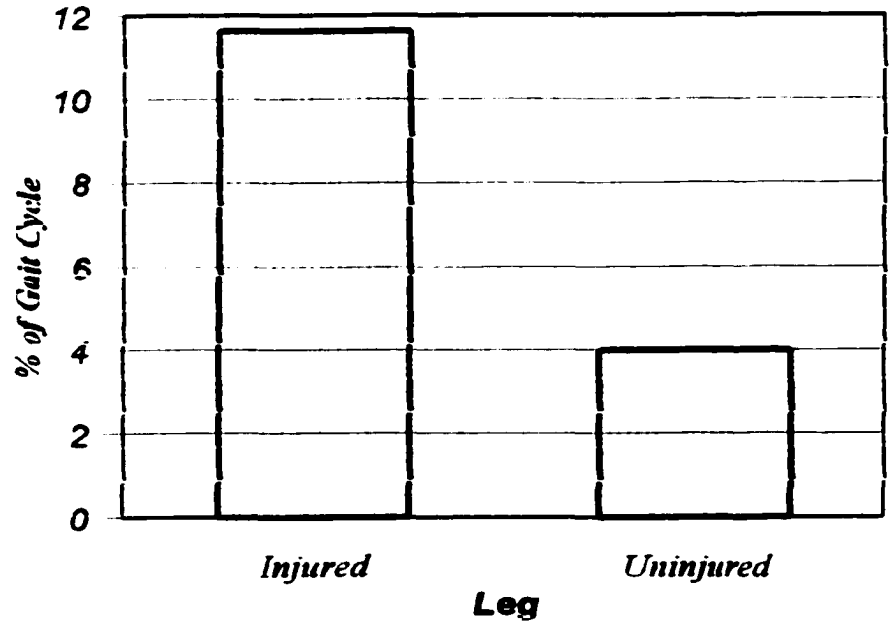

Figure 4:Main Effect of Injured vs Uninjured Leg on Hamstring Onset Timing

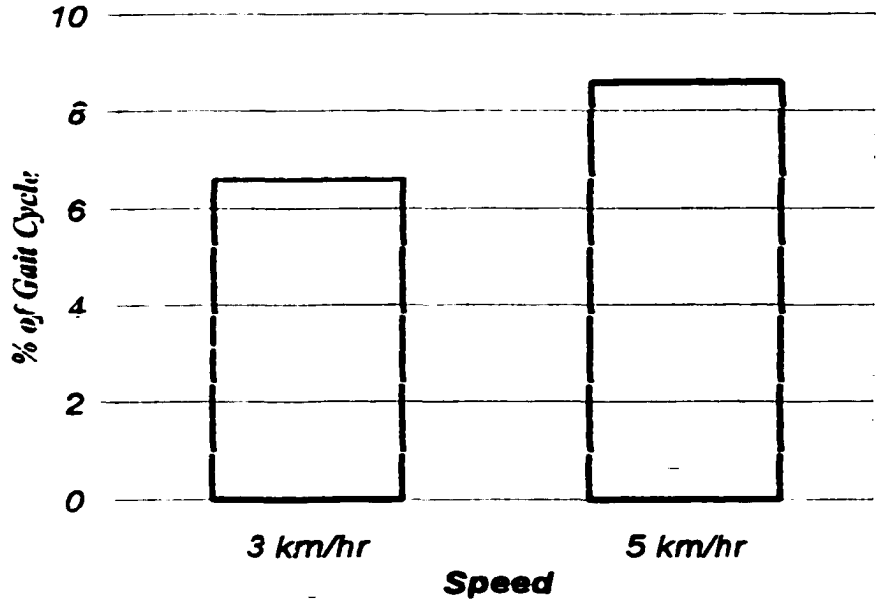

Figure 5:Main Effect of 3km/hr vs 5 km/hr Treadmill Speed on Hamstring Onset Timing

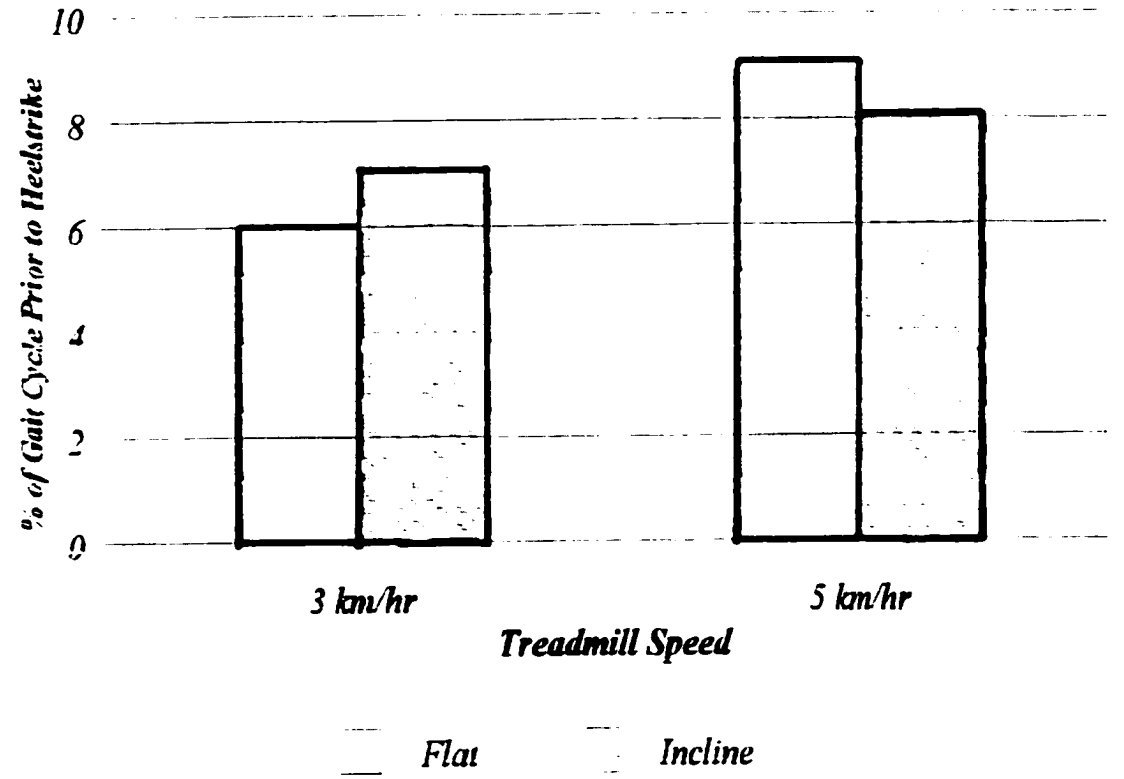

Figure 6:Interaction Effect of Speed & Incline Levels on Hamstring Onset Timing

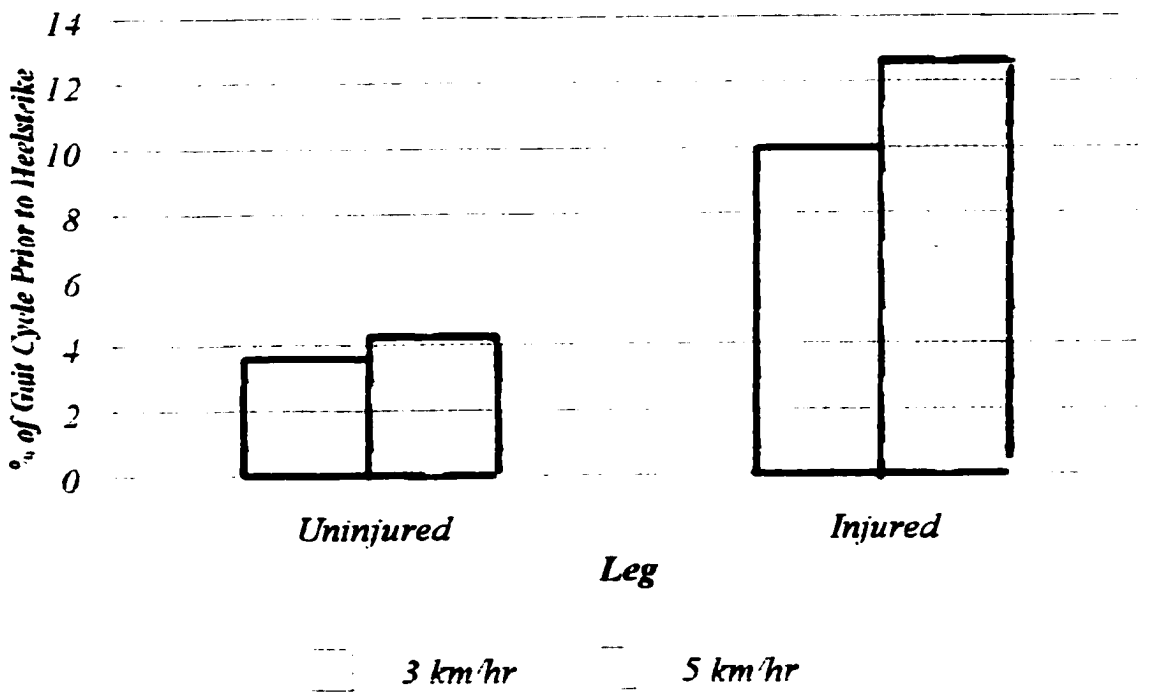

Figure 7:Interaction Effect of Leg and Speed Levels on Hamstring Onset Timing

Reliability Results

A test-retest reliability pretest for EMG raw recordings on an uninjured subject indicated an F ratio of 0.0130 within the critical area of F. There was no indication of significant differences between test trials and therefore the test reliability was verified (See Appendix VI).

CHAPTER 5

DISCUSSION

The purpose of the research was to measure electromyographical (EMG) timing patterns for hamstrings contraction in anterior cruciate deficient knees during walking gait patterns. A secondary purpose was to determine functional knee stability using a Lysholm scale correlated to hamstring onset timing.

Results indicate a significant difference in hamstring timing onset between injured and uninjured knees ($p<0.0001$)(See Table 3). The onset of hamstring contraction was found to be significantly earlier in the gait cycle in the ACL deficient knee compared to the uninjured leg.

A significant interaction effect ($p<0.002$) was found between incline and speed variations in both legs (See Figure 4). Gait at 3 km/hour on a 10° incline produced earlier contraction of the hamstrings when compared to level surface gait. Gait at 5 km/hour on a 10° incline produced later hamstring contractions relative to heelstrike when compared to level surface gait. A significant interaction effect ($p<0.0000$) was found between leg/speed variations on the injured leg (See Figure 5). Uninjured legs demonstrated non-significant differences in onset of hamstring contractions for each gait speed, however injured legs demonstrated significantly earlier contraction onsets at the 5 km/hr speed variation. Speed appears to have little effect on hamstring contraction timing in uninjured legs, but injured legs demonstrated significantly earlier contraction onsets. A negative correlation ($r= -0.60$)was found between scores on the Lysholm functional scoring scale and the onset of hamstrings contraction. Although this is a weak to moderate correlation, it suggests that an earlier contraction onset in the ACL deficient knee may be correlated to reduced functional knee stability.

The literature supports the theory that early hamstring contraction onset during the gait cycle may help protect the anterior cruciate ligament from anterior shear forces during heelstrike. The reflex has been demonstrated in static and dynamic movement during weight bearing and non-weight bearing.(Beard & Kyberd, 1993; Osternig et al., 1995; Kålund & Sinkjær, 1990) The literature also supports significant differences between ACL deficient and uninjured subjects for quadricep and hamstring strength, electromyographical patterns, and overall functional ability (Moore & Wade, 1989; Seto et.al, 1988; Kannus et.al., 1992; Hirokawa et.al., 1992). This study provides support to the theory that changes in muscular contraction patterns of the hamstrings occur in anterior cruciate deficient patients.

This study utilized a dynamic walking gait at different speeds and slopes as compared to static measures used by Jennings and Seedhom (1994) or Beard et al. (1993). Muscle spindle and golgi tendon organ (GTO) stretch reflex occurs in dynamic motion and is triggered by knee extension and greater hamstring tightness in the injured leg (Guyton, 1991). Hamstring tightness may induce earlier onset of hamstring contraction and a natural protective mechanism.

Explanations for changed timing patterns are varied, as the literature does not agree on the causal factor for hamstring contraction. Some studies attribute this reaction to receptors within the anterior cruciate ligament or joint capsule (Stener, 1959; Schultz et.al., 1984; Zimney et.al., 1986), while others suggest muscle tension information provided by golgi tendon organs and muscle spindles are responsible for initiating hamstring contraction (Schmidt, 1988; Solomonow et.al., 1987).

Another mechanism for early hamstring onset in the ACL deficient knee, is a motor schema, or motor program. Kålund et.al. (1990) suggested that a motor program, or schema,

may develop to provide an earlier contraction. The purpose for this earlier contraction may be to create a posterior shear force to counteract the anterior shear force of heelstrike. This counteraction may be required earlier in the injured knee due to a limp causing a significantly quicker heelstrike and a need for greater torque production from the hamstrings, due to increased knee flexion at heelstrike. Greater torque for increased tibial support and counteraction of forces during heelstrike may require increased flexion of the knee during heelstrike (Berchuk et.al., 1990). Adaptations to the injured leg may include, increased hamstring torque to provide more stabilization for the missing or damaged ACL or, decreased quadriceps force at heelstrike to reduce anterior shear force (Moore & Wade, 1989; Arms et.al., 1984; Seto et.al, 1988). As ACL proprioception decreases due to injury, a motor program may develop to provide this increased support (Magill, 1989; Schmidt, 1988). This is supported by the significant interaction effect of gait speed and injured leg. Speed increases were observed to affect the injured leg by initiating hamstring contraction earlier. No effect was apparent on the uninjured leg. This suggests that a possible motor program had developed to help protect the injured leg.

Early hamstring onset may also be affected by the incline of the contact surface during heelstrike. Greater incline would decrease the relative distance from the heel to the ground, shortening heelstrike time relative to hamstring onset. An earlier heelstrike may require compensation from the hamstrings. This is not supported in the literature in studies that factored hamstring onset relative to heelstrike. Kalund (1990) found no significant differences between heelstrike timing for injured and uninjured legs during walking. However, inclined walking was found to be significantly different for hamstring onset between incline levels (Kålund et.al.,

1990). Results from the current study do not support these findings for incline levels, except when speed and incline are examined together.

The interaction effect of incline and speed may be due to an increased level of muscle tension at increased speed levels. Montgomery (1994) found that higher paced activity required increased muscle tension in the hamstrings at knee extension prior to heelstrike. Due to the higher tension in the muscle tissue, threshold tension for hamstring contraction is reached earlier, initiating hamstring contraction.

The effect of incline at higher speeds is evident in the later hamstring contraction onsets observed with inclined gait. Heelstrike during inclined jogging, changes the angle of contact between the heel and the treadmill. As the treadmill is inclined 10°, the point of contact for the heel is closer, making the heelstrike earlier. Knee extension range during incline jogging may be reduced causing earlier heelstrike on an incline. Hamstring threshold tension may not be reached therefore limiting hamstring onset. Weresh et.al. (1994) concluded that the timing of muscle contraction was similar between injured and uninjured legs during ascending walking and ascending jogging. This is consistent with the findings of the current study.

Slower speed walking produced later hamstring onset in both flat and incline surfaces. This is demonstrated through the lack of significance in the Incline main effect (see Table 3, Appendix VII) which indicated no significant difference either between incline levels nor between incline levels factored with injured versus uninjured leg.. These results do not agree with Kålund et.al.(1990) who found a significant difference in hamstring onset timing between incline variations. The Kålund et.al. (1990) study, however, utilized a control group of uninjured subjects rather than comparing injured versus uninjured legs. Subjects may not have

been a homogeneous group as subjects may have simply had short hamstrings which could be tested by measuring timing bilaterally.

A negative correlation was found between scores on the Lysholm functional scoring scale and the onset of hamstrings contraction. The Pearson Correlation Coefficient (-0.6031) indicating a moderate correlation. The negative correlation indicates that, as hamstring onset occurred earlier, self perceived functional levels decreased and vice versa. The negative correlation is supported by Woijtys (1994) who found that a correlation did exist between muscular timing in the ACL deficient knee and subjective functional parameters. The study used a static position of 30° knee flexion and an applied anterior shear force, similar to Beard and Kyberd (1993) and Jennings & Seedhom (1994). The negative correlation demonstrates that earlier hamstring contraction was related to a lower functional score and suggests that the existence of an earlier contraction in the ACL deficient knee may not be a protective mechanism, as indicated. (Kålund et.al., 1990; Baratta et.al., 1988) An earlier hamstring contraction may be a deficiency due to the damage incurred in the injury. Alternatively, it may suggest that although earlier contraction of the hamstrings may be a protective mechanism, it may not be sufficient to change self perception of function as measured on the Lysholm scale. The scope of this study did not provide sufficient data to determine the cause of the relationship between hamstring timing and knee stability.

Conclusions

The results of the current study demonstrate differences in hamstring contraction timing between ACL deficient and uninjured knees. The observed difference between hamstring onset timing in ACL deficient knees and uninjured knees may be a protective mechanism or an unsuccessful attempt to compensate for anterior shear forces. The observed differences between injured and uninjured knees may, in fact, be an undesirable adaptation and this is supported by the relationship between hamstring contraction and lower functional scores. Another possibility to explain the observed changes may be that the effect is an unsuccessful attempt to compensate by the hamstrings. That is, the hamstrings contract earlier in compensation for the ACL laxity, however, the compensation may not be enough to prevent poor functional stability. Factors that may affect these differences in hamstring onset include decreased knee proprioception in determining joint position, tight connective tissue triggering a feedback loop to the motor neurons, initiating contraction early or altered nerve & proprioceptor conduction which may be responsible for an earlier firing pattern.

Recommendations for Future Studies

The following is suggested for further study:

As eight of ten subjects' knee functional scores were high, nearly all subjects tested in the current study were highly functional and had few problems. A repeated study with more acutely injured ACL patients and subjects with a wider range of function may provide a better understanding of the correlation between function and timing of hamstring onset. Including a 10° decline in the study design may factor out a question of whether heelstrike contact occurring earlier in inclined walking may have an effect on hamstring onset timing. The decline would

provide a later heelstrike and increased stretch as the knee extended further to reach the declining treadmill surface. It may also accentuate hamstring co-contraction. A larger subject sample would improve the subject range and variability.

The inclusion of synchronized video analysis would provide additional information to more accurately correlate EMG onset for eccentric contraction of the hamstrings. Video analysis could provide leg angular acceleration and deceleration and identify changes in velocity related to EMG onset. A training procedure, utilizing a gait retraining protocol including electrical stimulus, biofeedback or proprioception drills may provide hamstring timing stimulation to determine hamstring trainability.

APPENDIX I

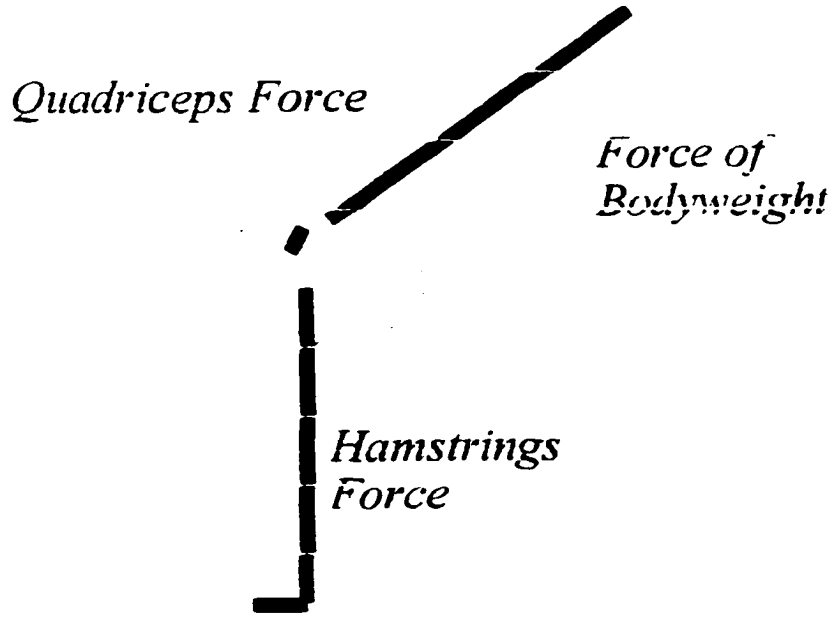

Figure 8:Knee Forces During Heelstrike (Flexion)

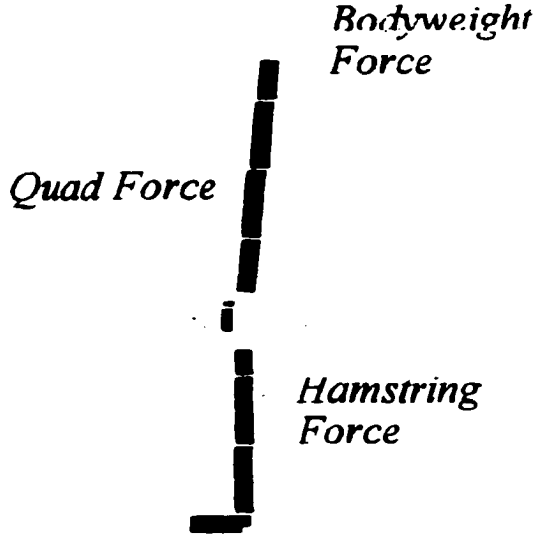

Figure 9: Knee Forces During Midstance (Extension)

APPENDIX II

TABLE 2: Lysholm Knee Scoring Scale	(Tegner & Lysholm, 1985)
	# of Points
Limp (5 Points)	
None	5
Slight or periodical	3
Severe and constant	0
Support (5 Points)	
None	5
Stick or crutch	2
Weight-bearing impossible	0
Locking (15 Points)	
No locking and no catching sensations	15
Catching sensation but no locking	10
Locking occasionally	6
Locking frequently	2
Locked joint on examination	0
Instability (25 Points)	
Never giving way	25
Rarely during athletics or other severe exertion	20
Frequently during athletics or other severe exertion (Or incapable of participation)	15
Occasionally in daily activities	10
Often in daily activities	5
Every step	0
Pain (25 Points)	
None	25
Inconstant and slight during severe exertion	20
Marked during severe exertion	15
Marked on or after walking more than 2 km	10
Marked on or after walking less than 2 km	5
Constant	0
Swelling (10 Points)	
None	10
On severe exertion	6
On ordinary exertion	2
Constant	0

Stair-climbing (10 Points)	
No problems	10
Slightly impaired	6
One step at a time	2
Impossible	0
Squatting (5 Points)	
No problems	5
Slightly impaired	4
Not beyond 90 degrees	2
Impossible	0

APPENDIX III
WARM UP PROCEDURES

1. Light Stretch Routine (each x 10)
   - -neck rotations
   - -shoulder shrugs
   - -arm circles
   - -trunk rotations
   - -seated hamstrings stretch -single leg at a time
   - -standing quadriceps stretch
   - -ankle rotations

2. Two Minute Treadmill Walking
   - -3 km/hr

3. 1 Minute Treadmill Walking
   - -5 km/hr

APPENDIX IV
INTERVIEW PROCESS

1. Interviewer: Have you had any previous injuries to your hips, knees, ankles or feet ? If so, please describe them.

Subject Response: _____

_____

_____

2. Interviewer: How were these injuries treated ? (If #1=no, skip)

Subject Response: _____

_____

_____

3. Interviewer: Do these previous injuries still affect your activity levels ? (If #1=no, skip)

Subject Response: _____

4. Interviewer: Do you participate in any sports ? If so, at what level do you compete ? (ie. Elite, Professional, Recreational, Occasional, etc.)

Subject Response: _____

_____

_____

_____

5. Interviewer: What type of work do you do ? How often do you work ?

Subject Response: _____

_____

6. Interviewer: Does your knee injury affect work, recreational activity, daily chores or sport activity ? If so, what is affected and to what degree ?

Subject Response: _____

_____

_____

_____

7. Interviewer: What were you doing when you injured you knee ?

Subject Response: _____

_____

_____

8. Interviewer: Did contact occur during your knee injury ? Ie. Did anyone "hit" you ?

Subject Response: _____

9. Interviewer: Was a twisting motion involved during your injury ?

Subject Response: _____

10. Interviewer: What methods were used in treating your current knee injury ?

Subject Response: _____

_____

_____

Appendix V
SIGNED CONSENT FORM

I _____ am a person with a damaged anterior cruciate ligament, and consent to take part in a study to help determine what relationship hamstring timing has to functional activity levels and if there is a difference between my injured and uninjured leg muscular timing. I have had the study explained to me and have read a brief synopsis of the study and thoroughly understand what my role in this study is. I hereby agree to participate. I have been given the opportunity to ask questions about the study.

I am aware that I will be asked to come to the School of Kinesiology at Lakehead University for a recording of the muscle activity of my thigh muscles. I understand that I will be asked to walk on a treadmill at a pace of 3 km/hr and 5 km/hr on both a flat surface and an incline. I understand that I have been asked to refrain from exercising 12 hours prior to the test and avoid drinking caffeine for 2 hours prior to the test to avoid affecting test results. I realise that I will be asked to complete a questionnaire about my knee injury and my daily functional activities. I understand that I must be as accurate and specific as possible on this questionnaire.
I understand that testing involves placing adhesive electrodes on the back thigh muscles to measure muscle activity during treadmill walking.
I realise that I may withdraw from this study at any time, and that I may contact one of the researchers for information about the study results after the study has been completed.

SIGNATURE: _____

DATE: _____

APPENDIX VI

Results of SPSS ANOVA for Reliability

TABLE 3: Analysis of Variance of Reliability
Variable ONSET (Hamstring Onset in Percentage of Total Gait)
By Variable TESTNUMB (Test Trial Number)

Source	D.F.	Sum of Squares	Mean Squares	F Ratio	F Prob.
Between Groups	2	.0000	.0000	.0130	.9871
Within Groups	27	.0074	.0003		
Total	29	.0074			

An ANOVA procedure was performed on three separate data collection trials using the procedures outlined in the Methods section. An ANOVA was used due to the fact that each trial produced several heelstrike cycles and the means of these cycles in each collection trial should not be significantly different. An ANOVA procedure was run on the data (ONSET) using the test trial number (TESTNUM) as a grouping variable. An F-ratio was calculated. The F-ratio of 0.0130 fell inside the critical area of F, indicating that a significant difference between test trials does not exist, therefore the data is considered reliable.

APPENDIX VII

Results of SPSS ANOVA Procedure for Data Set

TABLE 4: SPSS ANOVA on ACL deficient subjects

Source of Variation	Sum of Squares	DF	Mean Square	F Value	Sig of F
Main Effects	1.169	3	0.390	323.552	0.000 **
INCLINE	0.001	1	0.001	1.157	0.282
LEG	1.076	1	1.076	893.492	0.000 **
SPEED	0.058	1	0.058	47.828	0.000 **
2-Way Interactions	0.035	3	0.012	9.618	0.000 **
INCLINE LEG	0.002	1	0.002	1.597	0.207
INCLINE SPEED	0.011	1	0.011	9.414	0.002 **
LEG SPEED	0.022	1	0.022	18.050	0.000 **
3-Way Interactions	0.004	1	0.004	3.053	0.081
INCLINE LEG SPEED	0.004	1	0.004	3.053	0.081

** Significant difference at the 0.05 level of significance

APPENDIX VIII
Correlation Results

TABLE 5: Correlation Coefficients

	ONSET	LYSHOLM
ONSET	1.0000	-0.6031
	(835)	(835)
	P= .	P= .000
LYSHOLM	-0.6031	1.0000
	(835)	(835)
	P= .000	P= .

(Coefficient / (Cases) / 2-tailed Significance)

" . " is printed if a coefficient cannot be computed

APPENDIX IX
Descriptive Statistics

TABLE 6: Main Effect Descriptive Statistics

Group	Mean	Std. Dev.	Std. Error
Leg			
Injured	11.61	4.84	0.24
Uninjured	3.97	1.71	0.08
Speed			
3 km/hr	6.59	4.48	0.24
5 km/hr	8.58	5.6	0.25
Incline			
Flat	7.9	5.52	0.28
10°	7.65	5.02	0.24

TABLE 7: **Interaction Group Descriptive Statistics**

Interaction Groups	Mean	Std. Dev.	Std. Error
Incline/Speed			
Flat/3 km/hr	6.00	4.387	0.354
Flat/5 km/hr	9.09	5.82	0.373
10°/3 km/hr	7.06	4.51	0.329
10°/5 km/hr	8.08	5.34	0.337
Incline/Leg			
Flat/Injured	12.21	4.97	0.362
Flat/Uninjured	3.969	1.829	0.127
10°/Injured	11.113	4.681	0.311
10°/Uninjured	3.9691	1.581	0.1083
Leg/Speed			
Injured/3 km/hr	9.970	4.230	0.3344
Injured/5 km/hr	12.641	4.924	0.3083
Uninjured/3 km/hr	3.598	1.724	0.1281
Uninjured/5 km/hr	4.250	1.641	0.1062

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