Neuromuscular Control after Injury to the Knee:

An Anterior Cruciate Ligament Injury and Reconstruction Model

A Thesis

by

Laurie Anne Hiemstra

Submitted to the Faculty of Graduate Studies

In Partial Fulfillment of the Requirements

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List of Abbreviations

AANA – Arthroscopy Association of North America
ACL-QOL – Anterior Cruciate Ligament Quality of life
ANOVA – Analysis of variance
AOSSM – American Orthopedic Society of Sport Medicine
ACL – Anterior cruciate ligament
AAOS – American Academy of Orthopedic Surgeons
BPB – Bone-patellar tendon-bone
CASM – Canadian Academy of Sport Medicine
COA – Canadian Orthopedic Association
CORA – Canadian Orthopaedic Residents Association
DCR – Dynamic control ratio
EMG – Electromyography
FNE – Fine nerve endings
G - Gracilis
GPS – Global positioning satellite
H - Hamstring
H/Q – Hamstring/Quadriceps
HRP-WGA – Horse radish peroxidase – wheat germ agglutinin
ICC – Intraclass correlation coefficient
IKDC – International Knee Documentation Committee
ISAKOS – International Society of Arthroscopy Knee and Orthopaedic Sport Medicine
J - Joule
KE – Knee extensors
KF – Knee flexors
kg - kilogram
LAD – Ligament Augmentation Device
LCL – Lateral Collateral Ligament
m – Moment arm
ms – Millisecond
MK - Moment of the knee
MRI – Magnetic resonance imaging
N - Newton
Nm – Newton metre
PAN – Posterior articular nerve
RJM – Resultant joint moment
RJMK – Resultant joint moment of the knee
ROM – Range of motion
SD – Standard Deviation
SE – Standard error
SEP – Somatosensory evoked potentials
ST - Semitendinosus
VAS – Visual analogue scale
WB - Weightbearing
° - degrees
°/s – degrees per second

Abstract

The goal of this series of investigations was to elucidate and characterize the changes in neuromuscular control that occur in response to ligament injury, surgical reconstruction, and subsequent rehabilitation. Comprehensive strength analysis of the ipsilateral and contralateral knee after ACL reconstruction identified regional changes in strength limited to joint angle, speed and contraction type that were dependent on graft selection (Study 1). The concept of neuromuscular balance about the knee is further developed using a comprehensive depiction of agonist and antagonist strength in both active healthy and surgical groups (Study 2). Strength deficits were identified in the contralateral uninjured limb after ACL reconstruction when compared to controls (Study 3). Neuromuscular changes in the contralateral limb are particularly important because of the widespread use of the contralateral limb as a control limb in both the clinical and research settings. Strength deficits in the contralateral limb were demonstrated immediately after ACL injury consistent with a neural nociceptive crossover etiology (Study 5). The lack of strength deficits of the hip extensors in the presence of large knee flexor strength deficits after hamstring ACL reconstruction suggest differential alteration of bi-articular muscles (Study 6). Finally, the role of proprioception, muscle fatigue and the implications for dynamic joint stabilization is presented (Study 4).

The possible underlying physiological mechanisms responsible for regional and global strength deficits could be muscular, mechanical or neural in origin. Changes in motor unit recruitment and/or activation patterns of the agonist and antagonist muscles that control motion about the knee and hip joints appear to be the most likely candidates to account for regional variations in resultant joint moment. Afferent feedback from the

knee joint and peri-articular structures, spinal reflex changes and central and volitional mechanisms could mediate changes in neuromuscular activation.

Introduction

The understanding of the neural control of muscle activation in humans during functional tasks is slowly evolving and has implications in the fields of physiology, medicine and rehabilitation. A perturbation in the system, such as an injury or surgical intervention, will induce alterations in neuromuscular activation strategies. The underlying mechanisms of alteration of neuromuscular control strategies after injury or surgery are as of yet undetermined. We have chosen anterior cruciate ligament (ACL) injury and reconstruction as a model to help elucidate and characterize changes in neuromuscular control that occur in both the injured and contralateral limbs. This thesis reports a series of investigations designed to systematically evaluate the muscular and neural contributions to strength changes that occur after ACL injury and reconstruction in both the injured limb and the contralateral uninjured limb. Possible mechanisms include altered afferent feedback from the knee joint or peri-articular muscles, changes in spinal cord reflex pathways, neural crossover and modification by central and volitional control.

Strength assessment in humans involves eliciting maximal effort contractions of the prime movers, which in this model, primarily involves the knee extensors (KE) and knee flexors (KF). The forces produced by the muscles are dependent on both muscular properties and the neural activation strategies of the subject. Muscular properties include muscle architecture, cross-sectional area, muscle fibre pennation and mechanical properties such as moment arm and muscle length [98]. Neural activation strategies of a muscle include number of motors units activated, rate of motor unit activation, synchrony of activation, and selection of motor units [191, 242]. In addition, there are neural activation strategies involved in activation of other prime movers, motor pools that control the

distributions of force between synergistic muscles and antagonistic muscles. Thus, in vivo, muscle force is primarily dependent upon activation through the nervous system and the upper bounds of force are limited by the mechanical output characteristics of the muscle (force/velocity and force/length relationships). The resultant joint moment (RJM) represents the net rotary tendency arising from forces generated by all tissues spanning a joint. Each muscle generates a force that acts via its moment arm to contribute to the overall resultant joint moment. Changes in RJM that are specific to range of motion, speed, or contraction type (concentric, eccentric, or isometric) are most readily explained by modification of the neural activation strategies of homonymous, synergist, or heteronymous, including antagonist, motor unit pools because these regional strength deficits cannot be explained by alterations in muscular or mechanical properties.

Changes in strength of the knee flexors and extensors have been documented after ACL injury and ACL reconstruction [164, 234, 295, 390]. Synthesis of findings derived from these studies results in a fragmented compilation of information due in large part to the disparate data collection and analysis techniques [188]. Studies have principally used the peak moment generated by a muscle group for data analysis, regardless of the knee joint angle at which the moment was generated. Since moment generation is subject to the moment-angle and moment-angular velocity characteristics of the neuromuscular system, peak moments may occur at varying knee joint angles and angular velocities. In addition, there is no consistency among studies with the use of body mass normalization of moments, correction for the moment of the weight of the limb, and the number of angular velocities tested. Although the number of studies reporting concentric peak moments after ACL injury and surgery is substantial, there is a conspicuous paucity of literature

documenting changes in eccentric knee joint moments. These deficiencies and inconsistencies in data collection and analysis highlight the fact that current literature provides an incomplete portrayal of the neuromuscular performance of the muscles that control the knee in both normal and ACL injured subjects. During functional activities (daily living or sport) muscle must be activated in a variety of manners (different speeds, contraction types and ranges of motion). As such, the use of a single strength measurement limited to concentric contractions and a single speed has limited face and construct validity as a measure of functional outcome.

Using modern dynamometry techniques, we have identified regional strength deficits localized to specific joint angles, angular velocities, and contraction type (concentric and eccentric) after ACL reconstruction that have been previously unidentified (Study 1). The regional nature of these strength deficits is consistent with them arising from an alteration of neuromuscular activation strategies of the agonist and antagonist muscles that control the knee. The identification of these regional strength changes through Study 1 has immediate implications for our understanding of what is commonly referred to as 'muscle balance'. In this thesis, we use the term "neuromuscular balance" to more accurately reflect the possibility that the optimal balance (or imbalance) could arise from either neural or muscular properties.

Study 2 investigates traditional agonist-antagonist strength balance in both normal and ACL subjects. The alteration of agonist-antagonist balance would have implications for both prevention of injury [268] and for the functional outcome of patients after injury or surgery. Current estimators of agonist-antagonist balance such as the Hamstring/Quadriceps (H/Q) ratio do not take into consideration the moment-angle or

moment-angular velocity characteristics of dynamic contractions, nor do they consider the co-activation of muscles about a joint. Motor control strategies must include the dual requirements of the production of segmental motion about a joint while concomitantly maintaining joint stability. In this thesis, the concept of neuromuscular balance is further developed using a comprehensive depiction of agonist and antagonist strength in both active healthy and surgical groups (Study 2).

Study 3 examines side-to-side neuromuscular balance of the lower extremity. Altered neural activation strategies may not be confined to the ACL injured or ACL reconstructed limb but likely have cross-over effects on the contralateral limb. This is the first study to identify that strength deficits after ACL reconstruction were larger than the contralateral limb when compared to uninjured controls. Neuromuscular changes in the contralateral limb are particularly important because of the widespread use of the contralateral limb as a control limb in both clinical and research settings. Possible etiologies of the contralateral limb strength deficit include neural crossover inhibition and/or de-conditioning. Information as to the time course of the development of these contralateral limb strength deficits will help ascertain the contributions of alterations in neural and muscular properties to contralateral limb strength deficits (Study 5).

Finally, harvest of the semitendinosus (ST) and gracilis (G) tendons for hamstring ACL reconstruction may have muscular, mechanical and neural effects on the moment generating capacity about both the hip and the knee, since these are bi-articular muscles. The effect of ST and G tendon harvest on hip strength has not been previously examined (Study 6) and will provide insights into the alteration of neuromuscular control mechanisms after injury, surgery and subsequent rehabilitation.

Précis

This thesis is composed around six manuscripts each with a specific objective and hypothesis outlined below. An introduction of the model of ACL injury and reconstruction and a general review of the literature is provided as an introduction to the manuscripts. Following the general review of literature is a methodology section that outlines the key instrumentation and methodology common to the majority of manuscripts. An overview provided prior to each manuscript along with a supplemental literature review specific to the study. Following the manuscripts, a point form, concise summary of the key results of each study is provided. The thesis is completed with a synthesis discussion compiling the findings from each of the manuscripts.

For each study (1-3,5,6), the study design, methodology, and data interpretation were performed by L. Hiemstra and D. Kriellaars. L. Hiemstra was the principle person in charge of data collection and data analysis. L. Hiemstra prepared the manuscripts and figures for publication, as well as posters and presentations for conferences. L. Hiemstra was the principle author for the review manuscript (Study 4). These studies (1-3,5,6) have been presented at the American Academy of Orthopaedic Surgeons (AAOS), International Society of Arthroscopy, Knee Surgery and Orthopaedic Sport Medicine (ISAKOS), Canadian Orthopaedic Association (COA), Canadian Academy of Sport Medicine (CASM), Arthroscopy Association of North America (AANA), Resident and Fellows Arthroscopy Conference, Canadian Orthopaedic Residents Association (CORA), and the American College of Surgeons.

Thesis Objectives

The underlying physiological mechanisms mediating the changes in the neuromuscular system after ACL injury, reconstruction, and subsequent rehabilitation have yet to be determined. After a perturbation about the knee, such as ACL injury or ACL reconstruction, changes in motor control strategies that govern voluntary muscle activation have been documented [164]. These strength changes could arise from changes in muscular, mechanical or neural properties that control maximum voluntary muscle contraction. Changes in the muscular and mechanical properties of the knee flexors and extensors do occur after ACL reconstruction surgery, however the regional nature (specific to knee joint angle, speed and contraction type) of these strength changes suggest that there is an alteration in neural drive leading to a differential activation of motoneurones. Thus, injury and surgery have a non-uniform effect on neural activation strategies.

We have undertaken the task of performing a series of progressive investigations to further our understanding of alterations in the neuromuscular system associated with knee injury. These investigations will also help to identify the muscular and neural contributions to strength changes that occur after ACL injury and reconstruction in both the injured limb and the contralateral uninjured limb. The clinical nature of this ACL injury and reconstruction model allows us to directly relate physiological findings to the clinical situation (bench to bedside). The following objectives form the basis for the manuscripts presented in this thesis.

The first objective (Study 1: Graft Site Dependent Knee Strength Deficits after Patellar Tendon and Hamstring Tendon ACL Reconstruction) was to use comprehensive strength analysis to determine whether or not graft site selection (hamstring or patellar

tendon) for ACL reconstruction contributes to regional variations in strength dependent upon joint angle, angular velocity, or contraction type. This allowed us to assess the combined effect of the alteration of sensory and motor afferent information (knee joint, ACL, flexor mechanism, extensor mechanism) and rehabilitation on neuromuscular activation patterns after surgery as manifested by changes in maximum voluntary contraction strength. We hypothesized that global knee extensor strength deficits compared to control would exist in the knee extensors that are ACL injury dependent. We also hypothesized that regional knee flexor and extensor strength deficits will exist that are dependent on autograft selection (hamstring or patellar tendon).

The second objective (Study 2: Hamstring and Quadriceps Strength Balance in Normal and Hamstring ACL Reconstructed Subjects) was to evaluate and characterize the agonist-antagonist strength balance (H/Q Ratio and Dynamic Control Ratio) specific to speed, range of motion and contraction type. This study was designed to provide an overall portrayal of the neuromuscular balance about the knee to permit further development of our conceptual understanding of 'muscle balance'. Further, this agonist-antagonist balance was used to depict the impact on joint stability. In order to control segmental movement and maintain joint stability in the presence of that movement, an appropriate level of activation of the agonist and antagonist muscle groups must exist. Strength, under maximal effort conditions, sets the upper boundary for moment generation by the individual. Alterations in the upper boundary of strength may reflect limitations in setting the appropriate muscle activity levels for achieving the balance of joint stabilization in the presence of dynamic joint motion. We hypothesized that there will be systematic variation in the H/Q ratio and dynamic control ratio (DCR) based on

knee joint angle, angular velocity and contraction type. We also hypothesized that these ratios would be altered in the ACL reconstructed group in favour of protecting the ACL graft (relative knee flexor strength when strain on the ACL is the greatest).

The third objective (Study 3: Contralateral Leg Strength Deficits after Hamstring Tendon ACL Reconstruction) was to determine whether current post-operative ACL rehabilitation protocols return the strength of the contralateral limb knee flexors and extensors to that of an uninjured control group. These findings have important implications for the use of the contralateral limb as an appropriate control. This will demonstrate whether the effect of a unilateral insult or intervention is confined or limited to the ipsilateral limb, or whether it has effects on the contralateral uninjured limb. We hypothesized that after hamstring tendon ACL reconstruction and subsequent rehabilitation, bilateral strength normalization (defined as strength within 10% of the contralateral limb) would be achieved for the knee extensors but not for the knee flexors, and secondly, that both the knee flexor and knee extensor strength would be reduced when compared to an age and activity matched control group.

The fourth objective (Study 4: Effect of Fatigue on Knee Proprioception: Implications For Dynamic Stabilization) was to provide a review of the current knowledge regarding fatigue, knee joint proprioception and the implications for dynamic knee stabilization.

The fifth objective (Study 5: The Development of Contralateral Leg Strength Deficits after Acute Anterior Cruciate Ligament Injury) was to determine the effect of an acute ACL injury on the contralateral leg by documenting the time course of contralateral strength changes. We hoped to determine whether there are immediate neural

consequences with regards to motor output on the contralateral side to an injured limb as assessed by immediate changes in strength. We hypothesized that there would be substantive strength deficits of the contralateral limb and that these would necessarily arise from a neural mechanism (likely potent cross-over nociceptive inhibition). We hypothesized that the initial strength loss can likely be attributed to a neural mechanism and that this strength deficit resolves as the neural inhibitory mechanism reduces in the presence of the onset of de-conditioning atrophy.

The sixth objective (Study 6: Hip Extensor and Adductor Strength Following Hamstring Tendon Anterior Cruciate Ligament Reconstruction) was to determine whether the harvest of the semitendinosus and gracilis tendons for ACL reconstruction has effects on the resultant joint moment of the hip. This study will assess the impact of surgical intervention to bi-articular muscles and whether it has direct (mechanical or muscular) or indirect (altered movement strategy) effects at the hip joint. We hope to determine whether the effect of tendon harvest is different at the knee and at the hip in terms of the effects on neural activation of motoneurones during knee flexion and hip extension. We hypothesized that harvest of the semitendinosus and gracilis tendons for ACL reconstruction would lead to strength deficits of the hip extensors and the hip adductors.

The Model

Investigation into the changes in neural activation strategies and neuromuscular control that occur after injury in humans is logistically very difficult to perform. For this series of investigations, injury to the ACL and its subsequent surgical reconstruction was used as a model. ACL injuries are common in the active population and ACL reconstruction is a commonly performed procedure with approximately 500 per year performed in Winnipeg, Manitoba and London, Ontario. The rehabilitation program guidelines after ACL reconstruction have resulted in a level of standardization that emphasizes early range and motion exercises and early weight bearing of the operative limb.

This ACL injury and reconstruction model allows the investigation of neuromuscular changes on several fronts. First, rupture of the ligament may result in a loss of afferent information from the receptors signaling tension within the ACL. Second, knee joint instability secondary to ACL deficiency may cause altered afferent information from the capsule and ligament receptors about the knee joint. Third, the injury itself may introduce nociceptive signals from within the knee joint. Fourth, surgical reconstruction is in itself an insult with subsequent nociceptive effects and has a direct mechanical impact as it uses autograft material which can come from one of two major sources, the middle third of the patellar tendon or the pes anserine hamstring tendons (semitendinosus and gracilis). This allows access to two groups for comparison that have iatrogenically introduced alteration in the motor and sensory information from antagonist mechanisms (flexor and extensor).

The following section will briefly review the anatomy, biomechanics and techniques of surgical reconstruction of the ACL. In order to speculate as to the

mechanisms behind changes in neuromuscular control, detailed description of the afferent connections of the knee and the accompanying ligament muscle reflexes as well as a review of the current knowledge of ACL reconstruction neuromuscular outcomes is necessary.

Anterior Cruciate Ligament Anatomy and Biomechanics

The ACL is an intra-articular knee ligament that extends from the femur to the tibia. The femoral origin is an approximately 23 mm by 4 mm area on the posterior aspect of the medial surface of the lateral femoral condyle [16]. The tibial attachment is attached anterior and lateral to the tibial spine next to the anterior horn of the lateral meniscus [16]. The ACL averages 31-38 mm in length with an average width of 11 mm [133]. The ACL is composed of antero-medial and postero-lateral bands. The antero-medial band is tight in flexion and the postero-lateral band is tight in extension.

The primary biomechanical function of the ACL is to resist anterior tibial translation on the femur. Secondary functions are to resist internal rotation and varus/valgus forces [236]. The ultimate failure strength of the human ACL is 1725-2160 N/mm [384, 385] which may vary dependent on load as well as limb position during the time of injury [386]. The ACL bears load throughout the flexion-extension arc during normal activities [167]. Measurement of the in vivo strain of the ACL demonstrates that the greatest strain is during full knee extension with increasing forces from 0-30° of flexion [33]. ACL strain is decreased by co-contraction of the hamstrings and quadriceps [33].

Afferent Connections in the Knee

Neural Anatomy

Table 1 and 2 provide a tabular summary of the research in this field. Branches of the sciatic, femoral, and obturator nerves innervate the human knee joint [127]. The most consistent branch is the posterior articular nerve (PAN), a branch of the posterior tibial nerve that supplies the capsule, menisci, and the cruciate ligaments. These sensory afferent nerves carry signals from receptors within the peri-articular tissues about the knee. These receptors were originally classified based on their morphology and function by Freeman and Wyke [112] and later modified by Hogervorst and Brand [166]. Type I receptors, or Ruffini endings, are low threshold, slowly adapting receptors that respond to mechanical stress. They are believed to signal static joint position, joint movement and changes in intra-articular pressure [394]. Type II receptors, or Pacinian corpuscles, are dynamic, rapidly adapting, with a low threshold. Type II receptors are only active when the joint is moving and are thought to signal joint acceleration and deceleration [394]. Type III receptors or Golgi endings, are high threshold and slowly adapting and are active only in the extreme ranges of motion and at high stresses [394]. Type IV endings, or free nerve endings are high threshold, non-adapting pain receptors [394].

Using light microscopy techniques with gold or silver staining, afferent receptors have been identified in the ACL of animals [81, 205, 324, 366, 387] and humans [12, 81, 149, 157, 202, 309, 311]. Receptors in the ACL are located in the sub-synovial space and have been identified along the entire length of the ligament. These afferent receptors are less plentiful in the ACL when compared to other peri-articular structures such as the patellar tendon and the joint capsule. In 1989, Halata and Haus [149], first identified

mechanoreceptors in the human ACL using electron microscopy. Their findings correlated with previously published light microscopy findings.

Studies of the human ACL remnant found during reconstruction of the ligament have also been performed [5, 130]. Georgoulis et al [130] studied the ACL remnant and found that in remnants which had scarred down to the PCL, there were type I and II mechanoreceptors. In ACL remnants that had mushroom like ending with no proximal attachment, no mechanoreceptors were demonstrated.

Afferent receptors have also been identified in the reconstructed ACL [81, 366]. The development of immunohistochemical techniques allowed the identification of functional receptors suggesting that the reconstructed ACL becomes re-innervated [18, 117].

Table 1 - Identification of neural structures within the anterior cruciate ligament in animals. HRP-WGA – Horse Radish Peroxidase-Wheat Germ Agglutinin; FNE – Fine Nerve Endings; BPB – Patellar Tendon; ACL – Anterior Cruciate Ligament.

Paper	Technique	Subject	Receptor Type	Location
Wada 1997 [366]	Gold chloride	Rabbit	Pacinian Ruffini	Reconstructed BPB ACL
Gomez-Barrena 1996 [134]	HRP-WGA	Cats	Retrograde transport to spinal ganglia	ACL
Koch 1995 [205]	Gold chloride	Cat	Golgi	Entire length
Madey 1997 [229]	WGA-HRP	Cat	Ruffini Pacinian	Posterior capsule Entire length of ACL
Yahia 1991 [387]	Light microscopy	Dog	Ruffini Pacinian	Middle ACL
Denti 1994 [81]	Gold chloride	Sheep	Pacinian Free nerve endings	ACL reconstructed ligament
Aune 1996 [18]	Immunohisto-chemistry	Rats	Neural markers at 4 weeks	BPB autograft
Fromm 1994 [117]	Immunohisto-chemistry	Rabbit	Ruffini	Subsynovial ACL
Fromm 1994 [117]	Immunohisto-chemistry	Rabbit	None	ACL autograft
Tsujimoto 1993 [353]	Immunohisto-chemistry	Goat	Ruffini FNE	ACL ACL autograft
Sjolander 1989 [324]	Gold chloride	Cat	Type I, II, III, IV	Insertions ACL

Table 2 - Identification of neural structures within the anterior cruciate ligament in humans. FNE – Fine Nerve Endings; ACL – Anterior Cruciate Ligament; LCL – Lateral Collateral Ligament; BPB – Patellar Tendon.

Paper	Technique	Subject	Receptor Type	Location
Kennedy 1982 [202]	Silver stain	Human	FNE	Tibial origin of ACL
Halata 1989 [149]	Light and Electron Microscopy	Human	Pacinian Ruffini FNE	Subsynovial entire length
Georgoulis 2001 [130]	Gold chloride	Human	Mechanoreceptors FNE	ACL Remnant
Adachi 2002 [5]	Gold chloride	Human	Mechanoreceptors	ACL Remnant
Amir 1995 [12]	Gold chloride	Human	Ruffini Pacinian Golgi	Subsynovial Entire length Incr. In OA
Haus 1990 [157]	Light microscopy	Human	Ruffini Pacinian FNE	Subsynovial Entire length
Biedert 1992 [36]	Silver stain	Human cadaver	FNE	ACL
Zimny 1986 [395]	Gold chloride	Human	Ruffini Pacinian FNE	ACL localized to insertion points
De Avila 1989 [78]	Gold Chloride	Human	Spray shaped	LCL
Schutte 1987 [311]	Gold chloride	Human	Ruffini Pacinian Free nerve endings	ACL – tibial insertion
Schultz 1984 [309]	Gold chloride	Human	Golgi Free nerve endings	Surface of ACL
Denti 1994 [81]	Gold chloride	Human	All receptors	ACL stump
Aune 1996 [18]	Immunohisto-chemistry	Human	All neural markers except substance P	BPB autograft
Rivard 1993 [293]	Immunohisto-chemistry	Human	Ruffini Pacinian, FNE	Throughout ACL

Neurophysiology – Joint afferents

The identification of sensory receptors in the ACL changed current thinking of the ligament as a purely mechanical structure, passively preventing translation of the tibia on the femur [187]. The possibility of the ACL performing a neurosensory function and contributing to motor control was becoming a more plausible likelihood, at least from an anatomic standpoint. Thus, the task began to determine the physiological significance of these joint and ligament receptors.

Gomez Barrena et al. [134] injected HRP-WGA (horseradish peroxidase - wheat germ agglutinin) into the ACL of cats. They mapped fibres of the articular nerves to the dorsal root ganglia of lumbar segments 5-7. These segments innervate both the sciatic and femoral nerves suggesting that receptors in the ACL influence both the knee flexors and the knee extensors.

A number of studies demonstrated that stimulation of knee joint afferents led to a modulation in the efferent output to the muscles that control the knee [24, 111, 227, 330]. Freeman and Wyke [111] demonstrated that knee joint afferents contribute to spinal reflexes by observing that partial neurectomy of articular nerves caused changes in the quadriceps reflex. Baxendale et al [24] found that mechanical stimulation of the posterior joint capsule led to increased motor unit discharge of the quadriceps, an effect that was abolished by local anaesthetic. Lundberg et al [227] stimulated the knee joint afferent nerve in cats and recorded in the sciatic nerve. They found that joint receptor afferent signals facilitate Ib transmission and thereby influence reflexes initiated by Golgi tendon organs via Ib interneurons. Skoglund [330] found that stimulation of articular nerves caused an increase in quadriceps tension.

Studies in animals have demonstrated that the application of a direct load to the ACL will cause a change in the electromyographic (EMG) activation of the knee flexors and extensors [209, 250, 290, 335]. Solomonow et al [335] evaluated EMG from the knee flexors and extensors of a cat while applying direct load to the ACL. With high loads they documented greatly increased EMG in the knee flexors with decreased activity in the knee extensors. Krauspe et al [209] made electrophysiological recordings from the sciatic nerve of cats. They were able to identify a subpopulation of myelinated afferent fibres in PAN that had receptors in the ACL. These consisted of mostly Group II (79%) fibres with less Group III (12%), and Group IV (8%) fibres. They found that the afferent neurons from the ACL displayed no firing at rest, but increased firing with extension and internal, external rotation of the knee, motions that stressed the ACL. Raunest et al [290] recorded EMG of the quadriceps and hamstrings in sheep, then stressed the ACL with multiple loads, multiple speed of loading, and multiple joint angles. They found that loading of the anteromedial band of the ACL caused increased hamstring EMG and inhibited quadriceps EMG. The pattern was similar with static or dynamic loading and independent of the magnitude of the load. Miyatsu [250] studied loading of the ACL in unanaesthetised decerebrate-spinalised cats and dogs and found this had an excitatory effect on the knee flexors and extensors. Others have tried to demonstrate an ACL-hamstring reflex in animals but have been unsuccessful [281].

Several authors have studied the effect of ACL trans-section on the efferent output of the knee flexors and extensors with certain movements [136, 154, 204].Hasler et al [154] demonstrated that trans-section of the ACL caused unloading of the hind limb with decreased knee extensor activity and prolonged knee flexor activity prior to touchdown

compared to before trans-section. Gomez-Barrena et al [136] recorded increased knee flexor and extensor EMG activity in response to movements of the knee joint. Khalsa et al [204] examined the effect of ACL trans-section on the firing patterns of joint afferents and found that the responsiveness of the neurons was not changed significantly by cutting the ligament. Johansson [187] demonstrated that activation of mechanoreceptors had only weak effects on alpha motoneurones but exhibited powerful effects on motor output via fusimotor neurons (gamma motoneurones) through the effects on the muscle spindles afferents.

Studies to demonstrate a ligament-muscle reflex in humans have been more difficult to perform. Solomonow [335] demonstrated inhibition of the quadriceps and excitation of the hamstrings when the ACL intact knee was stressed. This inhibition remained however, when the ACL deficient knee was stressed. Tsuda et al [352] inserted electrical wires into the ACL of un-anaesthetized subjects with no knee injury. They demonstrated increased hamstring (biceps femoris and semitendinosus) EMG activity after stimulation of the ACL. The reflex latencies were between 50-80 ms and between 110-180 ms. They then anaesthetized the knee with xylocaine and observed no increase in hamstring EMG with ACL stimulation [352]. Dyhre-Poulsen et al [92] performed a similar procedure and were able to elicit semitendinosus contractions in response to stimulation of the intact ACL in the relaxed knee. This reflex contraction had a latency of 95 (35) ms. They repeated the experiment during maximal isometric contraction of the knee extensors and then the knee flexors. They demonstrated total inhibition of the hamstring muscles during a maximal knee flexor contraction with a latency of 65 (20) ms. Inhibition of the knee extensor muscles during a knee extension was seen with a latency of 70 (15) ms.

These long latencies are indicative of polysynaptic long loop reflexes and are therefore unlikely protective for the ACL. Grabiner [137, 138] has been unable to identify an ACL-muscle reflex in humans.

Tsuruike and Koceja [354] attempted to determine if the persistent knee extensor strength deficit after ACL reconstruction with a patella tendon autograft was associated with any changes in the segmental monosynaptic stretch reflex. They found similar reflex profiles in both the ACL reconstructed leg and the contralateral uninjured limb.

Biedert [37] attempted to demonstrate a patellar tendon autograft ACL to hamstring reflex. They measured EMG while pulling on the ACL in controls and the graft in the surgical subjects. Increased EMG signal was detected in the hamstrings of both groups when a Lachman test was performed. However, they were unable to detect hamstring EMG in response to the direct ACL manipulation in either group even though most subjects felt pain on the manipulation.

In more functional testing, EMG studies have demonstrated altered muscle recruitment in ACL deficient subjects [27, 190, 214, 335, 350, 382]. Wojtys and Huston [382] looked at neuromuscular control in a group of ACL deficient subjects (index and contralateral limbs) and compared to a control group. They measured EMG response to anterior tibial translation and demonstrated that acutely (<6 months) ACL deficient individuals used an altered muscle recruitment order and had an increased hamstring activation in response to anterior tibial translation. Those who recruited hamstrings first had decreased laxity between tense and relaxed states and had better subjective functional scores. Tibone et al [350] demonstrated that ACL deficient subjects had decreased floor reaction forces during running and cutting. Beard et al [27] found that the ACL deficient group had a significantly

longer reflex hamstring contraction than control and this correlated to functional instability (r=0.62) but not to KT1000 objective instability (r=0.26). Solomonow et al [335] studied hamstring and quadriceps EMG during concentric knee flexion and extension. In the ACL deficient subjects, there was an episode of decreased ability to generate moment that occurred at 37-46°. During this "moment failure", there was a burst of hamstring activity. This was presumed to be the hamstrings attempting to stabilize the joint. Kålund et al [190] studied walking up different grades in ACL deficient subjects. They measured EMG of the hamstrings and quadriceps and found that in uphill walking, the hamstrings were activated earlier in the ACL deficient subjects than in the controls. These findings were confirmed by Lass et al [214].

From these studies it is clear that the intact ACL performs a neurosensory function and that this is altered in the ACL deficient and ACL reconstructed states. Neuromuscular changes that occur in the pathologic state serve to help stabilize the unstable knee by modifying firing patterns in the hamstrings to enhance their ability to act as an ACL agonist. Decreased knee extensor activation will decrease anterior shear forces on the tibia.

Proprioception in the Knee

Afferent receptors are present, not only in the actual ACL, but also in the peri-articular tissues, especially the posterior capsule. These receptors may also be influenced by ACL rupture and reconstruction, as well as by instability of the knee joint. Joint receptors have been studied primarily with respect to their contributions to joint position sense and activation of the knee flexors and extensors. Two schools of thought exist, one

that joint receptors signal joint position sense throughout the full range of motion [44, 105] and the other that joint receptors act as limit detectors only at the ends of range of motion [50, 142, 144]. Regardless, it appears that joint receptors do contribute in some fashion to position sense in the knee [227].

ACL deficient subjects have been shown to have decreased kinaesthesia (ability to detect motion) [21, 71, 272] and position sense (the ability to match the angle of one leg to another) [71, 115] than controls. This decrease in proprioceptive ability was correlated to strength measurements [21, 71]. Adachi et al [5] correlated the number of mechanoreceptors in the ACL remnant to joint position sense. After ACL reconstruction, joint kinaesthesia [66] and joint position sense [267] seems to improve. Ochi et al [267] examined somatosensory evoked potentials (SEP) in ACL deficient, hamstring ACL reconstructed knees and control knees and attempted to correlate them with instability and position sense. They found that the number of reproducible cortical SEP's was 15/32 for the ACL deficient, increased to 22/23 in the ACL reconstructed, and 14/14 in the controls. There was an inverse correlation with SEP voltage and side to side instability testing in the ACL deficient and ACL reconstructed groups (r=.609). Joint position sense improved significantly with ACL reconstruction but they were unable to significantly correlate this with SEP voltage.

Valeriani et al [358] recorded SEP's from a group of controls and ACL deficient human patients. They correlated a kinaesthetic deficit with SEP abnormalities. They concluded that central somatosensory pathways are functionally modified in humans by lesions to peripheral mechanoreceptors. In a subsequent study, Valeriani [357] retested seven patients who demonstrated both kinaesthetic and SEP abnormalities after ACL

reconstruction with the medial third of patellar tendon. They found no change in kinesthetic sense or SEP pattern before and after surgery. Clark et al. [63] examined both position sense and kinaesthesia in humans with no knee injury. They found that anaesthesia of the joint and skin had no effect on the subjects' ability to perceive small changes in position. Position sense was influenced by joint anaesthesia in that there was a small shift towards increased extension after injection.

In vivo documentation of neurosensory function of the ACL and other intra-articular structures of the knee in humans was performed by Dye et al [90]. They performed knee arthroscopy with no anaesthesia and documented conscious sensation of force placed at different loads to the various structures in the knee. In the ACL, slight and poorly localized discomfort was felt in the mid-portion, and moderate to severe, poorly localized pain was felt when force was placed at the tibial or femoral insertions.

From both animal and human studies, it seems clear that the afferent input originating from the knee joint, be it ligamentous, capsular, or other does exert some influence in the activation of the muscles that control knee motion. Altered afferent feedback from the knee after ACL reconstruction could arise from the graft harvest site, loss of ACL afferent receptors, residual instability of the knee joint, or from the effects of the surgical procedure (notchplasty, tunnel drilling etc). There is evidence that alteration in the afferent feedback from the knee joint contributes to regional strength deficits.

Anterior Cruciate Ligament Injury and Reconstruction

ACL injuries are common in active individuals and arise from sporting, recreational, and vocational activities. The mechanism of injury is a sudden deceleration or pivoting maneuver (valgus, tibial internal rotation). Symptomatic individuals will complain of giving way or instability during cutting or pivoting movements. The current standard of care for active, symptomatic patients with a ruptured ACL is the arthroscopically-assisted intra-articular hamstring or patellar tendon autograft reconstruction [110, 196].

For the studies in this thesis, ACL reconstruction surgery was performed under routine general or spinal anaesthetic with injection with 20 cc of local anaesthetic (Marcaine/Xylocaine) for pre-emptive analgesia. An examination under anesthesia was performed on all patients. The range of motion, degree of laxity and presence or absence of a pivot shift was documented for both knees. After appropriate prepping and draping, all patients underwent a standard diagnostic arthroscopic examination, including evaluation of all three compartments of the knee. The presence of a torn ACL was confirmed during arthroscopy and meniscal surgery, if necessary, was performed at this time. Both procedures (hamstring and patellar tendon) were arthroscopically-assisted, one incision techniques performed under tourniquet control.

Hamstring ACL Reconstruction

Semitendinosus and gracilis tendon harvest was performed on the ipsilateral limb. A 2-3 cm longitudinal incision was made one thumbs breadth medial to the tibial tuberosity. The subcutaneous tissue was dissected down to the sartorius fascia. The tendons were palpated beneath the fascia. The sartorius fascia was incised in line with the

palpable tendons just superior to the gracilis. The tendons were sequentially grasped with a tendon hook, cleared of fascial attachments and stripped using an open-ended tendon stripper. The tendons were removed from the tibia in a conjoined fashion with a strip of tibial periosteum and brought to a table for preparation. In the cases where a ligament augmentation device (LAD) was utilized, the tendons were sutured to an LAD in a sandwich fashion. An absorbable 2- 0 tubing suture was used and great effort was made to bury the LAD. Interrupted 2-0 nonabsorbable suture was used for the tibial tunnel portion of the graft, where the suture fixation of the LAD to the graft was thought to be crucial. A tendon leader was applied, and the graft was set aside in a moist environment.

A 5.5 Cuda shaver (Linvatec, Largo FL) was used to clear soft tissue off of the over-the-top position so that it could be well-visualized (modified notchplasty). The tibial stump was cleared enough to allow for adequate visualization but an effort was made to preserve the tibial stump of the ACL for possible later ingrowth into the graft. A tibial guide was used to insert a tibial guide wire into the posterior part of the tibial footprint at a 35° angle to the horizontal. Position of the guide wire was checked in both flexion and extension to ensure good positioning. If the guide wire was appropriately positioned, the tibial tunnel was reamed 1 mm under the measured diameter of the graft. A shaver was placed up the tibial tunnel to clear it of soft tissue and a rasp was used to smooth the tunnel edges.

A femoral guidewire was drilled in the 10:30 or 1:30 position (depending on the limb) at the back of the notch approximately 6 mm anterior to the over-the-top position. The guide wire was drilled through the femur and out the lateral aspect of the thigh. The guide wire was over-reamed to 30 mm by an appropriate sized reamer. The graft was

threaded through the tunnels and fixed using a bioabsorbable interference screw. The graft was then pre-tensioned by flexing and extending the knee ten to fifteen times. The tibial side was fixed with the knee in full extension with a second interference screw. The graft was checked through the arthroscope in both flexion and extension to ensure proper positioning. The wounds were then well irrigated and closed in layers using absorbable sutures.

Patellar Tendon ACL Reconstruction

An incision was made just proximal to the inferior pole of the patella, continuing distally to the tibial tubercle. The central 1/3 of the patellar tendon was harvested, so as not to exceed 40% of the entire tendon bulk. A bone saw and osteotome were used to remove the bone plugs from the inferior patella and proximal tibia. Once harvested the bone plugs were sized with tunnel sizers and drilled, to accommodate sutures and fixation. Once the femoral and tibial tunnels were drilled, the graft was inserted through the tibial tunnel and directed proximally across the joint into the femoral tunnel. Endobutton fixation secured the graft at the lateral femoral cortex. The graft was tensioned and fixed within the tibial tunnel by an interference screw. Bone graft was placed into the defects left in the patella. A standard deep and superficial soft tissue closure was performed and a sterile dressing is placed over the wound.

ACL Reconstruction Outcomes

The primary goal of ACL reconstruction surgery is to provide a stable knee that enables patients to return to their previous level of activity and function. Radiographic analysis of ACL deficient and ACL reconstructed subjects ascending an 8 cm step has

demonstrated that ACL reconstruction restores anterior-posterior stability to the knee joint [16, 45]. It has been difficult to determine reliable objective measures of the success of ACL reconstruction. Isovelocity strength measurement of the knee flexors and extensors has traditionally been one of the more objective and reproducible measurements used to evaluate the success of ACL reconstruction.

Follow up studies on subjects who have had an ACL reconstruction show that the level of self-reported return to function is related to knee extensor strength [19, 295]. Other studies have demonstrated that good recovery of knee extension strength is the strongest determinant of patient satisfaction after ACL reconstruction [255]. Functional outcome has been demonstrated to have a positive correlation with strength measurements [314, 378]. Bush Joseph et al [52] related improved performance in high demand activity with quadriceps muscle strength. These studies indicate that knee extensor strength is a prerequisite to functional recovery after ACL reconstruction.

After patellar tendon ACL reconstruction (Table 3), strength testing of the knee extensors has consistently demonstrated that subjects have a 5-34% strength deficit compared to the contralateral limb at minimum one year follow-up [19, 109, 295, 300, 319, 349, 389]. Strength testing of knee flexors has shown strength equal to that of the contralateral limb [319, 350, 388, 390].

After hamstring tendon ACL reconstruction (Table 4), a knee extensor strength deficit of 9-17% has been demonstrated compared to contralateral leg. Early studies did not detect a knee flexor deficit [194, 225, 390], probably because of poor dynamometry techniques. Newer studies have consistently demonstrated a knee flexor deficit in this group when compared to control [164, 234, 258, 381].

Table 3- Review of Patellar Tendon ACL reconstruction outcome studies that include strength testing.

Paper	Number of subjects	Rehabilitation Protocol	BMN	Grav	ECC	Control	Angular Velocity (°/s)	Strength relative contra-lateral limb
Tibone 1988[349]	11 2 years	ROM - 6 weeks WB – 6 weeks	X	X	X	Contra	60/80/300 60/80/300	83-87% KE 95-102% KF
Sachs 1989 [300]	 1 yr	ROM - 6 weeks WB - 6 weeks	X	X	X	Contra	60 60	66.2% KE 88.1% KF
Shelbourne 1990 [319]	155 >2 yrs	ROM – early WB – early	X	??	X	Contra	60, 180, 240	90% KE 98% KF
Rosenberg 1992 [295]	10 1-2 years	ROM – early WB - 6 weeks	√	X	X	Contra	60/180/300 60/180/300	82% KE 90-100% KF
Bach 1994 [19]	62	ROM – early WB – 6 weeks	X	??	X	Contra	60 180 240	88% KE 91% KE 93% KE
LoPresti 1988 [226]	13	ROM - 4 weeks WB – 4 weeks	X	X	X	Contra	60/120/240 60/120/240	88-90% KE 100% KF
Beard 2001 [28]	30	ROM – early WB – early	X	X	X	Contra	??	81% KE 95% KF
Osternig 1996 [270]	14 >1 yr	??	X	√	√	Contra	60	87% KF
Aglietti 1994 [6]	30	ROM - early WB – early	X	X	X	Contra	60/120/180 60/120/180	91-94% KE 95-99% KF
Harter 1990 [151]	30	??	X	√	X	Contra	120	85.6% KE 95.2% KF
Marder 1990 [234]	72	ROM - early WB – early	X	??	X	Contra	60 60	88% KE 91% KF
Lephart 1993 [221]	15	ROM – early WB - 6 weeks	X	X	X	Contra	60 240	95% KE
Shelbourne 1993 [321]	33	ROM – early WB – early	X	??	X	Contra	180 180	83% KE 98% KF
Keays 2000 [198]	31	??	X	X	X	Contra	60/120 60/120	72-78% KE 94-97% KF
Carter 1999 [58]	38 6 months	WB early ROM – early	X	??	X	Contra	180 300	70% KE 82% KF
Feagin 1997 [103]	69	½ immobilized ½ early ROM/WB	?	??	X	Contra	60	96% KE 100% KF
Bush Joseph 2001 [52]	22	ROM – early WB – early	√	??	X	Contra	60 180 240	Sig lower KE 100% KE 100% KE 100% KF
Witvrouw 2001 [381]	17	ROM – early WB – early	X	X	X	Contra	60/240 60/240	77-80% KE 93-98% KF
Beynnon 2002 [346]	28	ROM - 1 week WB - 3 weeks	X	??	X	Contra	60/180/240 60/180/240	84-88 KE 99-103KF

BMN – Body Mass Normalization; Grav– Gravity corrected for the moment of the weight of the limb; ECC – Eccentric Contractions Tested; ROM – Range of Motion; WB – Weight Bearing; X – not performed; √ - performed; ?? unknown or unclear; Contra – Contralateral limb; KE – Knee Extensors; KF – Knee Flexors.

Table 4 - Review of hamstring tendon ACL reconstruction outcomes that include strength testing.

Paper	Number of subjects Follow-up	Rehabilitation Protocol	BMN	Mom Corr	ECC	Control	Angular Velocity (°/s)	Strength relative contralateral limb
Lipscomb 1982 [225]	51	ROM - 6 weeks WB – 12 weeks	X	?	X	Contra	60/240 60/240	95-98% KE 100% KF
Colombet 2002 [70]	200	ROM - early Full WB	??	??	X	Contra	??	83% KE 95% KF
Marder 1991 [234]	72	ROM – early WB - early	X	??	X	Contra	60	91% (19) KE 83% (16) KF
Aglietti 1994 [6]	30	ROM - early WB – early	X	X	X	Contra	60/120/180 60/120/180	89-95 % KE 92-96 % KF
Harter 1990 [151]	16	??	X	√	X	Contra	120	87.6% KE 91.2% KF
Sachs 1989 [300]	1 yr	ROM - 6 weeks WB - 6 weeks	X	X	X	Contra	60 60	66.2% KE 88.1% KF
Keays 2001 [199]	31 6 months	ROM - 2 weeks WB – early	X	??	X	Contra	60 120	88% KE 90% KF
Feagin 1997 [103]	68	½ immobilized ½ early ROM/WB	??	??	X	Contra	60	96%KE 100% KF
Carter 1999 [58]	68 6 months	WB early ROM – early	X	??	X	Contra	180 300	77.7% KE 79% KF
Nakamura 2002 [258]	74 2 yrs	ROM – 1 week WB – partial 3 weeks, full at 5 weeks	√	??	X	Contra	60 180 60 180	83.1(15) KE 89.5(13.9) KE 91.3(16.1) KF 86.1(17.6) KF
Beynnon 2002 [34]	28 12 months	ROM - 1 week WB - 3 weeks	X	??	X	Contra	60 /180/240 60/180/240	85-87 % KE 96-99% KF
Beard 2001 [28]	3 1 year	ROM – early WB – early	X	X	X	Contra	??	81% KE 87% KF
Witvrouw 2001 [381]	32	ROM – early WB – early	X	X	X	Contra	60/240 60/240	84-85% KE 86-89% KF
Majima 2002 [232]	62	ROM – early WB – early	??	??	X	??	60	100% KE 100% KF
Maeda 1996 [230]	41	ROM - cast 10d WB - partial 3 weeks, full at 5 weeks	X	X	X	Contra	60 180 60 180	90% KE 90% KE 95% KF 95% KF
Karlson 1994 [194]	64	WB - 6 weeks ROM – early	X	X	X	Contra	60	95% KE 106% KF
Kramer 1993 [207]	30	ROM -early WB - 6 weeks	X	√	√	Contra	60 180	Significantly decreased

BMN – Body Mass Normalization; Grav– Gravity corrected for the moment of the weight of the limb; ECC – Eccentric Contractions; ROM – Range of Motion; WB – Weight Bearing; X – not performed; √ - performed; ?? unknown or unclear; Contra – Contralateral limb; KE – Knee Extensors; KF – Knee Flexors.

Moment arm

Changes in strength can be of neural, muscular or mechanical origin. Harvest of the semitendinosus and gracilis has been demonstrated to alter the force generating capacity of the knee flexors [164, 234, 258, 381]. Primary muscle relationships, such as force-length and force-velocity are a function of the muscle itself. These physical characteristics are maintained during the time of the strength test. When a muscle is activated in a consistent manner such as through electrical stimulation, the moment-angle and moment-angular velocity relationships would be the angular correlates of the force-length and force-velocity relationships, that being they would largely reflect the muscle system. The only other major mechanical relationship that would effect the moment/angle and moment/angular velocity relationship would be the moment arm of muscle/joint angle relationship.

The moment that a muscle is able to generate is dependent on the force generated by a muscle multiplied by its moment arm. The moment arm is the perpendicular distance of the line of action of muscle force to the instant centre of joint rotation. The resultant joint moment (RJM) represents the sum total of all muscular, tendinous, ligamentous, cartilaginous, capsular, and bone-on-bone forces acting about the instantaneous axis of rotation of the joint. Knowledge of the moment arms of the knee extensors (patellar tendon) and the knee flexors (hamstrings), especially the semitendinosus and gracilis are necessary in order to theorize as to the changes that may occur in the resultant knee joint moment (RJM_K) after ACL reconstruction with a semitendinosus autograft. One possible effect of semitendinosus tendon harvest would be to change the moment arm/joint angle relationship of the semitendinosus muscle. Given a similar neural activation strategy, a

decrease in moment arm of the semitendinosus would result in a knee flexor strength deficit. If this were the case, the mechanical effect would be uniformly applicable to both the concentric and eccentric areas of a strength map (see below) and for all speeds of contraction.

The flexor moment generated by the hamstrings is the result of the combined moments of all muscles capable of flexing the knee. The moment arms of the hamstring muscles (biceps femoris, semitendinosus, and semimembranosus) change with different joint angles and the moment arms for the three hamstring muscles do not peak at the same angle [47, 161, 175, 338]. EMG data was collected from human subjects during flexion and extension with and without varus and valgus loading at six different joint angles [46]. It was found that semitendinosus has a primarily flexion moment angle and flexion EMG direction that was independent of joint angle. Gracilis, however, had activation during flexion as well as valgus directed loads and its EMG direction changed with knee joint angle. Harvest of the semitendinosus may alter the angle at which the combined moment arm of the knee flexors is the largest, thus altering the moment generating capacity of the knee flexors at certain angles.

Methods

The following section describes the general methodology common to the subsequent investigative studies. Specific protocols are described within each manuscript.

Knee Strength Tests

Dynamometry Evaluation

An isovelocity dynamometer (Kin-Com 500H, Chattecx Corporation, Hixson, TN) was used to evaluate knee flexor and extensor strength. This microcomputer-controlled, hydraulically driven device uses strain-gauge transducers to measure force, a tachometer to determine angular velocity of the actuator arm, and a potentiometer to detect angular position of the actuator arm. The Kin-Com dynamometer has been shown to be reliable with a reliability index of 0.948 for dynamic tests [102] and a coefficient of determination r^2 > 0.99 for comparisons with known measures of force, angle and velocity. Intraclass correlation coefficients (ICC) for between-day comparisons were >0.99 demonstrating a high degree of agreement between test days [241]. Intrarater test-retest reliability for the protocol used in these studies was performed previously [367]. The ICC was equal to 0.865 and Pearson's product moment correlation coefficient (r) was equal to 0.841. A one-way analysis of variance (ANOVA) was performed to determine whether time (initial test or second test) had any effect on the mean moments produced. No significant effect of time was revealed.

Warm –up

Subjects warmed on a cycle ergometer (35 Watts, 7-8 N at 70 revolutions per minute) for 5 minutes.

Subject Positioning and Stabilization

Subjects were seated on the dynamometer with the back of the dynamometer chair tilted to 110 °. The lateral femoral condyle was aligned with the axis of rotation of the actuator arm of the dynamometer. The distal thigh was stabilized using a Velcro strap. The leg was attached to the actuator arm of the dynamometer with a Velcro strap at a position that was comfortable approximately 1-5 cm above the medial malleolus. A seat belt was secured around the subject's waist. A strap stabilized the trunk against the seat. Both knee extension and knee flexion strength tests were performed in the seated position.

Evaluation of Knee Extensor and Knee Flexor Strength

Isovelocity (constant angular velocity) strength tests consisted of 3 maximal effort concentric and eccentric contractions at 5 speeds (50, 100, 150, 200 and 250 °/s). Range of motion of the dynamometer was set to 90 ° from 5° to 95° of flexion. Zero was defined as knee joint full extension. The medium acceleration setting on the dynamometer was used. This limits the maximum angular acceleration achieved when the dynamometer increases speed to the specified constant angular speed from a stationary position. Constant angular velocity is nominally achieved within 5 °.

Subjects were familiarized to the strength tests by performing 3 to 6 sub-maximal effort (less than 50% effort) concentric and eccentric repetitions at each speed prior to maximal effort testing. A concentric contraction was followed by an eccentric contraction with a five second pause provided between successive contractions at each velocity to

permit the subject to focus on the specific contraction required. Three repetitions were performed at each velocity with a two minute rest given between velocities.

Verbal Prompting

A consistent form of enthusiastic verbal prompting was given during the isovelocity strength tests.

Data Collection, Reduction and Analysis

The dynamometer's force transducer, tachometer, and potentiometer are interfaced with an on-board computer equipped with an A/D converter that samples the generated signals at a rate of 100 Hz. The moment arm of the dynamometer force transducer was recorded for each subject. Raw data consisting of time, angular position (°), angular velocity (°/s), force (N), and moment arm (m) was exported from the dynamometer's computer. This raw data was processed using Excel (Version 2000) and Isomap Dynamometry Software (Isodyne Inc., Winnipeg, MB). The dynamometer moment (Nm) was calculated as the product of force (N) and moment arm (m). Angular acceleration was derived from the unfiltered angular velocity waveform by numerical differentiation. Moments generated during concentric contractions were assigned positive angular velocities and moments generated during eccentric contractions were assigned negative velocities.

Correction for the Moment of the Weight of the Leg, Foot and Resistance Pad

Correction for the moment of the weight of the limb and resistance pad for all isovelocity strength tests was performed post hoc based on the estimated mass of the leg and foot derived from body segment parameter data [98].

Strength Parameters

Repetitions (one reciprocal repetition represented 2 contractions, an eccentric and a concentric) were derived using an angular acceleration threshold of $\pm 300°/s^2$ to identify regions of isovelocity data. Analysis of data was restricted to these regions of low or near zero acceleration (i.e. constant velocity sections) motion. An acceleration threshold of this magnitude would result in an absolute error in knee joint moment of less than 5 Nm (using the standard Newtonian equations of motion with an estimate of the moment of inertia of the leg and foot derived from typical body segment parameter data).

The following parameters were derived automatically by computer software; peak moment (Nm), angle of peak moment (°), work per repetition (J) and velocity at peak moment (°/s). Mean peak moments (average of peak moments from 3 repetitions); absolute peak moments (peak moment with largest magnitude from 3 repetitions) were determined for all concentric and eccentric contractions at all speed for the knee flexor and knee extensor strength tests.

Hip Strength Tests

Isovelocity hip extensor strength tests were performed in the standing position with the subject leaning slightly forward, stabilizing the torso on the dynamometer. The half seat of the dynamometer was removed to allow for hip flexion. The centre of rotation of the hip joint, as determined by palpation of the greater trochanter, was lined up with the centre of rotation of the actuator arm of the dynamometer. The actuator arm was strapped in a comfortable position to the posterior aspect of the subject's thigh. Subjects were familiarized to the strength testing protocol by performing 3 to 6 sub-maximal effort (less than 50% effort) concentric and eccentric repetitions at each speed prior to maximal

effort testing. Testing consisted of three maximal effort concentric and eccentric contractions at the two speeds. Strength was measured over a 50° range of motion (20° hip flexion to 30° hip extension) with neutral corresponding to the thigh being in line with the torso. A five second pause was provided between successive concentric and eccentric contractions at each velocity. A two minute rest was given between test velocities.

Isometric hip adductor strength testing was performed with the subject standing facing the actuator drive of the dynamometer. Subjects stood on a box to elevate them to avoid the foot hitting the floor. The centre of rotation of the hip joint was lined up with the centre of rotation of the actuator arm. Subjects were familiarized to the isometric strength testing protocol by performing 3 to 6 sub-maximal effort (less than 50% effort) at each angle prior to maximal effort testing. Hip adduction strength tests were performed at hip joint angles of 15 and 30° of adduction. Care was taken to maintain the centre of rotation of the hip at the centre of rotation of the actuator arm. Three maximal effort isometric contractions, held for 5 seconds, were performed at each joint angle, alternating between angles. Average moment data was collected for the three repetitions.

IsoMaps

Strength analysis software (Isomap software, Isodyne Inc. Winnipeg, MB) permits a comprehensive analysis and portrayal of data derived from standard, multi-speed isovelocity dynamometry data. Strength maps provide objective, quantified, comprehensive and easily interpreted information about moment generating capacity throughout a sampled strength domain (over a large range of motion, over a broad range of speeds and for concentric and eccentric contractions). The three-dimensional

moment/angle/angular velocity relationship can be expressed in the form of a two-dimensional relief-type strength map (Figures 1 and 2).

Figure 1

Upper Panel – Standard resultant joint moment-angular velocity relationship for the knee extensors.

Middle Panel - A knee extensor strength map for a representative subject. The vertical axis is knee joint angle (5 to 95°). The horizontal axis is angular velocity (±250°/s). The color-coded scale bar is the moment derived from the dynamometer (RJMK in Nm). The percentage area of the map associated with each color range of RJM_K is shown to the right of the map. The total work (J) corresponding to the volume under the strength map is shown on the right.

Lower Panel - Moment/angle plots showing three repetitions for eccentric (left) and concentric (right) contractions for one test speed (±150°/s). The moment/angle data is the raw data used to generate the strength maps.

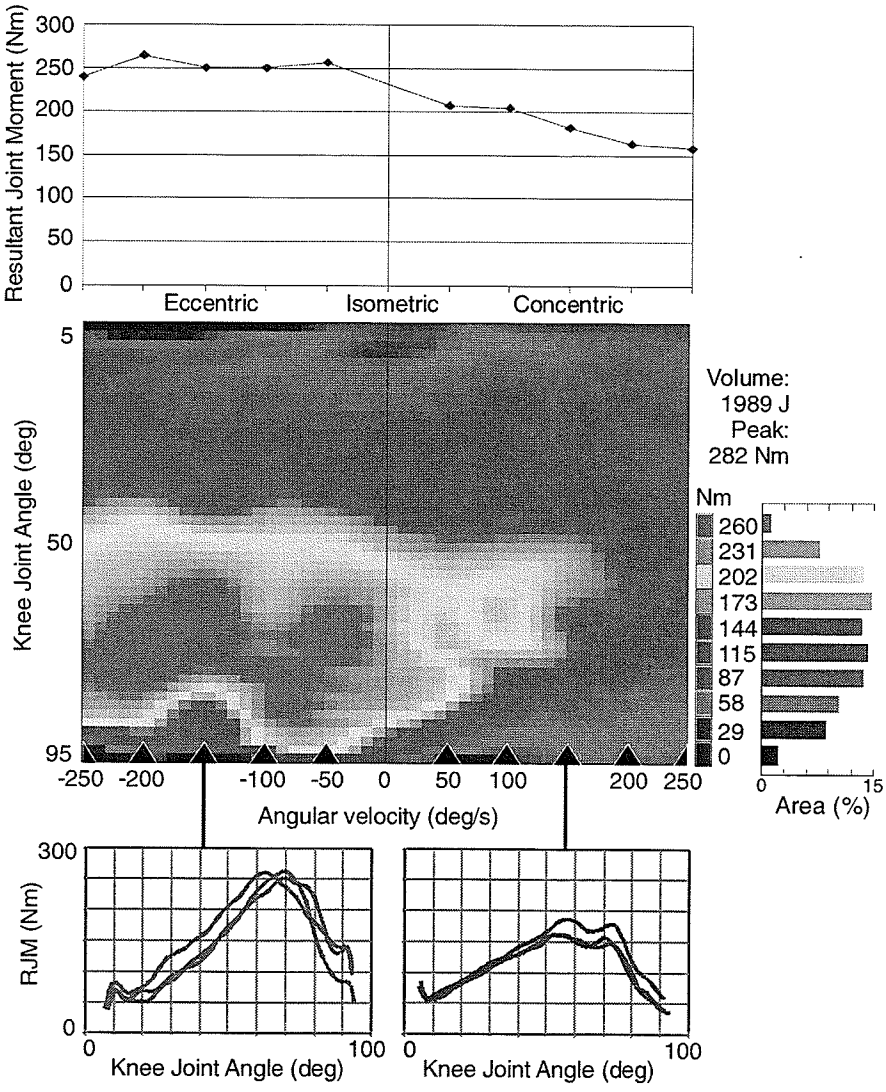
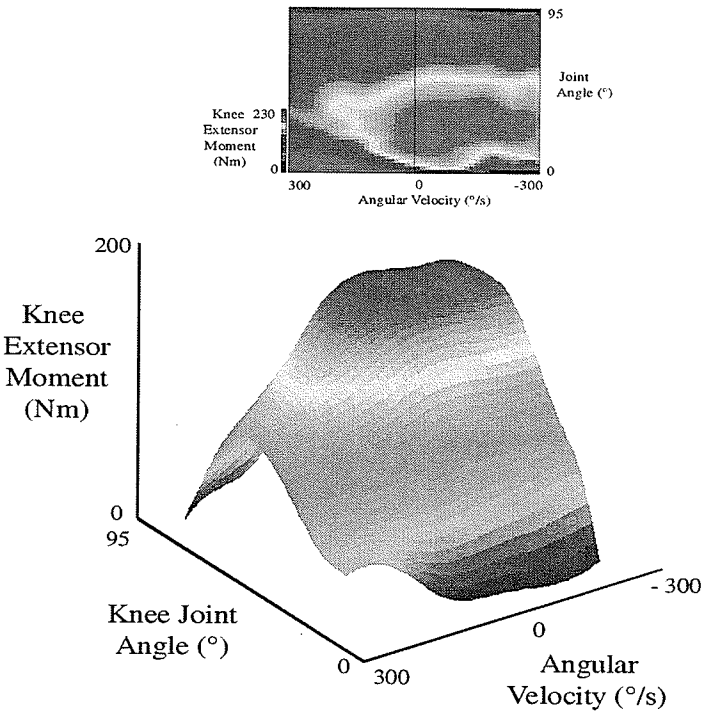


Figure 2 - Moment/Angle/Angular Velocity Relationship for the Knee Extensors comparing the two dimensional relief map to it's 3-dimensional correlate.



This allows visual inspection of strength maps that can be used to provide rapid qualitative detection of strength differences, as well as quantified differences over the entire sampled strength domain. The software automatically identifies regions of difference between groups, between individuals, or between right and left sides. The software locates the region of change (strength deficit or strength gain) by reporting the bounds in terms of range of motion, contraction type, and speed. This allows a more comprehensive portrayal of neuromuscular performance, allowing the evaluation of strength and strength deficits in post-operative or post-injury patients.

Strength Maps

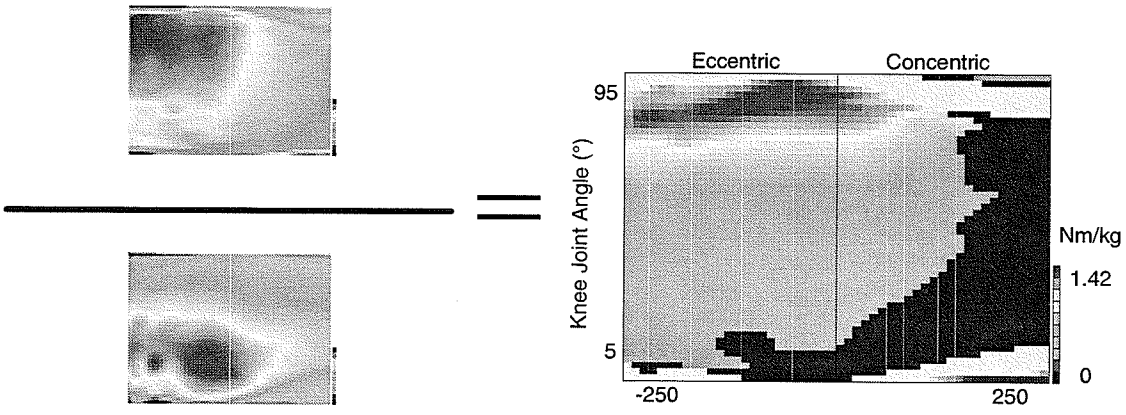
In the subsequent studies, strength maps were generated from standard dynamometry data from the isovelocity strength tests for knee flexion and knee extension using Isomap Dynamometry Software. Isomap uses a bicubic interpolating spline to fit all of the concentric and eccentric moment data to a 50 X 50 velocity/angle matrix. For the knee flexor and extensor strength maps the horizontal or x-axes (leg angular velocity) range from +250°/s to -250°/s with negative velocities representing eccentric contractions and positive velocities representing concentric contractions. The vertical or y-axes (knee joint angle) ranges from 5° to 95°. The color-coded values or z-axes (RJM_k) values will be body mass normalized (Nm/kg), with each relief level (i.e. each different color) representative of a ten percent increment relative to 98% of the maximum RJM_k moment of the map. This scaling will be produced automatically to generate consistent images (less sensitive to individual peak moment values). The volume (J) under the moment/angle/angular velocity surface will also be calculated and then body mass normalized (J/kg) for each individual knee flexor and extensor strength map. Knee flexor and knee extensor strength maps will be generated for each individual. Individual maps can be averaged to produce one body mass normalized map for the knee flexors and one for the knee extensors.

Difference Maps: The knee flexor and knee extensor "difference" maps can be generated between each time period or between matched groups to illustrate and quantify differences in strength. Difference maps are generated to allow for regional analysis of strength (strength differences specific to range of motion, contraction type, and velocity of movement). The difference maps highlight and quantify regions of greatest difference between groups.

Confidence Maps: "Confidence" maps can also be generated by performing 2500 velocity and angle matched t tests between the time periods. Each velocity and angle matched 1 X 1 grid will be accepted as being significantly different only if it is surrounded by at least 6 neighboring (and contiguous) grid squares that were also identified as different, and if the total contiguous area of difference is >= 6 squares. The benefit of using a minimum contiguous area of 6 squares is that it provides a means of ensuring that Type I errors are not allowed. The likelihood of having 6 contiguous Type I errors is less than 0.001. This provides ample protection for a large number of inferential tests (2500).

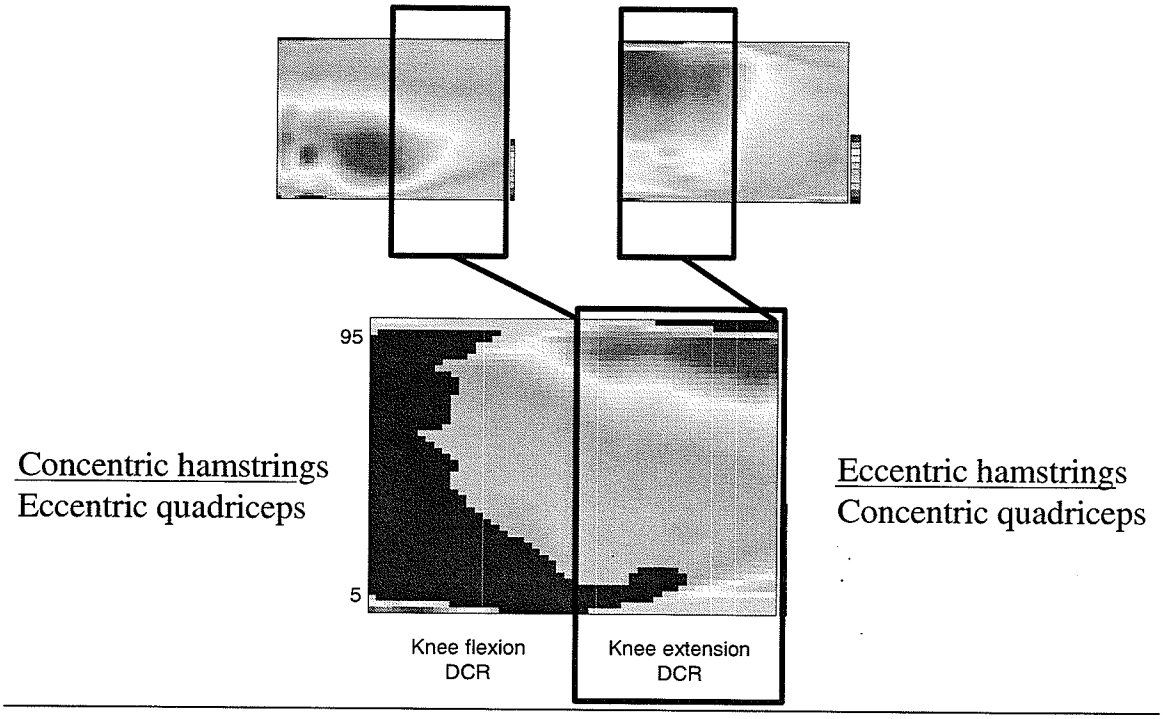
Ratio Maps: Knee flexor:extensor (H/Q) ratio strength maps were generated by dividing the average knee flexor map by the average knee extensor map for each group to provide an illustration of neuromuscular balance (Figure 3).

Figure 3 - Generation of a ratio map by division of the knee flexor map by the knee extensor map.



Dynamic Control ratio (DCR) maps were created by utilizing the eccentric knee flexor map divided by the concentric knee extensor map and the concentric knee flexor map divided by the eccentric knee extensor map. A standard ratio map then showed concentric hamstring/eccentric quadriceps on the left hand side and eccentric hamstring/concentric quadriceps on the right side (Figure 4).

Figure 4 - Generation of a dynamic control ratio map.



Map Quantification: All maps can be quantified by using threshold values (eg. .01 Nm/kg or 10%). The areas identified can be demarcated by range of motion, speed, and contraction type.

Mohtadi Quality of Life

The ACL Quality of Life Measurement (ACL-QOL) (Appendix A) is a patient-based, self administered, disease specific measure for chronic anterior cruciate ligament instability [251]. The ACL-QOL measure has been demonstrated to be valid, responsive to clinical change, and reliable [251]. This survey utilizes 100-mm visual analog scales on which patients rank their disease specific QOL in five domains: symptoms and physical complaints, work-related concerns, recreational activities and sports, lifestyle, and social and emotional well-being. The minimum and maximum scores are 0 and 100 respectively, where 0 is associated with severe symptoms, and 100 is associated with no symptoms. The ACL-QOL has been used in recent studies looking at post-operative outcomes in ACL reconstruction [100]. The use of this tool as a key outcome measure may help to elicit differences that directly affect the potential for patients to resume post-operative knee function suitable to their individual lifestyle requirements

KT-1000 Arthometry

Objective assessment of anterior-posterior displacement of the tibia relative the femur using a fixed load was performed using the KT1000 arthrometer (MEDmetric Corp. San Diego, CA). The KT1000 arthrometer measures anterior-posterior displacement of the tibia by measuring the movement of the tibial tubercle in relation to the patella. A side to side difference of greater than 3 mm predicts chronic ACL deficiency with a sensitivity of 99% and acute ACL deficiency with a sensitivity of 95% [289]. The relaxed limb was supported in 30° of flexion. The patellar sensor pad was stabilized on the patella with one hand; the other hand applied a strong anterior force to

the proximal calf. Three measurements were taken at maximum anterior displacement. Care was taken to prevent the knee from extending during the test. Displacement of the tibia was read off the dial and the average of three trials was used for analysis [76].

Lachman and Pivot Shift

The Lachman and Pivot shift tests were performed on all patients. The Lachman test is designed to assess anterior-posterior laxity of the knee. With a relaxed leg and the knee in 20-30° of flexion an anterior force was applied to the tibia. The amount of translation as well as the feel of the endpoint (firm or soft) was determined. The Lachman test was graded I (0-5 mm), II (5-10 mm), III (>10 mm). The pivot shift is a reduction maneuver. With the knee in full extension, an axial load, valgus and flexion maneuver was performed. As the tibia reduces from its subluxed position a glide or clunk was seen and felt. The pivot shift test was graded I (glide), II (clunk), III (locked out). In chronic ACL injuries, the Lachman test has been shown to have 84.6% sensitive and greater than 95% specific for diagnosing anterior cruciate ligament deficiency [197].

Results

This systematic investigation into changes in neuromuscular control will be presented in six manuscripts. Each manuscript section will include an introductory paragraph, a supplementary review of literature which will explore some key concepts in more detail than the manuscript. The manuscript will then be presented, followed by any supplemental data that was not included in the manuscript. A concise summary of results follows the manuscripts. Within the 6 manuscripts, a total of 105 subjects were evaluated with total subject visits of 132. Two of the manuscripts are reprints (with permission) from already published work. Two are in review by peer-reviewed journals and two are prepared for submission. These papers, as a group, have the **Resident Research Award,** Canadian Society for Clinical Investigation/Medical Research Council of Canada, 1999.

Study 1 - Graft Site Dependent Knee Strength Deficits after Patellar Tendon and Hamstring Tendon ACL Reconstruction

This study examines the strength of the knee flexors and knee extensors after two surgical techniques of ACL reconstruction and compares them to an age and activity level matched control group. In contrast to previous studies that compared strength after both types of autograft utilizing only single velocity concentric strength testing, we performed comprehensive strength analysis during both concentric and eccentric contractions, through a full functional range of motion and at five angular speeds. The goal of this study was to determine whether alteration in the flexor or extensor mechanism through ACL graft harvest would contribute to changes in neuromuscular activation patterns.

This manuscript has been published in *Medicine and Science in Sport and Exercise* 32(8): 1472-1479, 2000. Ethics approval for this study has been granted by University of Manitoba (E96:246). These papers have won **Frederick Robert Tucker Award for Orthopaedic Excellence,** Manitoba-Saskatchewan Annual Resident's Research Day, 1997; **Pennal Society Award,** Canadian Orthopaedic Resident's Association (CORA) meeting, Toronto, ON, 1997.

A review of the literature includes a discussion of the neuromuscular aspects of the strength test.

Strength Test is a Test of the Neuromuscular System

Strength assessment in humans involves eliciting maximal effort contractions of the prime movers, in this case the knee extensor and knee flexors. In vivo, muscle force is primarily dependent upon activation through the nervous system and the upper bounds of

force are limited by the mechanical output characteristics of the muscle (force/velocity and force/length relationships). Unlike the force/length or force/velocity relationships which are determined for isolated muscle during maximal activation evoked by electrical stimulation of the nerve, a strength test which involves the determination of the resultant joint moment/angle relationship is necessarily a test of the **neuromuscular** system, not directly of the properties of muscle itself.

Primary muscle relationships, such as force-length and force-velocity are maintained during the time of the strength test. If the muscle is activated in a consistent manner (i.e. through electrical stimulation), the moment-angle and moment-angular velocity relationships would be the angular correlates of the force-length and force-velocity relationships, that being they would largely reflect the muscle system. The only other major mechanical relationship that would effect the moment/angle and moment/angular velocity relationship would be the moment arm of muscle(s)/joint angle relationship. For example, during lengthening contractions of isolated muscle during maximal activation it has been clearly demonstrated that the muscle is capable of generating peak forces that are 1.5-2.0X greater than forces generated during an isometric contraction. However, during maximal **voluntary** contractions, it is evident from both strength and EMG data that neural activation is substantially limited during eccentric contractions [368, 374]. That is, the ratio of eccentric to isometric forces (those being greater than 1.5:1) for the linear muscle system should be preserved in the angular system, where the eccentric moments are 1.5X that of isometric moments. In fact, numerous publications have clearly revealed that maximal **voluntary** contractions exhibit eccentric moments **equal** to that of the isometric moment. Thus the moment-angular velocity relationship demonstrated during

voluntary contractions is not a steady state test in that the moment-velocity curves generated in isolated muscle are preserved in shape but not in magnitude. The only plausible explanation for this is the limitation of neural activation during eccentric contractions. This explanation is supported by studies which have evoked involuntary or "forced" lengthening contractions to induce 1.5:1 [368] or 1.25:1 [374] ECC/CON ratios in humans. Further, there are no known muscle properties which could account for these differences.

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Graft Site Dependent Knee Strength Deficits after Patellar Tendon and Hamstring Tendon ACL Reconstruction

Laurie A. Hiemstra†, Sandra Webber‡, Peter B. MacDonald† and Dean J. Kriellaars†‡

School of Medical Rehabilitation
Faculty of Medicine
University of Manitoba
Winnipeg, Manitoba
R3E 0W3

† Section of Orthopaedic Surgery, Faculty of Medicine, University of Manitoba, Winnipeg, Manitoba, Canada, R3E 0W3

‡ School of Medical Rehabilitation Faculty of Medicine, University of Manitoba, Winnipeg, Manitoba, Canada, R3E 0W3

Running Head: Strength after ACL reconstruction

Send correspondence to:

Dr. Dean Kriellaars e-mail: kriel@cc.umanitoba.ca
School of Medical Rehabilitation University of Manitoba
Winnipeg, Manitoba
Canada R3E 0W3

voice: (204) 787-3505
facsimile: (204) 787-1227

ABSTRACT

Twenty-four subjects who had undergone ACL reconstruction greater than one year previously were placed into one of two groups according to autograft donor site; patellar tendon (BPB; N=8), and hamstring (H; N=16) and compared to an active, control group (N=30). Knee flexor and extensor strength was evaluated using isovelocity dynamometry (5 speeds, eccentric and concentric, 5-95° ROM). Strength maps were used to graphically analyze strength over a broad operational domain of the neuromuscular system. Average strength maps were determined for each autograft group and compared to controls. A difference map (control minus graft group) and confidence (t-test) maps were used to quantitatively identify strength deficits. The combined ACL group (N=24) revealed a global 25.5% extensor strength deficit, with eccentric regional (angle & velocity matched) deficits up to 50% of control. Strength deficits covered over 86% of the sampled strength map area ($P<0.01$). These knee extensor strength deficits are greater than previously reported. In addition, the BPB group demonstrated a concentric, low velocity, knee extensor strength deficit at 60-95° that was not observed in the H group. Significant graft site dependent, regional knee flexor deficit of up to 50% of control were observed for the H group. Strength deficits localized to specific contraction types and ranges of motion were demonstrated between the ACL and control groups that were dependent upon autograft donor site. Post-operative rehabilitation protocols specific to these deficits should be devised.

Key Words: Dynamometry, Resultant Joint Moment, Neuromuscular Control

INTRODUCTION

The anterior cruciate ligament (ACL) of the knee functions as the primary restraint to anterior translation of the tibia relative to the femur. It is important for normal kinematics of the knee joint [119]. ACL injuries are common in active individuals and arise primarily from sporting and recreational activities. ACL rupture is associated with actions that have high relative acceleration between the femur and tibia, such as pivoting and cutting or with twisting falls with the tibia fixed. The current standard of care for active, symptomatic patients is the arthroscopically-assisted intra-articular autograft ACL reconstruction[110]. The tendons currently used for autograft are the middle third of the patellar tendon (BPB) and the hamstring tendons (semitendinosus (ST) or semitendinosus and gracilis (STG) [110].

Follow up studies on subjects who have had an ACL reconstruction show that the level of self-reported return to function is related to knee extensor strength [19, 295] and have demonstrated that functional outcome has a positive correlation with strength measurements [314, 378]. These studies indicate that knee extensor strength is a prerequisite to functional recovery after ACL reconstruction.

Considering this relationship between strength and function, a review of studies that report on single and multiple speed isovelocity dynamometry strength testing of the knee extensors after ACL reconstruction with the BPB procedure reveals a 5-34% strength deficit compared to the contralateral limb after rehabilitation [19, 151, 221, 234, 295, 321]. Studies of knee flexor strength after BPB reconstruction have not revealed knee flexor deficits when compared to the contralateral limb [151, 295, 321].

Knee flexor strength after hamstring (H) ACL reconstruction has been examined to a lesser extent and studies are limited to evaluation of only concentric contractions. Yasuda et al [390] at 60°/s, and Lipscomb et al [225] at 60°/s and 240°/s demonstrated no knee flexor deficit after H ACL reconstruction. In contrast, Marder et al [234] measured concentric peak moments at 60°/s, and showed that subjects having undergone an H ACL reconstruction had a knee flexor strength deficit of 17% when compared to the contralateral leg. These studies have revealed conflicting findings and there is a notable absence of data for eccentric muscle action of the hamstrings muscle group.

The etiology of knee extensor and knee flexor deficits after ACL reconstruction has not been fully elucidated. These deficits could be neural in origin, mechanical in origin, or secondary to de-conditioning. Altered motor unit recruitment and activation could be a result of modified input from a number of afferent pathways. Residual instability of the knee joint could result in altered feedback from mechanoreceptors located in the soft tissue about the knee joint [121, 204]. Nociceptive activity arising from the ruptured ligament or graft harvest site may alter neural control of the muscles that control the knee [186, 335]. Loss of information from receptors within the native ACL may contribute to changes in the ability to sense position (proprioception) and movement (kinesthesia) of the tibial and femoral body segments [21, 71, 290]. Altered mechanics of the musculo-tendinous unit as a result of graft harvest may effect changes in the force-length relationship of the knee flexors or extensors [161]. Finally, after an injury, an athlete will go through a variable period of decreased activity or detraining. A study of the knee extensors of humans showed that after 20 days of detraining there was a decrease in cross sectional area (equivalent to 0.1%/day), force production (0.3%/day), and EMG

(0.7%/day) [259]. The lack of formal rehabilitation in the post-injury but pre-surgical period to prevent de-conditioning, inconsistency in post-surgical rehabilitation protocols, and variable endpoints of re-conditioning may all be factors contributing to the knee extensor and possible knee flexor strength deficits after ACL reconstruction. In addition, the practice of using the contralateral leg as a control may be suspect given that, in an active individual, there will be a period of relative detraining of both the injured and un-injured legs. Thus, neural, mechanical, and rehabilitative factors may all be implicated in the development of strength deficits in the knee extensors and flexors after ACL reconstruction.

The purpose of this study was to examine the strength of the knee flexors and knee extensors after two surgical techniques of ACL reconstruction and compare them to an age and activity level matched control group. In contrast to previous studies that compared strength after both types of autograft utilizing only single velocity, concentric strength testing, we evaluate strength during both concentric and eccentric contractions, through a full functional range of motion and a large range of angular velocities. Further, we introduce a novel method of evaluation of standard dynamometry data for comprehensive assessment of strength in order to assess neuromuscular performance after injury, surgery, and rehabilitation.

We hypothesized the following; 1) that both patellar tendon and hamstring tendon reconstruction groups would reveal a substantial knee extensor strength deficit that was associated with ACL injury and not specific to the reconstruction or graft type when compared to a physically active control group, 2) that the hamstring reconstruction group would exhibit a graft site dependent knee flexor strength deficit, and 3) that the patellar

tendon reconstruction group would show a greater magnitude of knee extensor strength deficit than the hamstring group. Knowledge of graft site dependent, strength deficits would provide information that can be used to customize post-operative rehabilitation programs as well as aid in selection of surgical technique.

METHODOLOGY

ACL Reconstruction and Control Subjects

A physically active, control group (N=30) with no known knee pathology, and 24 persons who were greater than one year post ACL reconstruction served as subjects. All subjects provided written informed consent. Ethical approval for this study was granted by the Faculty Committee on the Use of Human Subjects in Research of the Faculty of Medicine, University of Manitoba. For the surgical subjects, exclusion criteria included other or subsequent injuries to the surgical limb, non-compliance to an accelerated rehabilitation program [228, 318], pain intensity greater than six on a ten centimeter visual analogue scale (VAS), use of performance enhancing drugs, and previous or present pregnancy. The surgical subjects, ACL, (N=24) were divided into two groups based upon autograft donor site, BPB (N=8), and H (N=16). Using conservative estimates of the expected mean differences in strength and variance, and a power index of 2.92 (0.05 alpha; 0.1 beta), the minimum number of subjects in each group was determined to be at least eight. The subjects with semitendinosus (N=7) and semitendinosus-gracilis (N=9) grafts were combined into a single group as no significant differences in strength were observed. Surgical subjects were compared to an active control group in order to avoid

limitations associated with using the contralateral limb as a control, that being under-estimation of strength deficits due to contralateral leg de-conditioning.

Surgical Technique & Rehabilitation

Arthroscopically-assisted intra-articular autograft ACL reconstruction was performed on all of the surgical subjects by one of two fellowship-trained orthopaedic surgeons. The BPB procedure was a free patellar tendon autograft with bone blocks on both ends that were fixed with interference screws. The H ACL reconstruction procedure used a double looped semitendinosus or semitendinosus and gracilis autograft, through femoral condyle intra-articular technique. Kennedy ligament augmentation device (LAD) was used in nine of the hamstring reconstruction procedures, 15 were non-augmented. Post-operative rehabilitation was based on an accelerated rehabilitation protocol [228, 318]. Patients were fitted with a hinged knee brace in the operating room. Immediate range of motion exercises and weight bearing as tolerated was allowed with the brace on.

Evaluation

The subjects were asked to fill in a questionnaire that included the subjective component of the International Knee Documentation Committee (IKDC) form (International Knee Society Meeting, Toronto, Canada, 1991). A 10 cm VAS was used to assess pain intensity. Active range of motion of both knees was evaluated using a universal goniometer. Clinical instability testing was performed on both of the subjects' knees by one examiner using the Lachman and pivot shift tests. The KT1000 arthrometer (Medmetric Corp. San Diego, CA) was used to assess anterior-posterior displacement of

the tibia relative the femur. Three trials were performed on each knee with the difference between sides used for data analysis.

Isovelocity Dynamometry

The knee extensors and flexors of the subjects' operative leg were tested on a Kin-Com 500H dynamometer (Chattecx Corporation, Hixson, TN) after warming up on a Monark 818E cycle ergometer (Monark AB, Sweden) at 35 W for five minutes. Subjects were seated on the dynamometer with the backrest reclined 15° from vertical. Straps were secured around the waist and distal thigh for stabilization. The axis of rotation of the dynamometer was visually aligned with the lateral femoral condyle when the knee was flexed 90° (full knee extension = 0°). The pad on the dynamometer actuator arm was then securely fastened about the subject's leg at a position approximately five centimeters proximal to the medial malleolus. The range of motion of the knee was set from 5° to 95° of knee flexion. Subjects were provided with consistent verbal prompting during the strength tests.

The subjects were tested using the isovelocity dynamometer mode (constant angular velocity) at ten angular velocities (±50, 100, 150, 200, 250°/s) with negative velocities corresponding to eccentric contractions of the prime movers. Three sub-maximal contractions were performed for familiarization prior to maximal effort contractions at each speed. Three maximal effort repetitions were recorded at each speed with a five-second pause between the concentric and eccentric contractions. Test-retest reliability of this protocol was very good (ICC = 0.89). The dynamometer moment (M_D in Nm) was derived as the product of the force (N) and the moment arm (m). The

moment arm corresponded to distance from the point of force application on the force transducer to the dynamometer's axis of rotation. The knee joint angle, angular velocity and M_D data were collected at 100 Hz sampling rate. The data was exported to custom software for analysis. The knee joint moment (M_K) was determined by correction of M_D for the moment of the weight of the leg and foot (plus resistance pad). For each repetition, the peak moment (Nm), the angle of peak moment (°) and the work (J) were derived. The M_K were normalized to body mass (Nm/kg) based upon a strong correlation ($r = 0.89$, $p < 0.01$) between M_K and body mass. We used body mass normalization of the lower limb strength data to permit valid comparison between groups with different male:female compositions, since this approach has been validated for multiple velocity and isometric moment data arising from young, active individuals [59, 165, 382].

Strength maps

A color-coded, relief map, termed a strength map (Figure 2), consisting of a 50 x 50 matrix of knee extensor moment (M_K) values was created for each subject using a bicubic interpolating spline from the sampled dynamometry data using custom dynamometry software (Isomap, Isodyne Inc., Winnipeg, Canada). In the spline process, the sampled moment/angle data is fit with a cubic polynomial equation and equally spaced moment values are generated over the tested range of motion for all velocities tested. Then, the spline is applied across velocities to perform an interpolation of the moment values over a 50 °/s span for concentric and eccentric contractions. This interpolation is permitted based upon the very good to high reliability that has been reported for peak moment and angle specific moment measurements [241, 279]. A 50 x 50

matrix provides a map resolution of 1.6 ° for knee joint angle and 10 °/s for angular velocity. The strength maps depict the strength or moment generating ability about the knee over a broad operational domain (±250 °/s, 5-95°, eccentric and concentric contractions). Strength maps were derived for both the knee extensor and knee flexor strength tests for each subject, using the average of the three repetitions at each angular speed. For each strength map, the overall body mass normalized peak moment (Nm/kg) and the body mass normalized total work (J/kg) were also determined. After visual inspection of individual strength maps, average strength maps were generated for each group (control, BPB, H and ACL). Difference maps were generated between groups to localize strength differences between groups. Further, the difference maps permit quantification (total area of the map and regional localization) of a strength deficit (or gain) by application of a clinically suitable moment threshold (i.e. 0.15 Nm/kg or expressed as a percentage of control, 5-10%). Confidence maps were generated for statistical comparison of strength changes between groups by performing an independent t-test between groups at each of the 50 x 50 map points. The resulting 2500 t-values were then portrayed in relief map format on the same axes. The critical t-value was used as a threshold to identify regions of statistical difference. These statistically different regions are automatically localized by software to the specific range of motion, contraction type and speeds. The likelihood of observing Type I errors increases with the number of inferential tests performed. We performed an iterative computer simulation to determine the occurrence of randomly distributed Type I errors within the map area and computed the probability of contiguous Type I errors in a 3x3 matrix area of the map to be less than

0.006. We used this minimal area as the criteria for acceptance of differences based upon t-test results to effectively guard against accepting Type I errors.

RESULTS

Subjects

All subjects were physically active and participated in exercise or sport on a regular basis. The BPB group (N=8) had an average body mass of 86.8±17.9 kg., average age of 29.3±7.9 years, and consisted of seven men and one woman. The H group (N=16) had an average body mass of 77.8±16.6 kg., average age of 26.2±7.6 years, and consisted of nine men and seven women. The control group (N=30, males) had an average mass of 78.8±13.1 kg, an average age of 26.3±4.8 years. There were no statistical differences in body mass or age between the groups. The average time since surgery was 30.0 ±8.5 months for the BPB group and 30.4 ±10.5 months for the H group.

In the BPB group there were two medial meniscal tears, one lateral meniscal tear and two medial and lateral meniscal tears. Two subjects had associated medial collateral ligament injury. Five of the subjects injured their dominant leg (the leg they kick a ball with) and three injured their non-dominant leg. In the H group, there were five medial meniscal tears, three lateral meniscal tears and one medial and lateral meniscal tear. Five subjects had associated medial collateral ligament injuries. Nine of the subjects injured their dominant leg while seven injured their non-dominant leg.

There were no differences between the surgical groups for knee joint range of motion. The KT1000 arthrometer measurements revealed non-significant side to side

differences between the BPB group (3.7 ±2.5mm) and the H group (2.5 ±2.3mm). The VAS pain intensity magnitudes were generally low (maximum BPB group = 3.5 cm, H group = 2.6 cm) and were not significantly different between the surgical groups (BPB: 1.35 ± 0.68 cm; H: 1.44 ± 0.15 cm).

Subjective assessment using the IKDC revealed no differences in pain, stiffness, or instability between the BPB and the H groups. The H reconstruction group showed a significantly higher self-reported level of function (BPB; 1.13 ±3.3, H; 0.56 ±0.5, $P<0.05$, Kruskal-Wallis one-way ANOVA).

Isovelocity Strength Testing

Peak Moment/Angular Velocity Plots. Peak moment/angular velocity graphs (Figure 1) were produced for the knee flexors and knee extensors. Peak moments were calculated using the average of three repetitions at each angular velocity. For the knee extensors (Figure 1a), the entire ACL group (N=24) was compared to the control group, as both the BPB and H group demonstrated similar knee extensor strength deficits using multiple velocity, peak moment measurements. Using peak moment data, the ACL group demonstrated a substantial knee extensor strength deficit ($P<0.05$) which was observable during both concentric and eccentric contractions. When compared to a control group, the knee extensor strength deficit was 25% averaged across all speeds and contraction types.

For the knee flexors, the peak moment/angular velocity graph (Figure 1b) was produced which compares the H ACL reconstruction group (N=16) to the control group. Using peak moment data, the H ACL reconstruction group demonstrated a strength deficit of the knee flexors during both concentric and eccentric contractions. When

compared to the control group, this knee flexor strength deficit ($P<0.05$) was 17% averaged across all velocities and contraction types.

Knee Extensor Strength Maps. Averaged strength maps were generated for the knee extensors of the control (Figure 2a) and ACL reconstruction (Figure 2b) groups. The peak moments generated by the knee extensors occur in the eccentric region, at lower angular velocities and at the joint angles around 70° (Figure 2a). Visual comparison of the ACL group (N=24) to the control group (N=30) knee extensor strength maps revealed that the basic topography of the strength maps was similar. Comparison of the body mass normalized, overall peak moment and the total work (volume under map surface) revealed an average 25.5% strength deficit (peak moment; 3.15 Nm/kg (control) vs. 2.32 Nm/kg, total work; 27.2 (control) vs. 20.5 J/kg). The difference map (Control group subtract ACL group, Figure 2c) permits quantification of the magnitude of difference between the two groups, as well as the identification of regional strength deficits. Examination of the difference map reveals that the peak strength deficits occur in the same region in which the peak moments are generated by the knee extensors. Using angle and velocity matched comparisons from the difference map regions in which the extensor deficit was up to 50% of control was identified (localized to low velocity eccentric and concentric contractions). Confidence map are used to identify regions in which the strength deficit was statistically significantly different ($P<0.01$). The statistically significant region of strength deficit covered 86% of the strength map area (Figure 2d). The region of higher t-values in the confidence map is associated with relatively smaller mean differences in moments between groups and relatively lower variance.

Additional knee extensor strength deficits were evident in the difference map between the BPB group and the H group. The BPB group showed a significant, low velocity concentric deficit at 65-90° of flexion ($P<0.05$) that was not evident in the hamstring reconstruction group.

Knee Flexor Strength Maps. Averaged strength maps were generated for the knee flexors for the control (Figure 3a) and the H ACL reconstruction (Figure 3b) groups. Visual comparison of the knee flexor strength maps revealed that the basic topography of the maps was similar. The peak moments generated by the knee flexors occur in the eccentric region, at higher angular speeds and at lower joint angles (around 15°). Comparison of the body mass normalized, overall peak moments and the total work revealed a substantial strength deficit (peak moments; 1.67 vs. 1.35 Nm/kg, total work; 16.4 vs. 12.5 J/kg). Using peak moment values derived from the strength maps, a strength deficit of 19% was observed. Using total work, a strength deficit of 24% was determined. The difference map (Control group subtract H ACL group, Figure 3c) revealed a significant ($P<0.01$) graft site dependent knee flexor weakness. The greatest magnitude of knee flexor strength deficit was localized to the eccentric portion of the map, with a substantial band of weakness over a 60-95° range of knee flexion. Using angle and velocity matched comparisons, regions were identified in which the flexor deficit was up to 50% of control (high speed eccentric contractions at joint angles greater than 60°). The confidence map (Figure 3d) portrays the statistically significant ($P<0.05$) area over which the knee flexor strength deficit exists representing over 95% of the entire map. It is important to note that unlike the knee extensor strength deficit, the greatest magnitude of knee flexor strength deficit occurred at knee flexion angles over 60 °, substantially away

from the angle of peak moments. A knee flexor strength deficit was not identified in the BPB reconstruction group.

DISCUSSION

Using strength maps, we have provided a comprehensive portrayal of the strength of the knee flexors and knee extensors in subjects after two methods of ACL reconstruction. An important novel finding is that the H autograft reconstruction group demonstrated a graft-site dependent, regional knee flexor strength deficit that was not observed in the BPB group which has not been detected using conventional dynamometry (single test velocity, concentric, peak moments). All subjects who had undergone ACL reconstruction, independent of autograft donor site, demonstrated a substantial global knee extensor strength deficit compared to an active control group. The peak magnitude of this deficit is greater than previously reported. In addition, subjects who had undergone a BPB autograft had an additional low velocity, knee extensor strength deficit that was not present in the H group.

The existence of a knee extensor strength deficit in subjects who have had an ACL reconstruction has been previously characterized using peak moments [19, 221, 295, 320, 321]. These studies found strength deficits of the knee extensors ranging from 5–34%. Evaluation of the knee extensors using strength maps clearly illustrates that both the BPB and the H tendon ACL reconstruction subjects show an average global 25.5% knee extensor strength deficit when compared to a physically active control group. This deficit was up to 50% of control in specific regions of the operational domain of the knee extensors. This ACL injury-dependent knee extensor deficit was consistent between

surgical groups and was largely independent of graft type. The largest difference in knee extensor moment between the control and ACL reconstruction groups occurs in the same region as where the peak moments are generated, that being low velocity, eccentric contractions at joint angles ranging from 50-80°. The use of multiple velocity, peak moment data would identify a knee extensor strength deficit of this nature, as has been reported in the literature. The larger knee extensor deficits observed in this study compared to previous studies can be explained in several ways. First, the use of strength maps allowed us to examine areas within the operational domain of the knee extensors apart from where the peak moments are generated. Angle and velocity matched comparison of groups resulted in identification of regions of strength deficits which were larger than those reported using conventional dynamometry assessment. Second, in all previous studies examining strength after ACL reconstruction, the contralateral leg has served as the control for comparison to the injured leg. Our study reveals that bilateral strength comparison results in under-estimation of the magnitude of the strength deficit in this patient population. Unpublished strength data from our lab on the non-operated, or contralateral, lower limb also shows a strength deficit similar to the ACL reconstructed leg, when compared to an active control group.

In contrast to previous studies, with the exception of Marder [234], we have identified a substantial knee flexor strength deficit in the H reconstruction group. The knee flexor deficit has the greatest magnitude in the high speed, eccentric region of the operational domain of the knee flexors. This knee flexor strength deficit is related to hamstring autograft and is not seen in the BPB surgical group. The existence of previously unrecognized regional knee flexor strength deficits could be explained in

several ways. First, the assessment of knee flexor strength using angle and velocity-matched data allow the identification of deficits that occur away from the area where peak moments are generated by the knee flexors. Previous studies that used peak moment data would have been unable to identify these deficits. Second, comparison of knee flexor strength after ACL reconstruction to the contralateral limb may underestimate the strength deficit. Using conventional angular velocity – peak moment graphs and comparing to control, we were able to identify a knee flexor deficit. However, this deficit was still underestimated when compared to angle and velocity matched moment data.

The functional consequence of this eccentrically localized knee flexor deficit is unknown, however, the knee flexors would be used eccentrically for knee joint stabilization (co-contraction during concentric knee extensor contractions) [290, 335] as well as for leg deceleration. The compromised ability of the knee flexors to dynamically stabilize the knee during functional activities may lead to increased risk of ACL graft rupture or other injury as well as interfere with return to previous levels of functioning. Based upon the bi-articular nature of hamstring muscle group, there are implications for the ability of the hamstring muscle group to generate hip extensor moments based upon the knee flexor strength deficit. Further study is needed to investigate this possibility.

The BPB group displayed a regional deficit during low velocity, concentric contractions from 65-90° range of motion. This is consistent with the hypothesis of graft site dependent, knee strength deficits. The importance of this small regional deficit is probably overshadowed by the large global deficit seen in the knee extensors after ACL reconstruction but may have implications for specific activities where concentric knee extensor strength is required (such as cycling).

One criticism of this paper may be the use of both men and women in the ACL reconstruction groups. It is well described in the literature that there are significant sex differences in absolute strength of the knee flexors and extensors [240]. These differences, however, are based on absolute values and have not been corrected for body mass or muscle mass. Studies have shown that older women are able to generate less force per unit mass about the knee than men [77, 292], while others demonstrate that correction for body mass eliminated the sex difference in strength [118, 292]. In contrast to studies on older subjects, studies confirm that women are able to generate similar knee moments for the knee flexors and extensors as men when corrected for body mass and when activity matched [59, 165, 382]. Our data is consistent with these findings for young, active individuals. When the average knee flexor and extensor strength maps of the males and females were constructed and compared, the vast majority of the strength differences in this young, active group are corrected by body mass normalization. Knee extensor peak moments for the women were 93.7% that of the men. Knee flexor peak moments for the women were 91% that of the men. These small, sex specific strength differences were global in nature and did not affect the overall topography of the strength map. This might contribute to a small over-estimation of magnitude of strength deficits observed in the H ACL reconstruction group. However, we performed a comparison of the strength maps and difference maps based upon male data only and the results were not altered.

The implications of a global knee extensor strength deficit, and graft site dependent regional strength deficits in both the knee flexors and extensors are important to consider. The tendon autograft chosen to perform ACL reconstruction may be individualized based on the patient's previous symptoms, such as patellofemoral pain. In

addition, patients who participate in sports that require prominent use of the biarticular hamstring muscle group may want to avoid the possibility of a knee flexor deficit. These findings have implications for post-operative rehabilitation of these patients. Physiotherapy programs can be tailored to reduce or eliminate these regional strength deficits found after ACL reconstruction by the incorporation of exercises that are velocity, range of motion and contraction type specific to the strength deficits observed.

These data provide insight into the mechanisms underlying the neuromuscular control of the knee after injury and surgery. The existence of regional strength deficits may arise from alteration in neural activation patterns, mechanical alterations about the joint and changes in muscle properties. Neural control of the motoneurons innervating the lower limb musculature include proprioceptive and nociceptive input from the native ACL, the soft tissue spanning the joint, the muscles involved in movement about the joint, as well as propriospinal, and supraspinal pathways including volitional control. The ACL reconstructed knee has endured a host of stimuli including the original trauma, a period of de-conditioning, the surgical intervention, rehabilitation, and return to activity. If the strength deficit seen in the ACL reconstructed limb was purely a consequence of a change in muscle properties, one would predict specific, uniform effects. For instance, a change in muscle property, such as in the length/tension relationship could cause a shift in moment generating ability with joint angle, however, the deficit would be consistent for all speeds, and both concentric and eccentric contractions. A change in the force/velocity characteristics of the muscles would result in shifts in moment generating ability at specific speeds but not at specific joint angles. The most parsimonious hypothesis is that these non-uniform, regional strength deficits arise from an alteration in

neural activation of the muscles involved resulting from altered proprioceptive/nociceptive afferent feedback from the knee region, pain or volitional control. The prominent localization of these regional deficits to the eccentric regions of the strength maps is consistent with the findings of studies revealing substantial neural regulation of eccentric contractions [95, 368]. The underlying physiological mechanism of these regional strength deficits has yet to be elucidated, however, altered activation of joint proprioceptors, nociceptors, and mechanoreceptors may play an important role in altering motoneurone activation patterns responsible for the identified strength deficits.

This study has provided detailed information as to the nature of strength deficits that occur after ACL reconstruction. A global knee extensor strength deficit was discovered in all subjects after ACL reconstruction (independent of graft site) that is larger than has previously been reported. Regional strength deficits of the knee flexors and extensors following patellar tendon and hamstring tendon ACL reconstruction were dependent on autograft donor site. The consequences that these strength deficits will have in terms of post-operative functional ability and predisposition to further injury have yet to be determined. Objective recommendations as to autograft donor site selection, therefore, would be premature. However, post-operative rehabilitation protocols should be modified in order to reduce or eliminate these deficits by including velocity, range of motion and contraction type specific exercises.

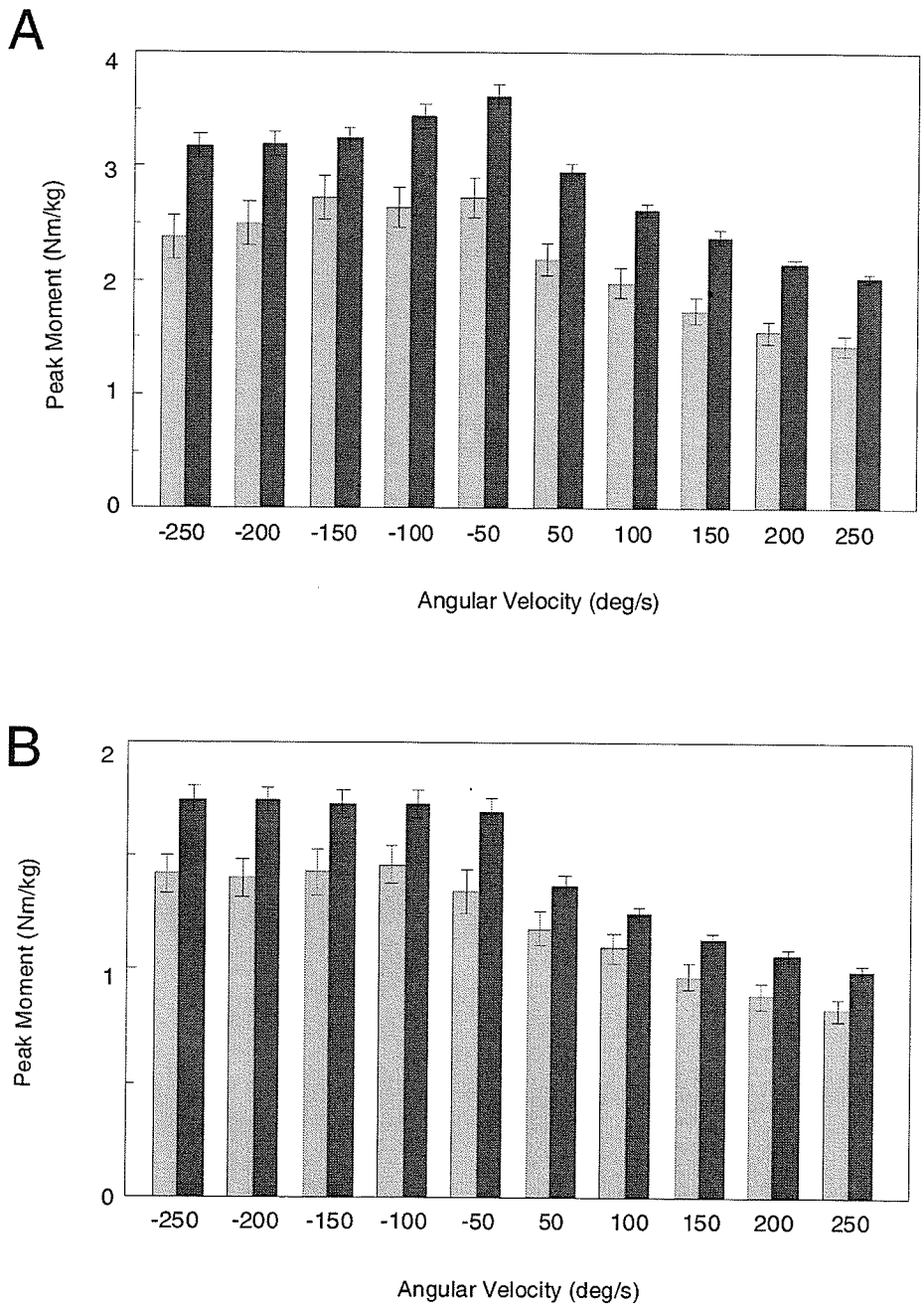

Figure 1. Knee joint moment-angular velocity relationship for the knee extensors (A) and flexors (B) for the control (dark bars) and ACL (lighter bars) groups. The mean (± SE), body mass normalized peak knee moments are plotted for each angular velocity. Positive velocities correspond to concentric contraction of the knee extensors (negative velocities – eccentric).

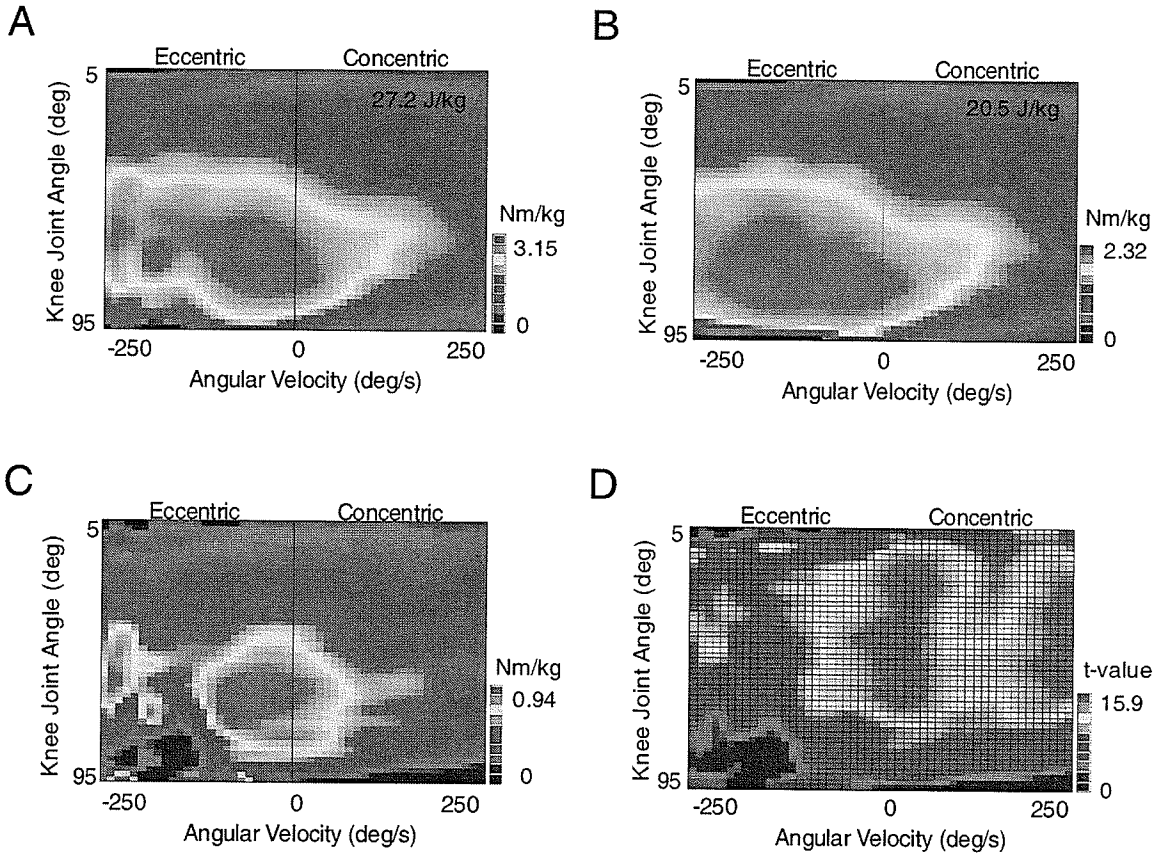

Figure 2. Average strength maps of the knee extensors for control (A) and ACL reconstructed (B) groups. The strength maps show the moment generating ability of the knee extensors for concentric (positive angular velocity) and eccentric contractions over a range of angular velocities (±250°/s) through a 5-95° range of motion. The magnitude of the body mass normalized moments (Nm/kg) generated by the knee extensors are color coded based upon the scale bar. Although the shape of the strength maps (A & B) are similar, substantially lower overall peak moments (control: 3.15 Nm/kg; ACL: 2.32 Nm/kg) and work (control: 27.2 J/kg; ACL 20.5 J/kg) were observed for the ACL group. Regional strength deficits are revealed by the difference map (C) that shows the algebraic differences between control and ACL knee extensor strength maps The results of angle and velocity matched independent t-tests (t values) are shown using the confidence map (D). Statistically significant differences ($P < 0.01$) in knee extensor strength are shown by the hatched area and covers 86 % of the entire map. The largest difference in moments generated by the knee extensors occurs in the same region as the peak moments.

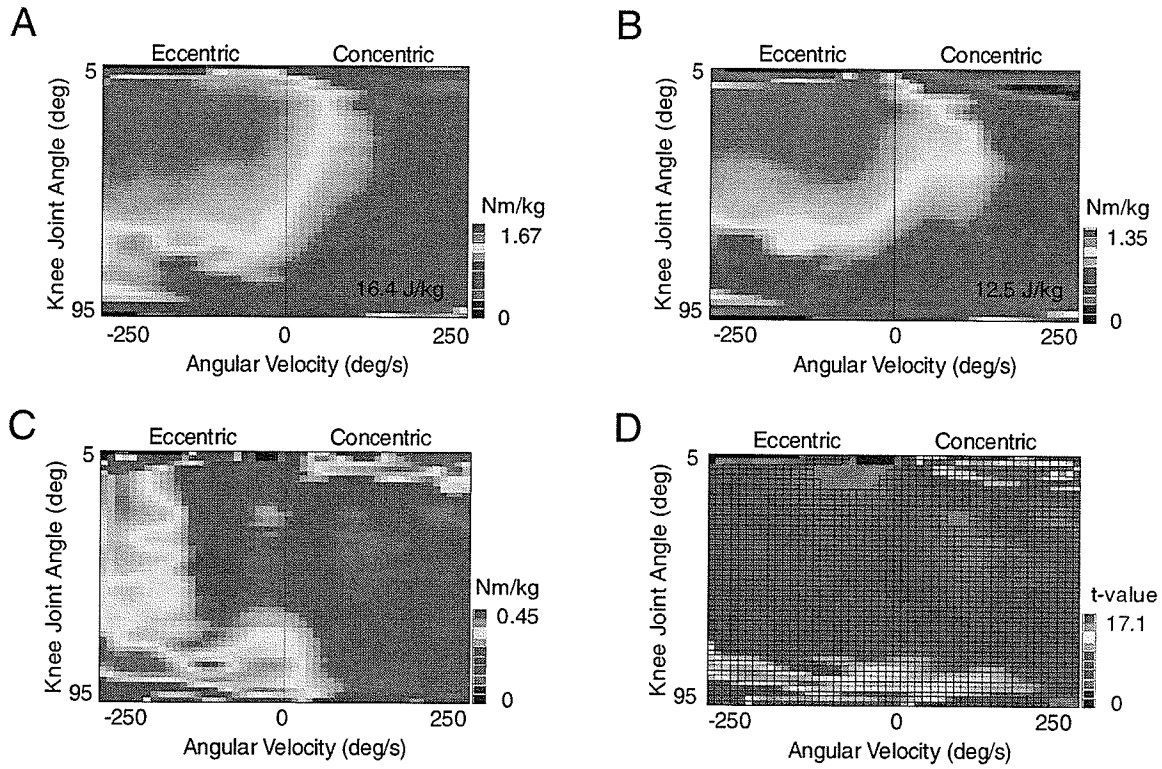

Figure 3. Average strength maps of the knee flexors for control (A) and hamstring ACL reconstructed (B) groups. The strength maps show knee joint moments for concentric (positive angular velocity) and eccentric contractions of knee flexors over a range of angular velocities (±250°/s) through a 5-95° range of motion. The magnitude of the body mass normalized moments (Nm/kg) generated by the knee flexors are color coded using the associated scale bar. Although the shape of the strength maps are similar, substantially lower overall peak moments (control: 1.67 Nm/kg; ACL:1.35 Nm/kg) and work (control: 16.4 J/kg; ACL 12.5 J/kg) were observed for the ACL group. Regional strength deficits are revealed by the difference map (C) that shows the algebraic differences between control and ACL knee flexor strength maps. The results of independent t-tests (t values) at each velocity and angle are shown using the confidence map (D). Statistically significant differences ($P < 0.01$) in knee flexor strength are shown by the hatched area and covers 95% of the entire knee flexor strength map. The largest difference in moments generated by the knee flexors is observed in a region distinct from the range of motion where the peak knee flexor moments occur.

Study 2 – Hamstring and Quadriceps Strength Balance in Normal and Hamstring ACL Reconstructed Subjects

This study was designed to provide an overall portrayal of the neuromuscular balance about the knee to permit further development of our conceptual understanding of 'muscle balance'. The goals of this study were to evaluate and characterize strength balance about the knee in the healthy, active subject using a joint angle, angular velocity, and contraction type specific portrayal of standard dynamometry strength data. In addition, a more physiologic ratio (DCR) that incorporates the concept of contraction-type specific moments as would be generated during functional muscular co-contraction is presented. The same concepts were applied to an ACL reconstructed subject group, in order to determine whether strength balance about the knee was altered after surgery and subsequent rehabilitation. Given that these maximum voluntary effort contractions will form the upper limit of knee flexor and extensor moment development during dynamic movement, alteration may have implications for movement control and joint stabilization.

This Manuscript will be submitted to the *Journal of Clinical Biomechanics* in February 2003. Ethics approval for this study has been granted by the University of Manitoba (E96:246). This paper has won the **Runner-up Best Research Paper** at the American College of Surgeons, Manitoba Chapter Annual Meeting, Winnipeg, Manitoba 1998; **Runner-up Best Resident Research Paper,** Manitoba-Saskatchewan Resident Research Day, Saskatoon, SK, 1998.

A review of literature includes a review of neuromuscular balance including the hamstring/quadriceps ratio and the dynamic control ratio.

Neuromuscular Balance

A formal definition of neuromuscular balance has never been delineated; however the assessment of strength balance about a joint has typically involved comparing the strength of the agonist, or prime mover, to the antagonist, or stabilizing, muscles that span it. Operationally, we employ the term **neuromuscular balance** to reflect the fact that strength arises from both the neural and muscular systems. The hamstrings-quadriceps ratio (H/Q ratio) has been used both clinically and in the laboratory as an indicator of neuromuscular balance about the knee. Strength deficits, both global and regional, of the knee flexors and extensors will affect the "muscle balance" about the knee.

Traditionally, the H/Q ratio is calculated using peak moment values of the knee flexors and the knee extensors generated during an isometric contraction or during a concentric contraction at a specific angular velocity. There have been many studies done to define the H-Q ratio in healthy people. Studies state that there is marked inter-subject variability and the testing protocols vary, but the H/Q ratio in healthy uninjured people using current evaluation techniques with gravity correction is about 0.5–0.6 [1-3, 29, 55, 74, 298, 347, 351, 373, 375]. In ACL deficient subjects, the H/Q ratio is found to be increased [192, 223]. We were unable to find any studies assessing the H/Q ratio after ACL reconstruction.

The alteration of normal neuromuscular balance about the knee may affect the ability of the subject to achieve optimum functioning as well as protect against further injury. Objective evidence of the functional significance of the H/Q ratio is difficult to find, although alteration of the normal neuromuscular balance about the knee is felt to contribute to injury [268, 284, 333, 340]. Others argue that these strength deficits may help

protect the knee against further injury [30, 104]. For example, the knee flexors act as an ACL agonist [291, 335], and therefore alteration of the neuromuscular balance about the knee in favour of the knee flexors after ACL reconstruction may be an adaptation to help protect the injured knee. The role of the knee flexors in the stabilization of the knee joint during movement has been well documented [20, 84, 178, 291, 335].

The inability of the traditional H/Q ratio to provide objective, reliable information that can predict injury or guide rehabilitation may be a function of the measurement itself. The conventional way of studying muscle balance using concentric peak moment values to generate a H/Q ratio fails to take into consideration the moment-angle and moment-angular velocity relationship of the knee flexors and extensors. Both the knee joint angle and angular velocity are necessary elements to consider when attempting to comprehensively evaluate neuromuscular balance about the knee [263].

Peak moments generated by the knee extensors and flexors occur at very different knee joint angles [164]. The peak moments generated by the knee extensors occur at 60-70° of knee flexion, while the peak moments generated by the knee flexors occur at 10-20° of knee flexion. This occurs because the knee flexors and extensors are at different positions on their respective length-tension curve. During dynamic contractions, the use of peak moment data generated during knee flexion and extension to calculate the traditional H/Q ratio value is not physiologic, as the peak moments of each muscle group occur at widely disparate angles. Some studies have used angle specific H/Q ratios in an attempt to address this problem [3, 55, 256, 298, 373]. These studies have confirmed that H/Q ratio values are highly dependent on the knee joint angle at which the measurement is taken.

During isovelocity contractions, it has been well demonstrated that the peak moments that are generated by the knee flexors and extensors during concentric contractions decrease with increasing angular velocity [3, 98, 164, 368, 377]. In contrast, moments generated during eccentric contractions are relatively independent of angular velocity [164, 368, 377]. Studies using gravity corrected moments have shown an increase [55, 256, 340], a decrease [284] or no change [3, 298, 375] in H/Q ratio with increasing angular velocity. Finally, during isovelocity strength testing higher moments are generated during eccentric contractions than during concentric contractions [164, 368, 377].

Dynamic Control Ratio

Calculation of traditional H/Q ratios from isovelocity strength testing, involves using the concentric moments from both knee flexors and extensors. Westing and Seger [375] demonstrated that the gravity corrected H/Q ratio was higher for an eccentric H/Q than for a concentric H/Q. During dynamic movement, however, the agonist, or prime mover, will be contracting concentrically while the antagonist, or stabilizer, will be contracting eccentrically. For example, during knee extension, the knee extensors are contracting concentrically while the knee flexors are contracting eccentrically. If the moments generated by the knee flexors and extensors are dependent on contraction mode then calculating an H/Q ratio using an eccentric knee flexor moment divided by a concentric knee extensor moment would be more physiologic. This more "functional" way of characterizing moment generation about the knee led to the concept of a dynamic control ratio that was initially introduced by Dvir [89] and later applied by others [2, 3, 29].

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Hamstring and Quadriceps Strength Balance in Normal and Hamstring ACL Reconstructed Subjects

Laurie A. Hiemstra, MD, FRCSC, Sandra Webber, BMR(PT),MSc,
Peter B. MacDonald, MD, FRCSC and Dean J. Kriellaars, PhD

Sport Medicine Centre
University of Calgary, Calgary, Alberta T2N 1N4

School of Medical Rehabilitation, Faculty of Medicine
University of Manitoba, Winnipeg, Manitoba, R3E 0W3

Investigation performed at the Human Performance Laboratory, Winnipeg.

Running Title: Neuromuscular Balance of the Knee Joint

Corresponding Author:

Laurie A. Hiemstra, MD, FRCS(C)
Sport Medicine Centre
University of Calgary
2500 University Dr. NW
Calgary, AB
T2N 1N4

Phone 403-220-5077
fax 403-282-6170
email hiemstra@ucalgary.ca

ABSTRACT

Introduction The traditional hamstring/quadriceps (H/Q) ratio using concentric peak moments does not account for the different muscle characteristics (force/moment-length/velocity), the moment arm variations with joint angle, moment-angular velocity and the contraction-specific relationships of moments generated by the knee flexors and extensors. This study was designed to comprehensively evaluate 'muscle balance' about the knee joint.

Methods Dynamometer knee joint strength testing was performed on 16 subjects after hamstring anterior cruciate ligament (ACL) reconstruction and compared to 30 active, healthy control subjects. Knee flexor and extensor strength testing was performed over 5-95° knee joint range of motion, at 10 angular velocities (±50, 100, 150, 200, 250 °/s, concentric corresponding to positive velocities). Strength maps were generated for each muscle group, and then averaged among subjects. Angle and velocity matched H/Q ratio maps and Dynamic Control Ratio (DCR) maps were produced for each group to depict muscle balance for the knee joint. Difference maps between ratio maps (H/Q and DCR) allowed quantification of the differences in neuromuscular balance between the groups.

Results Angle and velocity matched H/Q ratio maps demonstrate systematic variation based on joint angle, velocity, and contraction type for both the control (H/Q; near 0-1.42; DCR; near 0 to 1.57) and the ACL reconstructed (H/Q; near 0-1.33; DCR; near 0-1.35) groups. Difference maps between ratio maps for each group demonstrate regional (angle and velocity specific) alteration in the ratios between the ACL reconstructed and control groups

Conclusions This study provides a comprehensive analysis of strength balance about the knee joint. Unlike the single value H/Q ratio, strength balance about the knee as represented by the angle and velocity matched H/Q ratio varies in a complex manner. Specific imbalances were demonstrated in the ACL reconstructed group compared to control. In angles of knee flexion, the low H/Q ratio may represent a compromised ability of the hamstrings to stabilize the knee joint throughout the full range of motion. Near full knee extension shifts in favour of the knee flexors may represent an attempt to stabilize the knee at the angle of greatest ACL strain. These finding have implications for graft donor site selection, post-operative rehabilitation as well as provide insight into the neuromuscular control of the knee.

Key Words: dynamometer, specificity, moment, torque, H/Q ratio, dynamic control ratio

INTRODUCTION

The assessment of strength and strength balance is an important aspect of training and rehabilitation, however little is known about the appropriate or optimal balance of strength between muscles especially as it relates to restoration of strength after injury, or training for specific tasks especially after injury or surgery. Strength is a measure of the ability of a muscle group to generate moment about a joint and is dependent on both the neural activation of the muscle as well as the intrinsic properties of the muscles themselves and musculoskeletal mechanical characteristics of the muscle. Thus, a strength test, which involves the determination of the resultant joint moment/angle relationship, is necessarily a test of the entire neuromuscular system. The H/Q ratio has been used both clinically and in the laboratory as an indicator of the balance of strength among muscles spanning the knee. Although there have been no long-term studies done to validate the prognostic or predictive value of the H/Q ratio for functional recovery, it continues to be used as a marker in fitness and rehabilitation fields. Certainly, the alteration of normal strength balance about the knee by injury, detraining or surgery may affect the ability of patients to achieve optimum recovery of function and alter their ability to protect against further injury. Studies on subjects who have had an ACL reconstruction have demonstrated that the level of self-reported return to function is dependent upon knee extensor strength and have demonstrated that functional outcome has a positive correlation with strength measurements [314, 378]. Thus, the assessment and restoration of strength and strength balance about the knee remain important objective outcomes after ACL reconstruction and subsequent rehabilitation. In addition, the

assessment of strength balance and changes that occur in strength balance with injury and/or surgery allow us to explore an aspect of the neuromuscular control of that limb.

Conventionally, the H/Q ratio is calculated using peak moment values of the knee flexors and the knee extensors generated during an isometric contraction or during concentric contractions at a low specific angular speed (typically 60°/s). There have been numerous studies performed to define the H/Q ratio in healthy, uninjured subjects. These studies state that there is marked inter-subject variability and the testing protocols vary, but the H/Q ratio in healthy uninjured people using current evaluation techniques with gravity correction for the moment of the weight of the leg and foot ranges between 0.5–0.6 [2, 3, 29, 373, 376]. Without consideration for the angle of peak moment, this would reflect that the knee flexors are approximately half as strong as the knee extensors. In patellar tendon ACL reconstructed subjects, the H/Q ratio has been demonstrated to be increased compared to control during isometric contractions at 60° knee joint angle [393].This increase in H/Q ration is likely due to a knee extensor strength deficit known to exist after patellar tendon ACL reconstruction. This study will determine whether this finding is maintained in subjects who have had a hamstring ACL reconstruction.

Objective evidence of the functional significance of the H/Q ratio in it's current form is lacking, although alteration of the normal strength balance about the knee has been demonstrated in ACL deficient subjects [223] and altered H/Q ratio is felt to contribute to injury [284, 333]. The inability of the conventional H/Q ratio to provide objective, reliable information that can predict injury or guide rehabilitation may be a function of the measurement itself.

The conventional way of studying neuromuscular balance using concentric peak moment values to generate a traditional H/Q ratio fails to take into consideration known muscle properties such as the force-length and force-velocity relationships. The angular correlates of these muscle properties, the moment-angle and moment-angular velocity relationships of the knee flexors and extensors are necessary elements to consider when attempting to comprehensively evaluate strength balance about the knee.

Consistent with the moment-angle relationship, peak moments generated by the knee extensors and flexors occur at very different knee joint angles [164]. The peak moments generated by the knee extensors occur at 60-70° of knee flexion, while the peak moments generated by the knee flexors occur at 10-20° of knee flexion. These data are compatible with findings that during isometric contractions at varying angles, the H/Q ratio increases as the knee flexors become dominant with increasing knee extension [373]. During dynamic contractions, the use of peak moment data generated during knee flexion and extension to calculate the traditional H/Q ratio value is not physiologic, as the peak moments of each muscle group occur at widely disparate knee joint angles. Some studies have used angle specific H/Q ratios in an attempt to address this problem [3, 256, 373]. These studies have confirmed that H/Q ratio values are highly dependent on the knee joint angle at which the measurement is taken[164].

During maximal voluntary isovelocity contractions on a dynamometer, it has been well demonstrated that the peak moments generated by the knee flexors and extensors during concentric contractions decrease with increasing angular velocity [3, 164, 368]. During voluntary eccentric contractions, this dependency on angular velocity is less pronounced or absent than during concentric contractions [368, 376]. Studies using gravity corrected

moments have shown an increase [256], a decrease [284] or no change [3, 376] in H/Q ratio with increasing angular speed of concentric contractions velocity in normal subjects. Since the moment/angular velocity curves of the knee flexors and extensors are similar but not identical [3], it would contribute to a velocity dependent change in H/Q ratio.

Finally, moment generation during isovelocity knee strength testing is contraction-type specific with higher moments generated during eccentric contractions than during concentric contractions [164, 368, 376]. When calculating traditional H/Q ratios from isovelocity strength testing, the concentric moments generated by the knee flexors and extensors are utilized. During dynamic movement, however, the agonist, or prime mover, will be contracting concentrically while the antagonist, or stabilizer, will be contracting eccentrically. This has led to the concept of a dynamic control ratio, or H_{ecc}/Q_{con} [2, 3, 29, 339].

The goals of this study were to more rigorously develop and refine the concept of strength balance about the knee in the healthy, active subject using a joint angle, angular velocity, and contraction type specific portrayal of standard dynamometry strength data. We also present a more physiologic ratio (DCR) that incorporates the concept of contraction-type specific moments as would be generated during functional muscular co-contraction. Finally, the same concepts were applied to an ACL reconstructed subject group, in order to demonstrate the changes in neuromuscular balance that occur after injury or surgery.

MATERIALS AND METHODS

Subjects

A physically active, control group (n=30; mass 78.8±13.1 kg; age 26.3±4.8 years; 30 males) with no known knee pathology was compared to a surgical group (n=16; mass 77.8±16.6 kg; age 26.2±7.6 years; 9 males and 7 females). The surgical group was evaluated an average of 2.5 years (range 1-4) following hamstring tendon autograft ACL reconstruction. All subjects were physically active and participated in sport on a regular basis. All subjects classified themselves as participating in strenuous activity (IKDC) prior to their injury. For the surgical subjects, exclusion criteria included other or subsequent injuries to the ipsilateral limb, pain intensity greater than 6 cm on a 10 cm visual analogue scale during the strength test, use of performance enhancing drugs, and previous or present pregnancy. Written informed consent was obtained from all subjects. Ethical approval for this study was granted by the Faculty Committee on the Use of Human Subjects in Research of the Faculty of Medicine, University of Manitoba.

Surgical Technique & Rehabilitation

Arthroscopically assisted intra-articular autograft ACL reconstruction was performed on all of the surgical patients by one of two fellowship-trained orthopaedic surgeons. The procedure utilized a double looped semitendinosus (ST) (n=7) or semitendinosus and gracilis (STG)(n=9) autograft, through femoral condyle intra-articular technique. Kennedy ligament augmentation device (LAD) was used in nine of the subjects (3-ST, 6-STG). Post-operative rehabilitation was based on an accelerated

rehabilitation protocol [228] with immediate range of motion exercises and weight bearing as tolerated.

Subject Assessment

The surgical subjects completed a questionnaire that included the subjective component of the International Knee Documentation Committee (IKDC) form (International Knee Society Meeting, Toronto, Canada, 1991). A 10 cm VAS was used to assess pain intensity. Active range of motion of both knees was evaluated using a universal goniometer. The KT1000 arthrometer (Medmetric Corp. San Diego, CA) was used to assess anterior-posterior displacement of the tibia relative the femur. Three trials were performed on each knee with the difference between sides used for data analysis [76].

Isovelocity Dynamometry

Comprehensive multi-velocity strength testing of the knee extensors and flexors was performed on a Kin-Com 500H dynamometer (Chattecx Corporation, Hixson, TN). The subjects were tested using the isovelocity dynamometer mode (constant angular velocity) at ten angular velocities (±50, 100, 150, 200, 250°/s) with negative velocities corresponding to eccentric contractions of the prime movers. Three sub-maximal contractions were performed for familiarization prior to maximal effort contractions at each speed. Three maximal effort repetitions were recorded at each speed with a five-second pause between the concentric and eccentric contractions. Test-retest reliability of this protocol was very good (ICC = 0.89). The resultant knee joint moment was gravity corrected for the moment of the weight of the leg and foot [3, 107, 376]. All resultant joint

moment were normalized to body mass (Nm/kg) which has been validated for multiple velocity and isometric moment data arising from young, active individuals [382].

Data Analysis

Conventional H/Q ratio's were calculated for the surgical and control groups. The average moment of the three trials performed at each velocity was recorded. The peak moment generated by the knee flexors were divided by the peak moments generated by the knee extensors to determine the H/Q ratio.

Colour-coded relief maps, termed strength maps, were created from the standard dynamometry data using custom dynamometry software (Isomap, Isodyne Inc., Winnipeg, Canada). The dynamometer moment (M_D in Nm) was derived as the product of the force (N) and the moment arm (m). The moment arm corresponded to distance from the point of force application on the force transducer to the dynamometer's axis of rotation. The knee joint angle, angular velocity and M_D data were collected at 100 Hz sampling rate. The data was exported to custom software for analysis. In the spline process, the sampled moment/angle data is fit with a cubic polynomial equation and equally spaced moment values are generated over the tested range of motion for all velocities tested. Then, the spline is applied across velocities to perform an interpolation of the moment values over a 50 °/s span for concentric and eccentric contractions. This interpolation is permitted based upon the very good to high reliability that has been reported for peak moment and angle specific moment measurements [20,23]. A 50 x 50 matrix provides a map resolution of 1.6 ° for knee joint angle and 10 °/s for angular velocity. The knee joint moment (M_K) was determined by correction of M_D for the moment of the weight of the leg and foot (plus resistance pad). For each repetition, the

peak moment (Nm), the angle of peak moment (°) and the work (J) were derived. The M_K were normalized to body mass (Nm/kg) based upon a strong correlation ($r = 0.89$, $p < 0.01$) between M_K and body mass. We used body mass normalization of the lower limb strength data to permit valid comparison between groups with different male: female compositions, since this approach has been validated for multiple velocity and isometric moment data arising from young, active individuals [59, 165, 382].

Strength maps were developed for the knee flexors and extensors of both the control and the hamstring ACL reconstruction groups. Individual maps were individually inspected for consistency and then averaged for each group. Division of the averaged knee flexor strength map by the averaged knee extensor strength map created an angle and velocity matched H/Q ratio map. The colours portrayed on the H/Q ratio map represent the H/Q ratio, however; each moment is matched on a point-to-point basis for both knee joint angle and angular velocity, effectively creating a display of 2500 angle and velocity matched ratios.

Modified dynamic control ratio (DCR) maps were also created by modifying the knee flexor strength map so that the eccentric contractions were assigned a positive velocity. (horizontal mirror image). Dividing the modified knee flexor map by the knee extensor map then created H/Q ratio maps. Again, each point represents the knee joint angle and angular velocity matched DCR ratio and we can now evaluate both H_{ecc}/Q_{con} and H_{con}/Q_{ecc}. Difference maps were created by subtraction of the averaged control group map and the hamstring ACL group for both the H/Q Ratio maps as well as the DCR maps in order to quantify differences between the groups.

Subject data

At the time of testing, 13/16 of the surgical subjects had returned to a strenuous level of activity while 3/16 described their activity as moderate (IKDC). The surgical group (n=16, 9 males, 7 females) had an average body mass of 77.8 ± 16.6 kg, an average age of 26.2 ± 7.6 years. The control group (n=30, males) had an average mass of 78.8 ± 13.1 kg and an average age of 26.3 ± 4.8 years. There were no statistical differences in body mass or age between the groups.

In the surgical group, seven subjects injured their non-dominant limb and nine injured their dominant limb. Intra-operatively, there were 5 medial meniscal tears, 3 lateral meniscal tears and 1 medial and lateral meniscal tear. Five subjects had associated medial collateral ligament injuries that were treated non-operatively. There was an average 24 months between injury and time of surgery.

Post-operatively, subjects attended physiotherapy and average of 11.6±9.4 months (range 1-28). The average time between surgery and strength testing was 30.4 ±10.5 months. KT1000 values at time of testing demonstrated an average side-to-side difference of 2.39 (range –1-7.3). There was no significant correlation between limb dominance, physiotherapy length, or KT1000 values and H/Q ratio's or DCR's.

RESULTS

Conventional H/Q Ratio's

Using conventional methods for calculation of the H/Q ratio demonstrated an average H/Q ratio in the control group of 0.438 and in the ACL reconstructed group of 0.55. This was statistically significantly different with a P-value of less than 0.001.

Hamstring/Quadriceps Ratio Maps

Control Group

Angle and velocity matched H/Q ratio maps were reproducible for control subjects Variation based on knee joint angle and angular velocity was demonstrated in the H/Q ratio throughout the H/Q ratio map (figure 1A). With the knee at near full extension, the knee flexors are able to generate larger moments than the knee extensors and the H/Q ratio reaches 1.42 during eccentric contractions. At angles of increasing knee flexion the knee extensors are able to generate larger moments than the knee flexors and the H/Q ratio drops to below 1. During concentric contraction, especially at larger angles of flexion, the H/Q ratio approaches 0.0. Variation in the H/Q ratio due to angular velocity was less obvious than with joint angle. During eccentric contractions (negative angular velocity) the H/Q ratio remained relatively constant but during concentric contractions, the H/Q ratio decreased with increasing angular velocity. Variation in the H/Q ratio was clearly apparent between contraction types. The H/Q ratio was much higher during eccentric contractions than during concentric contractions for all joint angles and all angular velocities.

ACL Reconstructed Group

Angle and velocity matched H/Q ratio maps were reproducible for hamstring ACL reconstruction subjects. Variation was demonstrated with respect to knee joint angle, angular velocity and contraction type (figure 1B). At angles of near full knee extension, the knee flexors are able to generate larger moments than the knee extensors and the H/Q ratio reaches 1.33. The H/Q ratio remained relatively constant during

eccentric contractions but decreased with increasing angular velocity during concentric contractions. The H/Q ratio was much higher during eccentric contractions than during concentric contractions for all joint angles and all angular velocities.

Discernable differences were detected between the H/Q ratio maps of the control and the ACL reconstruction groups. The hamstring ACL reconstruction group demonstrated a much larger proportion of the map area where the knee extensors dominate (a H/Q ratio that approaches 0.0 (blue area)). This area covers 43.6% of the map compared to 25.9% of the map in the control group.

A difference map (control-ACL reconstructed) was created in order to quantify differences between the control and ACL reconstruction groups (figure 1C). During eccentric contractions, the H/Q ratio decreases by up to 44.3% compared to control. This deficit occurs at higher joint angles, through all angular velocities. During concentric contractions, the H/Q ratio decreases by up to 24.6%. This deficit occurs during slow to medium angular velocities, through the middle range of motion of the knee joint.

Dynamic Control Ratio Maps

Control Group

Angle and velocity matched DCR maps were reproducible for control subjects. DCR maps for knee extension (H_{ecc}/ Q_{con}), demonstrated variation based on knee joint angle with ratio's varying from near zero (during high speed eccentric contractions) to 1.57 (during concentric contraction at knee joint angles nearing full flexion (figure 1A). Less variation with knee joint angle is seen in the knee flexion DCR (H_{con}/Q_{ecc}) with ratio's

peaking near unity. Variation based on angular velocity is more apparent during the knee flexion DCR than the knee extension DCR.

ACL Reconstructed Group

Angle and velocity matched DCR maps were reproducible for hamstring ACL reconstruction subjects. Variation was seen with respect to knee joint angle, angular velocity and contraction type in the ACL reconstructed group (figure 2B). During knee extension, the DCR reaches 1.35, and during knee flexion it reaches 0.75.

Discernable differences were detected between the control and the ACL reconstruction groups. A much larger proportion of the ACL reconstruction DCR map area has a ratio that approaches 0.0 (blue area), where the knee extensors dominate. This area covers 47.4% of the map compared to 29.9% of the map in the control group.

The difference map highlights and quantifies the differences between groups (figure 2C). During both knee extension and knee flexion, the DCR decreases by up to 42% of control. The knee extensor deficit occurs at higher joint angles, across all angular velocities, but is greatest at the high velocities. The knee flexor deficit occurs during slow to medium angular velocities, through the middle range of motion of the knee joint.

DISCUSSION

This paper represents a unique look at strength balance about the knee in both normal subjects and subjects who have undergone hamstring ACL reconstruction. In contrast to the single value traditional H/Q ratio, the use of H/Q Ratio and modified Dynamic Control Ratio maps demonstrate variation that is dependent on knee joint angle, angular velocity and contraction type. Changes in the H/Q ratio or DCR after hamstring

ACL reconstruction represent regional strength changes in the knee extensors, the knee flexors, or both. These findings highlight the importance of a comprehensive portrayal of strength (knee joint angle specific, multiple angular velocities, concentric and eccentric contractions) as the use of traditional peak moment data will not identify regional strength changes. A change in the single value H/Q ratio would not necessarily occur even though there were substantial changes in strength balance about the knee at angles other than the angle of peak moment, speed or contraction type tested.

The H/Q Ratio map and the DCR map are sensitive to discern between an active healthy control group with no knee injury and an ACL reconstructed group.

The role of strength balance for the optimization of performance in healthy and injured athletes is still being investigated. Some studies have demonstrated that there may sport specific alterations in the H/Q ratio in certain groups depending on the performance demands [145]. Theoretically, the alteration of the H/Q ratio can be extended to rehabilitation after injury and surgery. The role of the knee flexors as an ACL agonist in the stabilization of the knee joint during movement has been well documented [291, 335]. In patients who have undergone ACL reconstruction, graft rupture continues to be an ongoing concern. Thus, restoration or alteration of the H/Q ratio in an ACL injured or reconstructed population may have important implications for the athlete's ability to function at high-level sport and for the prevention of further injury.

In healthy active subject in this study, the H/Q Ratio map and the modified DCR map demonstrate a shift in favour of the knee flexors near full knee extension. This compares with the data from Aagaard [2], where functional H/Q ratios approach unity for fast knee extension (240°/s at 30°-knee flexion) in healthy individuals. The knee flexors

have been demonstrated to provide a stabilizing posterior directed force on the tibia during contraction of the knee extensors [291, 335]. Kellis [200] demonstrated that co-contraction of the hamstrings during maximal effort knee extension significantly alters the resultant joint moment of the knee extensors. The increase in the H/Q ratio and DCR in extended joint angles represents a relative dominance of the knee flexors. This may represent an improved ability of the knee flexors to stabilize the knee joint when the strain on the ACL is the greatest [33]. The shift in DCR at angles of knee extension may also reflect a limitation in knee extensor motor unit recruitment at joint angles of greatest ACL strain.

In the ACL reconstructed group, this dominance of the knee flexors in relative knee extension is more pronounced. This finding is consistent with the theory that knee flexor dominance at angles of greatest ACL strain may exist to help protect the knee with an ACL injury. In the ACL reconstructed subject, in the presence of a knee extensor deficit, residual instability, even slight, may require maintenance of hamstring strength, at extended joint angles to dynamically stabilize the knee joint. This theory is consistent with Hiemstra et al [164], who demonstrated that after hamstring tendon ACL reconstruction, knee flexor deficits were seen in regions of knee flexion but not during knee extension. It is also consistent with the finding that the muscle recruitment order in an unstable knee is altered in favour of earlier hamstring and gastrocnemius firing [335, 382]. Thus, regional strength changes in the knee flexors and knee extensors after ACL reconstruction lead to an overall increase in the H/Q ratio, favouring the knee flexors at full knee extension.

The physiological mechanisms behind regional strength deficits leading to altered strength balance about the knee joint are not fully understood. The most plausible explanation for the regional strength deficits is from altered motoneurone recruitment/activation during different speeds, contraction types and ranges of motion. The most plausible & parsimonious hypothesis (awaiting testing) is that some alteration in neural regulation of motoneurone activity has taken place that has resulted in variations in the moment producing capabilities of the knee flexors and extensors resulting in regional strength deficits, altering the strength balance about the joint. In addition to altered neural control from higher centers, one possible explanation in this ACL reconstructed population is altered afferent input from the knee joint itself. It has been well documented that peri-articular tissues, including ligaments, joint capsule, menisci have afferent nerve endings in the form of mechanoreceptors and nociceptors [395]. Certainly in animal models, the relationship between knee joint afferent information and efferent motor output has been well established. Stimulation of the knee joint afferents has been demonstrated to cause increased EMG in the knee flexors and inhibited EMG in the knee extensors [24, 290]. Grigg and Hoffman [144] hypothesized a direct effect on alpha motor neurons producing a protective reflex to potentially harmful stimuli. More likely, joint afferents play a role in the ongoing regulation of muscles about the knee joint through the gamma spindle system [187]. Certainly, afferent information from the knee joint does play a role in influencing the moment generating capability of the muscles about the knee. Marshall and Tatton [239] injected local anaesthetic into the knee joint and demonstrated alteration of the EMG response of the agonist and antagonist pairs causing an 'unbalancing" of co-contraction. In addition to periarticular afferent receptors, signals

from the muscle spindle and Golgi tendon organs in the muscle that span the knee joint could alter motor unit recruitment via Ia, Ib, and type II afferent systems [24].

In human subjects, the direct influence of nociceptive afferent information directly or indirectly influencing motor output has been more difficult to demonstrate. In this study, the ACL reconstruction subjects have multiple sources for altered afferent input from the injured knee. After the initial trauma causing rupture of the ACL a period of detraining may induce muscle atrophy. Interruption of proprioceptive information from the ACL can contribute to altered afferent input. Persistent knee pain and effusion can contribute to ongoing nociceptive input. Surgical intervention consists of arthroscopy, debridement, notch-plasty, hamstring tendon harvest, tibial and femoral tunnel drilling and graft fixation. All of these may play a role in the alteration of afferent input that originates from the knee and surrounding tissues. This altered afferent input may then contribute to changes in motor output leading to specific strength changes that alter the balance of strength about the knee joint.

In this study, we have attempted to more clearly define strength balance about the knee joint by portraying standard dynamometry data as angle specific, angular velocity specific and contraction type specific H/Q ratio and DCR strength maps. The marked systematic variability in the H/Q ratio seen in these maps reveals the limitations of single value peak moment traditional H/Q ratio in trying to describe neuromuscular balance about the knee. We have clearly demonstrated that neuromuscular balance about the knee is actually an imbalance. Changes in neuromuscular balance about the knee do occur with injuries such as ACL rupture and reconstruction. The feasibility of using these

maps to portray muscle balance is shown by the ability to differentiate specific imbalances present in the ACL reconstructed group when compared to control.

The exact etiology of the changes in muscle balance about the knee after injury and rehabilitation has yet to be determined. However, these insights into the neuromuscular control of the knee have practical clinical application. Dependent on the post rehabilitation goals of the patient, graft donor site selection may be directed by the knowledge of specific strength deficits known to occur after specific autografts. More importantly, if regional strength deficits and alteration in H/Q ratio are known to occur after ACL reconstruction, post-operative rehabilitation programs can be modified to minimize or correct these specific imbalances. This may lead not only to an improved functional outcome for the patient, but may also prevent further injury.

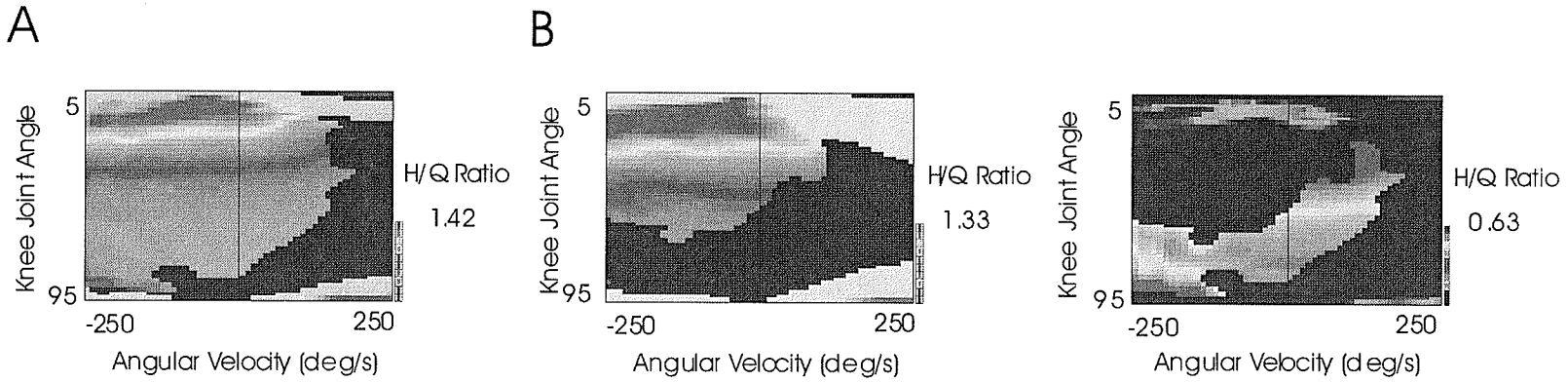

Figure 1: Hamstring/Quadriceps ratio maps of the A. Control group (n=30), B. Hamstring ACL reconstruction group (n=16). C. Difference map showing the subtraction of the ACL H/Q ratio map from the control H/Q ratio map. Colours on the map represent the H/Q ratio. Knee flexors dominate at the more extended joint angles (red areas) and during eccentric contractions. Knee extensors dominate at more flexed joint angles and during concentric contractions (blue areas). Alteration in H/Q ratio occurs during more flexed knee joint angles with preservation of knee flexor dominance in angles where strain on the ACL is greatest. The single value traditional H/Q ratio of 0.5-0.6 would be green in colour.

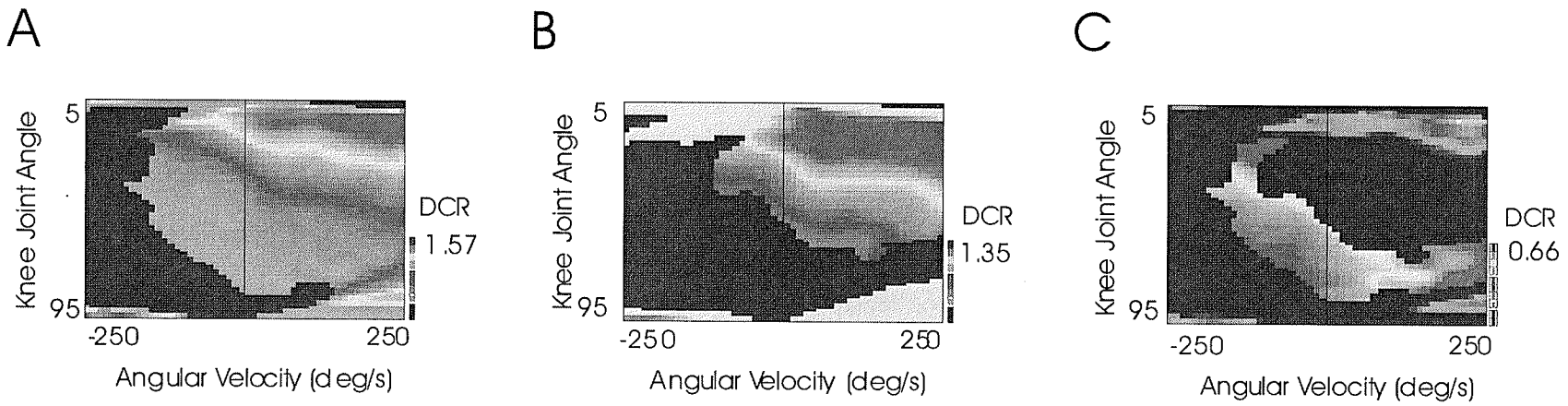

Figure 2: Dynamic Control Ratio (eccentric hamstring/ concentric quadriceps) of the A. control group and B. hamstring ACL reconstruction group. C. Difference map showing the subtraction of the ACL DCR map from the control DCR map. The colours now represent the DCR. The knee extension DCR is on the right side of the map and the knee flexion DCR is on the left side of the map. After hamstring ACL reconstruction the knee flexor dominant region (red) becomes larger. This is the knee joint angle at which strain on the ACL is the greatest. The single value traditional H/Q ratio of 0.5-0.6 would be green in colour.

Study 3 – Contralateral Leg Strength Deficits after Hamstring Tendon ACL Reconstruction

The objective of this study was to determine whether the strength of the contralateral limb knee flexors and extensors returns to that of an uninjured control group. This will demonstrate that the neuromuscular effects of a unilateral insult or intervention, such as an ACL reconstruction, are not confined to the ipsilateral limb but have effects on the contralateral limb. This will have important implications for the appropriate use of the contralateral limb as a control limb. In addition, it may have important implications for returning subjects to their previous levels of sport and function.

This Manuscript was submitted to *The Journal of Clinical Biomechanics* in September 2002. Ethics approval for this study has been granted by the University of Manitoba (E96:246). This study has won the **Aircast Award for Clinical Science** at the American Orthopaedic Society for Sports Medicine (AOSSM), Annual Meeting, Keystone Colorado, 2001; **Best research paper,** American College of Surgeons, Manitoba Chapter Annual Meeting, Winnipeg, Manitoba, 1999; **Frederick Robert Tucker Award for Orthopaedic Excellence,** Manitoba-Saskatchewan Annual Resident's Research Day, 1999.

A review of literature includes strength studies in cross-education, neural cross over effects in animals and humans, nociceptive crossover. A review of de-conditioning, which applies to this manuscript can be read in the literature reviews for manuscript 4.

The studies included in this thesis use a healthy active group with no previous knee injury as a control. In contradistinction, published studies testing strength after ACL

reconstruction have largely used the contralateral limb as a control (Table 3 and 4). Several studies have identified strength deficits when compared to a control group which are larger than when compared to the contralateral, supporting the theory that that the contralateral limb may also be altered [52, 164]. Given that the contralateral uninjured limb may not necessarily be unaffected by injury, surgical intervention and subsequent rehabilitation, the role of neural cross-over needs to be investigated.

Cross-Education of Strength Gains

In the strength literature, effects on the contralateral limb have been well described. Strength training of one limb has shown to cause strength gains in the contralateral untrained limb for isometric [67, 317, 372], concentric [172, 174, 193, 370], eccentric [173, 371] and stimulated contractions [53, 171].

Neural Cross-Over in Animals

In the neuroscience literature, cross-over or contralateral effects have been demonstrated in animals. Appelberg et al [13] demonstrated that full extension of the contralateral limb of the cat increased the dynamic sensitivity of the primary afferent nerves from the soleus. This effect disappeared with contralateral joint de-nervation. With intra-articular lidocaine injection, the effect disappeared but ankle plantar flexion was still able to induce increased sensitivity. In addition, contralateral leg extension caused changes in amplitude and rate of primary afferent spindle response [14]. There were no changes detected during contralateral leg flexion and no associated changes in EMG. They attributed this to effects on the gamma system. In further experiments extension of the leg was compared to mechanical stretching of the hamstrings (biceps and semitendinosus) and triceps surae [15]. There was no effect of triceps surae stretch and only 40% of the hamstring stretches caused afferent firing.

Neural Cross-Over in Humans

Robinson et al [294] delivered a stimulus to the posterior tibial nerve in the popliteus fossa in healthy women and measured H reflex (excitability of the motoneurone pool). They demonstrated facilitation of the motoneurone pool of the contralateral limb with a latency of 75-300 ms. Other researchers have demonstrated H reflex facilitation in response to cutaneous stimulation of the contralateral limb [278]. In other human studies of healthy subjects, active and passive movement of the contralateral limb causes inhibition of the soleus H-reflex [61, 69, 247].

Cheng et al [61] demonstrated that during passive movement of the contralateral limb there was inhibition of the soleus H-reflex. This inhibition is highly velocity dependent but was not angle (phase) dependent. There was no interaction found between the positions of the two limbs. Maintenance of this inhibition during tonic soleus contraction suggests that the mechanism of inhibition is via presynaptic inhibition. The insignificance of the inhibition once 30% maximum voluntary contraction is reached for the tonic contraction suggests the reflex modulation is under control of higher centres. McIlroy et al [247] showed that with passive movement of the contralateral limb, there is inhibition of the soleus H-reflex. The level of inhibition is about half way between that of still sitting and movement of the ipsilateral limb. Collins et al [69] showed that passive movement of the contralateral limb caused inhibition of the soleus H-reflex and that this was velocity dependent. McIlroy et al [247] showed that with active movement of the contralateral limb, there was a phase dependent inhibition of the soleus H-reflex. The reflex was inhibited at 50 and 75% of the cycle (about half of sitting). However, at 25 and 100%, the inhibition was equal to that of ipsilateral or bilateral movement. The greatest amount of inhibition occurred during the flexion phase of the movement.

In ACL deficient subjects, EMG of the knee flexors and extensors after fatiguing exercise demonstrated decreased muscle activity in both the ACL deficient and the contralateral limb compared to control [362]. Wojtys et al [382] demonstrated decreased intermediate response (130-170 ms) and voluntary activity in both knee extensors and knee flexors of the uninvolved limb in ACL deficient subjects. Afferent feedback from the injured knee could affect the strength of the contralateral limb via contralateral connections in the spinal cord. These contralateral effects also involve the central nervous system. Kristeva [211] demonstrated that the motor cortex is activated bilaterally during a unilateral task.

Nociceptive Cross-over

Finally, specific activation of nociceptors has been demonstrated to have contralateral effects. Solodkin et al [334] induced unilateral hind limb inflammation in the rat. They sacrificed the rats 6 hours later and observed bilateral staining in the spinal cord for NADPH-d activity, which may function like nitric oxide synthase, a contributor to spinal nociceptive processing. Bileviciute-Ljungar et al [40] created experimentally induced inflammation into the hind paw of a rat. They showed that the contralateral local injection of anaesthetic reduced pain related behaviour and reduced edema formation. Further studies demonstrated that the injection of an opioid receptor agonist into the contralateral hind paw attenuated anti-nociceptive reflexes in response to the experimentally induced inflammation. This effect was not observed during systemic administration of the drug and was abolished by denervation of the sciatic nerve [41].

The preceding review of literature demonstrates that there are contralateral effects to an ipsilateral perturbation in both humans and animals. Strength gains have been demonstrated in the untrained contralateral limb which are contraction type dependent [370,

[371]. Movement of the contralateral limb has been demonstrated to alter motoneurone output to the ipsilateral limb in both humans and animals [14, 69, 247]. Finally, nociception has been demonstrated to have both ipsilateral and contralateral effects [40]. The existence of pathways for both afferent and efferent information to the contralateral side are consistent with the theory that after injury, neuromuscular effects are not confined to the injured limb but may have effects on the contralateral uninjured limb.

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Contralateral Leg Strength Deficits after Hamstring Tendon ACL Reconstruction

Laurie A. Hiemstra, MD, FRCSC, Sandra Webber, BMR(PT),MSc,
Peter B. MacDonald, MD, FRCSC and Dean J. Kriellaars, PhD

Sport Medicine Centre
University of Calgary, Calgary, Alberta T2N 1N4

School of Medical Rehabilitation, Section of Orthopaedic Surgery, Faculty of Medicine
University of Manitoba, Winnipeg, Manitoba, R3E 0W3

Corresponding Author:

Laurie A. Hiemstra, MD, FRCS(C)
Sport Medicine Centre
University of Calgary
2500 University Dr. NW
Calgary, AB
T2N 1N4

Phone 403-220-5077
fax 403-282-6170
email hiemstra@ucalgary.ca

ABSTRACT

Objective The purpose of this study was to compare knee flexor and extensor strength of the ACL reconstructed limb with the contralateral uninjured limb and an uninjured control group.

Design Cross-sectional study

Background Bilateral strength normalization is a rehabilitation milestone after ACL reconstruction where strength of the reconstructed limb is within 10% of the contralateral limb. The underlying assumption is that the contralateral limb serves as a control that is unaffected after ACL rupture, reconstruction and rehabilitation.

Methods Subjects with a hamstring tendon ACL reconstruction (n=12) were compared to an active control group (n=30). Bilateral knee flexor and extensor isovelocity strength was evaluated (five speeds, 5-95°, concentric and eccentric contractions). Quality of life was assessed using a validated questionnaire.

Results After hamstring tendon ACL reconstruction and rehabilitation, bilateral strength normalization is achieved by the knee extensors but not the knee flexors when compared to the contralateral uninjured leg. When compared to the uninjured control group, the knee extensors and knee flexors of both the ACL reconstructed and the contralateral uninjured legs demonstrated large and statistically significant strength deficits. A positive correlation was observed between concentric knee extensor peak moment and the quality of life assessment. The contralateral knee flexor strength exhibited an eccentric strength deficit when compared to control data.

Conclusions These findings limit the validity of the use of a contralateral leg as a rehabilitation endpoint or as a control in the ACL reconstructed population.

Relevance This paper presents important findings regarding strength deficits that occur in the contralateral uninjured leg after ACL reconstruction. Possible physiologic mechanisms including de-conditioning, incomplete rehabilitation, and neural-mediated effects on contralateral strength are discussed.

Keywords: strength, dynamometer, rehabilitation, moment, torque, ACL, isovelocity

INTRODUCTION

The primary goal of anterior cruciate ligament (ACL) reconstruction surgery is to re-establish a stable knee joint that enables patients to return to their previous activities and functional level after a period of rehabilitation [110]. Functional outcome has been shown to have a positive correlation with strength measurements [314, 378] and knee extensor strength has been shown to be related to self-reported return to function [19, 255, 295]. Studies examining knee extensor strength after hamstring tendon ACL reconstruction and subsequent rehabilitation have demonstrated an average strength deficit of 13% (9-17% range) when compared to the uninjured contralateral leg [151, 164, 234, 390]. When compared to age and activity matched uninjured peers, Hiemstra et al [164] have demonstrated a relatively larger knee extensor strength deficit of 30%.

Given the widespread utilization of the semitendinosus and gracilis tendons as autografts for ACL reconstruction, there is need to examine the impact on knee flexor strength. Studies measuring knee flexor strength after hamstring ACL reconstruction and subsequent rehabilitation have reported an average strength deficit of 13% (10-17% range) when compared to the uninjured contralateral leg [34, 234, 258]. Other researchers,

however, have failed to observe knee flexor strength differences after hamstring tendon ACL reconstruction [151, 390]. Comparison to age and activity matched uninjured peers has demonstrated larger strength deficits of up to 25% in the knee flexors (fast and slow contractions, eccentric greater than concentric, angles greater than 60°)[164]. The inconsistency of these findings can, in part, be attributed to methodological differences. Studies showing knee flexor strength deficits used a more comprehensive method of dynamometry and analysis including multi-speed testing, body mass normalization, and gravity correction for the moment of the weight of the leg and foot.

In the current rehabilitation and sport medicine literature there have been no objective measures validated to help indicate the endpoint of rehabilitation after ACL reconstruction. Most therapy programs use the achievement of 'bilateral strength normalization', defined as the equalization of the strength of a muscle group of the operated or injured leg to within 10% of the contralateral uninjured leg, as an objective outcome measure for returning to sport and activity. Limitations however have been identified with using the contralateral limb as a control [356]. Bilateral strength normalization can therefore, only represent an endpoint of rehabilitation if the contralateral uninjured leg is unaffected.

Many factors may negatively impact on the ability of the contralateral uninjured leg to generate moments about the knee after ACL rupture. During the post-injury, pre-operative period, a substantial reduction in physical activity could result in de-conditioning of the contralateral leg [97]. Both neural and muscular factors can play a role. For instance, inhibition of the contralateral leg motoneurones has been demonstrated after arthroscopy [345]. Finally, a lack of angle specific, velocity specific and contraction type

specific exercises for both the knee flexors and extensors during the preoperative and postoperative period can contribute to both regional and global strength deficits in the contralateral leg. The determination of the exact nature of the strength deficits that exist in the ACL reconstructed population will provide useful information for: 1) modification of post-operative rehabilitation protocols, 2) establishing appropriate rehabilitation goals, and 3) establishing pre-operative, post-injury protocols.

The objective of this study is to measure strength in ACL reconstructed and contralateral legs of ACL patients and compared the results to the strength of uninjured age and activity matched control subjects. We hypothesize that, after hamstring tendon ACL reconstruction and subsequent rehabilitation, bilateral strength normalization would be achieved for the knee extensors but not for the knee flexors, and that both the knee flexor and knee extensor strength would be reduced when compared to an age and activity matched control group.

Methods

Twelve subjects greater than one-year post hamstring tendon ACL reconstruction were compared to a physically active, control group (n=30) with no known knee pathology. All subjects provided written informed consent to participate in the study. Ethical approval for this study was granted by the Faculty Committee on the Use of Human Subjects in Research of the Faculty of Medicine, University of Manitoba.

The control group consisted of 30 male subjects. The average mass of the group was 78.8±13.1 kg, and the average age was 26.3±4.8 years. All subjects reported that

they participated in exercise or sport greater than two times per week at moderate or high intensity.

The surgical group consisted of seven males and five females. For the surgical subjects, exclusion criteria included other or subsequent injuries to the contralateral or surgical limb, pain intensity greater than six on a ten centimeter visual analogue scale (VAS) at time of recruitment, use of performance enhancing drugs, and previous or present pregnancy. The average age of the group was 26.6 ±6.7 years, and the average body mass was 69.8 ± 13.8 kg. The average time since hamstring ACL reconstruction was 40.6 ± 14.9 months. The semitendinosus tendon autograft was utilized in nine subjects and the semitendinosus and gracilis tendons were utilized in three subjects. Comparisons of the semitendinosus to the semitendinosus and gracilis harvest groups demonstrated no difference in knee flexor or extensor resultant joint moment between the groups, and were therefore analyzed together. Hamstring tendon autograft ACL reconstruction was performed using an arthroscopically-assisted technique. For all the subjects, the post-operative rehabilitation was based on an accelerated rehabilitation protocol with early weight bearing and early range of motion [228, 318] (Table 1). There were no documented post-rupture, pre-operative rehabilitation interventions in any of the subjects.

Sample size was determined using conservative estimates of mean expected difference and variance as well as a power index of 2.92 (.05 alpha; 0.1 beta). The minimum number of subjects required for the study was determined to be eight.

Evaluation

Operative subjects underwent subjective and objective assessment using a modified International Knee Documentation Committee (IKDC) form (International Knee Society Meeting, Toronto, Canada, 1991). Active range of motion of both knees was evaluated using a universal goniometer. The Lachman and pivot shift tests were performed to test clinical knee instability. One examiner tested all subjects and testing was completed bilaterally. The KT1000 arthrometer (Medmetric Corp. San Diego, CA) was used to assess anterior-posterior displacement of the tibia relative the femur. Three trials were performed on each knee and averaged. The difference between sides used for data analysis [76]. Eleven of the subjects also completed a modified quality of life questionnaire [251].This questionnaire provides a patient-based assessment of quality of life (maximum score, 100) with domains of symptoms and physical complaints, work-related concerns, recreational activities and sport participation or competition, lifestyle, social and emotional.

Strength Testing

The surgical group underwent strength testing of the knee extensors and flexors of both the operative leg and the uninjured contralateral leg using a Kin-Com 500H dynamometer (Chattecx Corporation, Hixson, TN). The standardized warm-up was completed on a Monark 818E cycle ergometer (Monark AB, Sweden) at 35 W for five minutes. The subjects were tested using the isovelocity dynamometer mode (constant angular velocity) at ten angular velocities (±50, 100, 150, 200, 250°/s) with negative velocities corresponding to eccentric contractions of the prime movers. Subjects were provided with submaximal familiarization trials with each speed of testing. The range of motion of the knee was set from 5° to 95° of knee flexion with full knee extension as the

zero degree reference. A detailed description of the technique for strength testing has been outlined previously [368].

The knee joint moment (M_K) data was corrected for the moment of the weight of the leg, foot and resistance pad. The peak moment for each velocity was determined as the average of the peak moments for each of the three repetitions. The angle of peak moment and work were also determined for each repetition. M_K was normalized to body mass (Nm/kg) based upon a strong correlation ($r = 0.89$, < 0.01) between M_K and body mass. Body mass normalization of lower limb strength in active healthy men and women has been validated in the literature [59, 101, 164, 165, 379, 382].

Strength maps (Isomap, Isodyne, Inc. Winnipeg, Canada) were constructed for the knee flexors and extensors of the contralateral and control legs of the surgical subjects. Strength maps portray standard isovelocity dynamometry data as a two-dimensional relief map. Details of strength map generation are found in Hiemstra et al [164] and Massey and Kriellaars [210]. Average knee extensor and knee flexor strength maps were then constructed for the contralateral and ACL reconstructed leg in the surgical subjects and for the dominant leg of the control group. Difference maps between legs and between surgical and control groups were made to identify regional strength changes and to quantify any strength deficits. A clinically significant strength deficit was defined as 10% of the contralateral uninjured leg or the equivalent of 0.3 Nm/kg which is approximately equal to 21 Nm for the knee extensors and 0.15 Nm/kg and approximately 10.5 Nm for the knee flexors.

Results

Questionnaire

Eleven of the twelve subjects completed a quality of life questionnaire suitable for ACL reconstruction patients. The average score was 75.9 ± 11.9 out of 100. Ten subjects scored above 70, while one subject scored 43. Pearson correlation coefficients were calculated for the concentric and eccentric peak moments and the quality of life score. Significant positive correlation was found between the quality of life score and concentric knee extensor peak moments ($r=0.61$, $p=0.027$).

Isovelocity dynamometry

Knee extensors

ACL vs. Contralateral

A comparison of knee extensor peak moments between the ACL reconstructed and contralateral uninjured leg showed no statistically significant strength differences between legs for either concentric or eccentric contractions and at any of the speeds tested (Figure 1). Using peak moments, the average strength deficit of the surgical leg was 5.3% (range 0 to -8.5%). There were no individual velocity or contraction type bilateral strength deficits found between legs using our definition of bilateral strength normalization (a 10% deficit). Evaluation of knee extensor strength maps (not shown) revealed a consistent pattern of M_k generation over the range of motion and over speeds in all individuals. Difference maps showed a significant 10.8% (0.26 /2.4 Nm/kg) difference in peak moment between the ACL reconstructed and the contralateral uninjured leg ($p<0.01$) which occurred mainly during eccentric contraction and at knee

joint angles 55-90°. There was a small difference in body mass normalized work (3.7% or 0.8/21.5 J/kg) performed between the ACL reconstructed leg and the contralateral leg.

ACL and Contralateral vs. control

Comparison of the contralateral uninjured and ACL reconstructed limb knee extensor peak moments with the age and activity matched control group revealed statistically significant strength deficits (Figure 1). Strength deficits were observed for the ACL reconstructed (24.8%, 0.707 Nm/kg averaged across all velocities) and contralateral (21.0%, 0.585 Nm/kg averaged across all velocities) limbs. The strength deficit was significant at each angular velocity. Evaluation of the strength maps (not shown) demonstrated regional differences in knee extensor strength for both the ACL reconstructed and contralateral leg located in the region where the peak moments are generated by the knee extensors (knee joint angle 55-90°). This finding is similar to the regional strength deficits reported by Hiemstra et al. [164]. In comparison to the control group, the ACL reconstructed leg revealed a 29.8% peak moment strength deficit (Nm/kg) and a 23.9% decrease in work performed (J/kg). The contralateral uninjured leg showed a 28.2% decrement in overall peak moment and a 21.0% decrease in body mass normalized work (J/kg) when compared to control.

Knee flexors

ACL vs. Contralateral

A comparison of knee flexor peak moments between the ACL reconstructed and the contralateral uninjured leg (Figure 2) revealed a statistically significant strength

deficit of 13.3% averaged across all angular velocities ($p<0.05$). Evaluation of knee flexor strength maps (not shown) revealed a consistent pattern of moment generation over the full range of motion and across speeds for each subject. Difference maps showed a 19.9% (0.28/1.41 Nm/kg) difference in moment between the ACL reconstructed and the contralateral uninjured leg ($p<0.01$) located during low velocity eccentric contractions, through all joint angles. There was a 14.5% (2.0/13.8 J/kg) difference in total body mass normalized work between the ACL reconstructed leg and the contralateral leg.

ACL and contralateral vs. control

When knee flexor peak moments of the contralateral and ACL reconstructed legs were compared to the control group, a statistically significant strength deficit was identified (Figure 2). The ACL reconstructed leg demonstrated an average 26.8% (0.349 Nm/kg) knee flexor deficit observed for all speeds and both contraction types when compared to controls. This deficit was found to be larger during eccentric contractions (0.46 Nm/kg) than during concentric contractions (0.23 Nm/kg) which was statistically significant ($p<0.01$).

The contralateral uninjured leg demonstrated an average 13.5% (0.18 Nm/kg) knee flexor peak moment strength deficit across all angular velocities when compared to controls. This difference was significant for all speeds (50-250 °/s) of eccentric contraction, as well as for low speed (50 °/s) concentric ($p<0.05$). The average contralateral uninjured knee flexor strength deficit during eccentric contractions was 0.26 Nm/kg or 18.1% and 0.095 Nm/kg or 8.9% for concentric contractions when compared to controls. Evaluation of the strength maps (not shown) demonstrated regional differences

in knee flexor strength for both the ACL reconstructed and contralateral leg located primarily to the region where the peak moments are generated by the knee flexors (angle 5-50°, high angular velocities, eccentric>concentric). A comparison to the control group using overall peak map moments revealed a 31.7% strength deficit, and a 28.7% decrease in work performed (J/kg) in the ACL reconstructed leg ($p<0.01$). The contralateral uninjured leg showed a 24.0% strength deficit based upon peak map moment and a 17% decrease in work when compared to control ($p<0.01$).

Discussion

This study investigates strength changes that occur in each lower limb after hamstring tendon ACL reconstruction and subsequent rehabilitation. This study demonstrates that bilateral normalization of knee extensor strength is possible using current accelerated rehabilitation protocols in hamstring tendon autograft ACL reconstructed patients. However, when compared to a control group of age and activity matched peers a statistically significant reduction of up to 25% knee extensor strength exists in both the ACL reconstructed and contralateral legs. In contrast to the knee extensors, bilateral strength normalization of the knee flexors was not achieved after hamstring ACL reconstruction. Bilateral deficits were greater eccentrically (13.8%) than concentrically (12.9%). When compared to age and activity matched controls, statistically significant strength deficits were observed in both the ACL reconstructed (20.0% concentrically; 26.9 % eccentrically) and the contralateral uninjured legs (8.1% concentrically; 15.2% eccentrically) were observed.

Since the early 1990's, knee extensor strength deficits have been documented after ACL reconstruction. Studies using modern isovelocity dynamometry techniques with moment of weight correction (gravity correction) have documented knee extensor strength deficits of 9-17% after hamstring ACL reconstruction when compared to the contralateral leg [151, 234, 390]. In this study, a bilateral strength comparison revealed a deficit of 5.3 ± 2.7 % for the operative limb at multiple speeds and for both eccentric and concentric contraction types. These data indicate that the goal of bilateral strength normalization is achievable with current rehabilitation programs. The success may be dependent on factors such as "accelerated" rehabilitation, quality of rehabilitation, and the fact that the knowledge of this strength deficit may have led to increased emphasis on knee extensor strengthening in recent years. This study demonstrates, however, that even though bilateral strength normalization is achieved for the knee extensors, large and significant bilateral strength deficits of up to 30% of control for all angular speeds evaluated still exist when compared to a group of age and activity matched peers.

Knee flexor strength deficits after hamstring tendon ACL reconstruction have been less consistently identified [34, 164, 234, 258]. This study has demonstrated that after hamstring tendon ACL reconstruction and subsequent rehabilitation, bilateral strength normalization is not achieved in the knee flexors, and a large significant knee flexor strength deficit persists in the ACL reconstructed leg when compared to the contralateral uninjured leg. This right-left strength difference is up to 20% and tends to be greatest during eccentric contractions. When comparison is made to an age and activity matched control group, both the ACL reconstructed and the contralateral uninjured leg demonstrate large knee flexor strength deficits of up to 30%.

Knee extensor strength training is an integral part of ACL rehabilitation and currently, bilateral strength normalization is the minimal requirement or guideline for returning to sport. The existence of knee extensor and flexor deficits in this athletic population after rehabilitation suggests that these active healthy individuals may be limited in their ability to function at their previous level in sport, recreation or vocation. Currently our ability to document functional restrictions in this population is limited to self-reported assessment of functional status. The relationship between function and knee strength has been previously reported [19, 295, 314, 378]. The correlation between concentric knee extensor strength and scores on the Quality of Life Questionnaire [251], in this study supports the concept that the residual physical impairment in this population may lead to functional limitations and confirms the importance of strength for the recovery of function and patient satisfaction. Given the existence of bilateral strength deficits of both the knee flexors and extensors when compared to control, a cautionary approach to the use of the contralateral uninjured limb as a control is necessary.

The functional significance of knee flexor and extensor strength deficits is less clear. Certainly, performance will be affected by a bilateral strength deficit and may put athletes at a disadvantage when compared to their peers. The existence of knee flexor deficits in this population may have significance for subsequent or re-injury. It has been reported that hamstring grafts may have an increase in re-rupture rate when compared to patellar tendon grafts [265]. Given that the hamstring tendons have been shown to act as an ACL agonist, resisting anterior tibial translation [291], a knee flexor deficit may compromise the ability to protect the graft in certain situations. Further, contraction type specificity must be considered as the eccentric deficits are substantially larger than

concentric deficits. Since the primary stabilization role of the hamstrings is eccentric, persistent large eccentric strength deficits may be important for dynamic stabilization of the knee and have implications both for protection of graft re-rupture as well as premature overloading of a fresh ACL graft. In addition, the existence of larger eccentric than concentric strength deficits in the contralateral uninjured limb will lead to a strength imbalance. This may increase the risk of rupture of the contralateral ACL which has been demonstrated to be more likely in subjects with one previous ACL rupture [150].

The etiology of the described strength changes in the ACL reconstructed and contralateral uninjured leg have yet to be fully elucidated. Certainly de-training, incomplete rehabilitation and neural inhibition need to be considered. The global strength deficit in the contralateral uninjured leg may also be secondary to de-conditioning. After an acute injury, this active population undergoes a period of reduced activity corresponding most closely to the model of training-detraining. Large decreases in strength and EMG have been demonstrated in healthy subjects after a period of training followed by a period of detraining [147, 174]. We can postulate, therefore, that after ACL reconstruction, and upon entrance into a post-operative rehabilitation program, these patients already have bilateral strength losses when compared to their pre-injury status.

It is reasonable to assume that the inability of current rehabilitation programs to achieve bilateral strength normalization in the knee flexors is related to the fact that knee flexor deficits after ACL reconstruction with a hamstring autograft have been unrecognized to date and therefore, current protocols may not be tailored to eliminate these deficits. Furthermore, the use of periodic objective testing of the progression of knee flexor strength is not a part of current rehabilitation protocols. The goals or

endpoints of rehabilitation continue to concentrate on returning the strength of the ACL reconstructed leg to that of the contralateral uninjured leg. The lack of recognition that the contralateral uninjured leg is most often de-conditioned and requires specific strengthening may further contribute to contralateral leg strength deficits. The use of pre-operative maintenance physiotherapy regimes concentrating on the contralateral leg, may limit the development of the contralateral strength deficits seen after the post-operative ACL reconstruction rehabilitation. In addition, studies on motivated elite athletes has suggested that rigorous rehabilitation may be able to reverse muscle inhibition after knee injury [176].

In addition to the effects of detraining and incomplete rehabilitation other neuromuscular factors may play a role. Afferent information from the ACL reconstructed knee and the hamstring graft harvest site could exhibit crossover effects in the contralateral leg. Appelberg [15] has demonstrated in the cat that afferent feedback from one leg can modify the primary afferent spindle response of the opposite leg. In humans, the H-reflex, an experimentally induced test of the monosynaptic reflex, can be elicited during different test conditions. It has been demonstrated that both passive and active movement of the contralateral limb causes inhibition of the H-reflex and that this inhibition is dependent on the velocity of movement [61, 69, 247]. During strength training it has been demonstrated that strength gains are seen in the contralateral limb when single limb strength training is performed [96]. Neural inhibition of both injured and contralateral legs has been demonstrated in patients with unilateral knee pathology when compared to uninjured reference population [345]. Based on these findings, neurally mediated alteration in the motor unit recruitment of the contralateral limb musculature may contribute to the

global and regional strength deficits identified in the contralateral limb. This explanation would account for strength differences demonstrated between contraction types and velocities.

De-conditioning, deficits in the rehabilitation process, as well as neural effects may all contribute to the existence of contralateral leg strength deficits after ACL reconstruction. Modification of the existing rehabilitation programs by the addition of contraction specific, velocity specific and joint angle specific exercises may reduce or help eliminate identified regional and global strength deficits. Initial rehabilitation goals or landmarks should be the achievement of bilateral strength normalization of both the knee extensors and the knee flexors. Once bilateral strength normalization is attained, a significant strength deficit remains when compared to age and activity matched controls. This bilateral strength deficit must be rectified before these patients can return to previous levels of sport and function. With current rehabilitation programs, further strength gains of 20-30% are required for both the knee extensors and the knee flexors before strength levels are consistent with control. Further rehabilitation should include sport specific and position specific strength training. If post injury but pre-surgical de-conditioning is contributing to the contralateral strength deficits, pre-selected individuals who are likely to require ACL reconstruction could be entered into a maintenance strengthening program for their contralateral uninjured leg. If successful, pre-surgical strength maintenance of the uninjured leg will help prevent the contralateral leg strength deficit. If this strength maintenance is achieved, then bilateral strength normalization will become a better measure of functional outcome.

In summary, we have demonstrated that current rehabilitation protocols are able to achieve their goal of bilateral strength normalization for the knee extensors. Substantial eccentric and low velocity concentric strength deficits are identified in the knee flexors of the ACL reconstructed leg when compared to the contralateral leg. In addition, this study has clearly demonstrated that after rehabilitation, strength deficits exist in both the ACL reconstructed and contralateral leg when compared to an age and activity matched control group. We recommend that bilateral strength normalization should be viewed, not as an endpoint, but rather as a milestone in the process of full rehabilitation after ACL reconstruction. The restoration of knee flexor and extensor strength to the level of that of peers may be important for protection of the ACL graft, the prevention of subsequent knee injury, and for optimum athletic performance. These findings suggest that a need for more comprehensive rehabilitation protocols exist. Suggestions for modification may include contraction type, knee joint angle, and angular velocity specific exercises as well as improved global strengthening. The addition of a maintenance-strengthening program for the contralateral leg in the pre-surgical stage may help prevent the contralateral leg strength deficit that is seen post operatively. Finally, and foremost these findings call into question the widespread use of the contralateral leg as a control when studying the ACL reconstructed population.

Conclusions

This study demonstrates that large and significant strength deficits are present in the contralateral uninjured limb after hamstring tendon ACL reconstruction and subsequent rehabilitation. In this population, bilateral strength normalization, or the

equalization of strength between legs to within 10% of the contralateral uninjured leg, of the knee extensors was achieved but not for the knee flexors. The results of this investigation support a cautionary approach in use of bilateral strength normalization as a rehabilitation endpoint and challenge us to use caution in the use of the contralateral limb as a control. The functional consequences of these knee flexor strength deficits requires further investigation however resolution of the eccentric knee flexor strength deficits may improve the ability to stabilize the knee joint. This will play a role in both in graft protection, contralateral limb ACL injury as well as in returning these patients to their previous levels of functioning.

Table 1. Accelerated rehabilitation protocol for hamstring tendon ACL reconstruction

ACCELERATED REHABILITATION PROTOCOL FOR HAMSTRING ACL RECONSTRUCTION
0-2 Weeks – Home based program Full-protected weight bearing ambulation No range of motion restrictions Passive knee extension and passive terminal knee flexion Active knee range of motion exercises Hip and ankle maintenance exercises Isometric quadriceps contractions with the leg in full extension Straight leg raises when tolerated
2-4 weeks – Physiotherapist based program Stationary bicycle exercises Gentle mobilizations of the patellofemoral and tibiofemoral joints
4-8 weeks – Physiotherapist based program Closed-chain exercises (leg presses and 30° squats) Progressive resisted exercises of the hip abductors, adductors, and extensors Isotonic and eccentric hamstring muscle strengthening in the standing position Ambulation without crutches was allowed when a near full range of motion was present (maximum, -5° extension) with no quadriceps muscle lag, the patient had no limp, and the patient could do a straight leg raise with 15 pounds of weight on the tibial tubercle
8-12 weeks – Physiotherapist based program Progressive isotonic resisted hamstring muscles exercises using free weights Hamstring and quadriceps muscle strengthening on an isokinetic dynamometer with 30° block Isotonic hip girdle exercises Swimming allowed, avoiding the whip kick Skating allowed (slow speeds, no explosive movements) Outdoor bicycling allowed (no hills, avoid full extension) Jogging commenced at 10 weeks
12-16 weeks – Physiotherapist based program Isokinetic exercises were continued with no extension block Return to hockey and non-pivoting sports was allowed at 16 weeks Figure-of-8 and agility training with a brace were commenced at week 20 Pivoting sports were allowed at 24 weeks (6 months). Brace should be used for sports for one year after return to sports

Figure 1

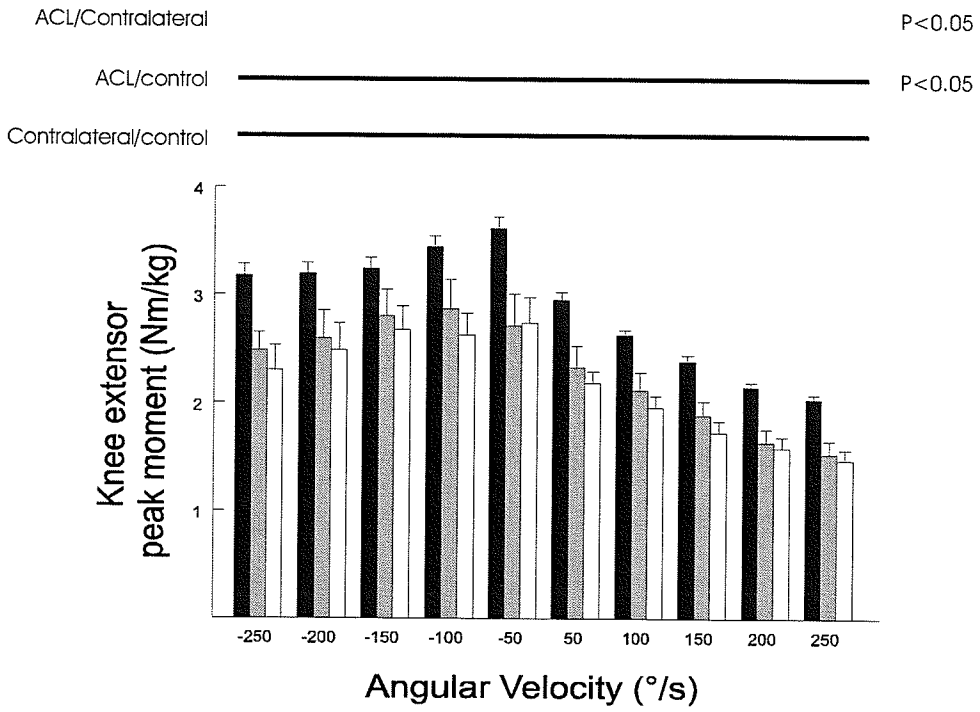


Figure 1. Peak moment/angular velocity relationship for the knee extensors. Knee extensor peak moments for the control group (black), the contralateral uninjured limb (gray), the ACL reconstructed limb (white). Negative angular velocity corresponds to eccentric contraction. Statistical differences are shown with filled bars ($p<0.05$).

Figure 2

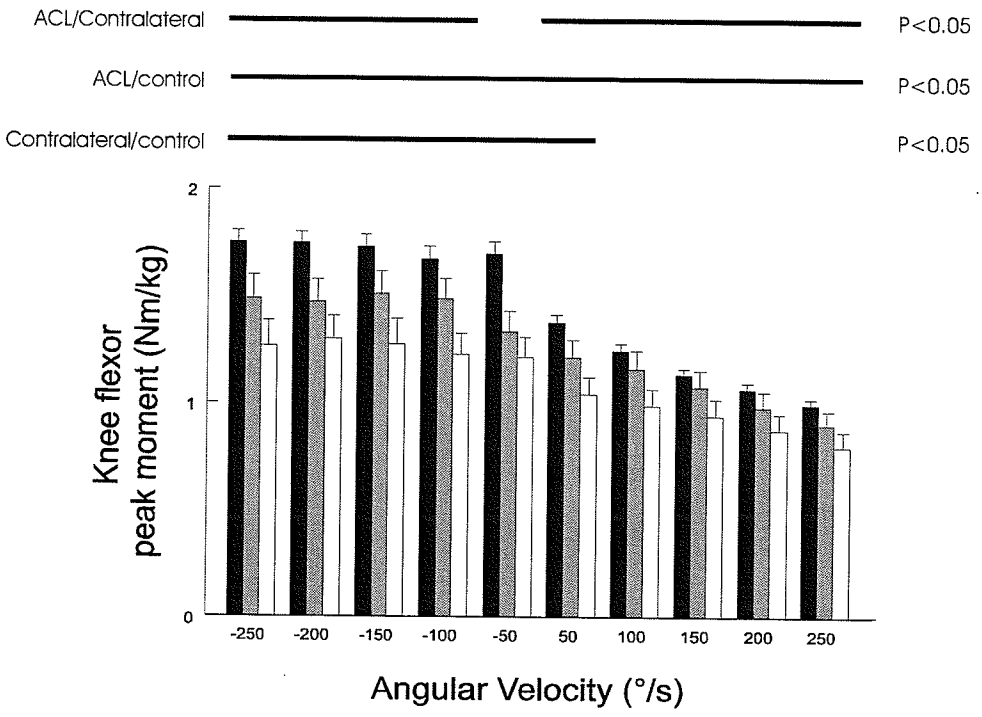


Figure 2. Peak moment/angular velocity relationship for the knee flexors. Knee flexor peak moments for the control group (black), the contralateral uninjured limb (gray), the ACL reconstructed limb (white). Negative angular velocity corresponds to eccentric contraction. Statistical differences are shown with filled bars ($p<0.05$).

Supplemental Figures

Figures 3 and 4 represent strength map data that is referred to but not referenced in the manuscript above.

Figure 3. Averaged strength maps of the knee extensors of the contralateral uninjured limb (A), and the ACL reconstructed limb (B). The difference map (C) quantifies the differences between the two limbs and highlights the regional strength deficits that exist after hamstring tendon ACL reconstruction. Hatched areas represent statistically significant differences in strength ($P<0.01$).

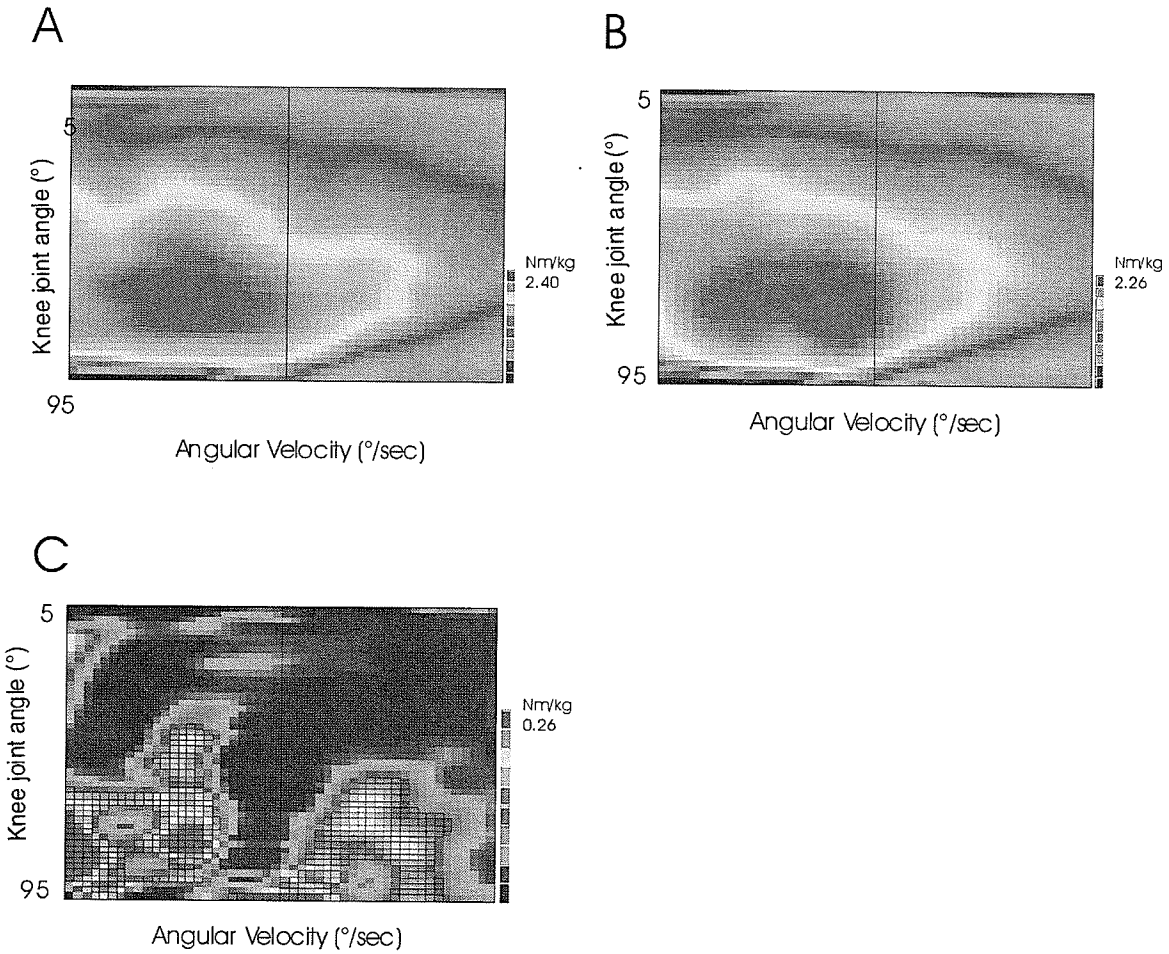
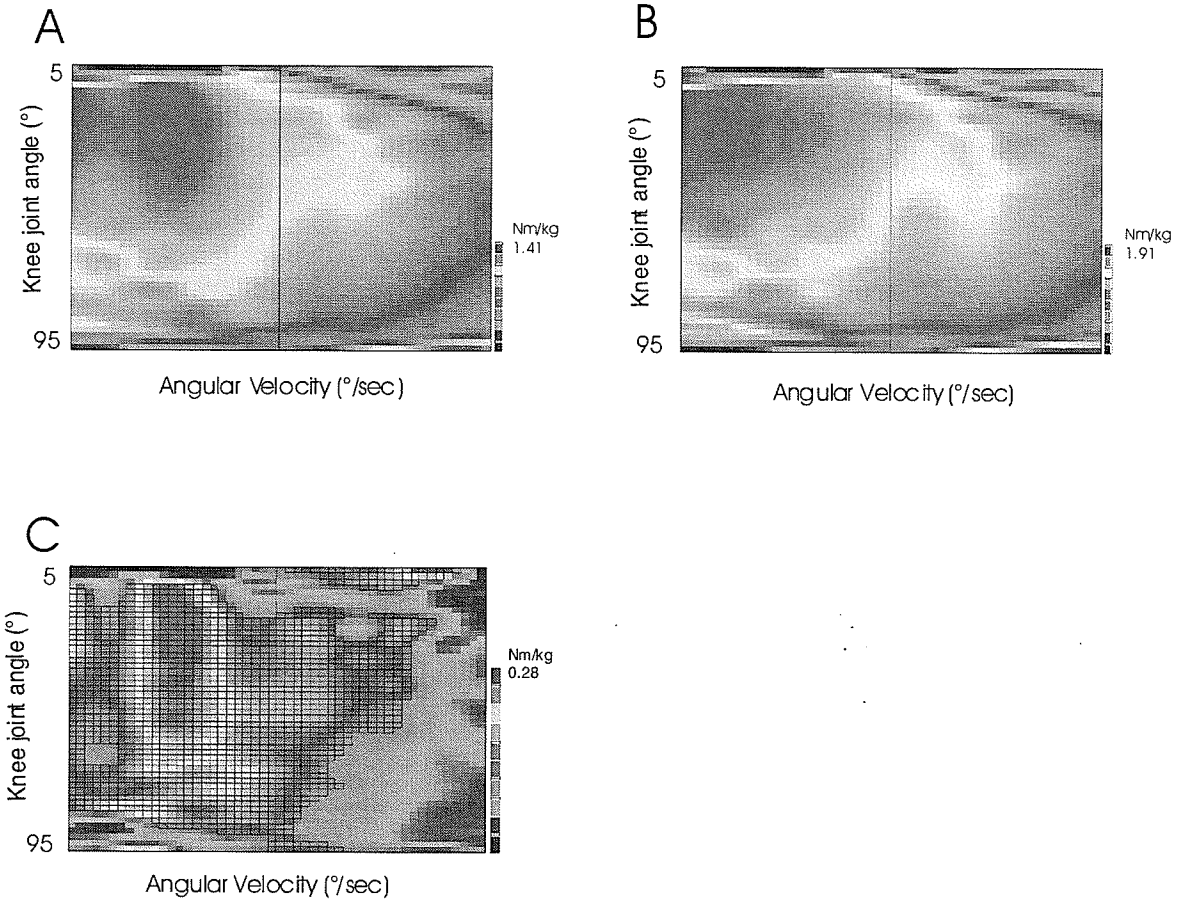


Figure 4. Averaged strength maps of the knee flexors of the contralateral uninjured limb (A), and the ACL reconstructed limb (B). The difference map (C) quantifies the differences between the two limbs and highlights the regional strength deficits that exist after hamstring tendon ACL reconstruction. Hatched areas represent statistically significant differences in strength ($P<0.01$).



Study 4 - Effect Of Fatigue On Knee Proprioception: Implications For Dynamic Stabilization

Investigation into changes in neuromuscular control that occur after injury and surgery revealed a lack of synthesis of some important areas in the study of human motor control. We felt that a review of the relevant areas of fatigue, proprioception and dynamic joint stability would enhance understanding of this complex area. This Manuscript reviews experimental evidence of fatigue-induced changes in knee joint position sense and movement sense or kinaesthesia. The possible physiological mechanisms behind these changes, including the role of joint and muscle receptors in proprioception and neuromuscular control of the knee, the role of fatigue in changes in afferent output from muscle and joint receptors will be discussed. Finally, the implications that alteration in proprioception may have for dynamic stabilization of the knee joint are discussed.

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EFFECT OF FATIGUE ON KNEE PROPRIOCEPTION: IMPLICATIONS FOR DYNAMIC STABILIZATION

Laurie A. Hiemstra, MD

Ian K.Y. Lo, MD

Peter J. Fowler, MD

Please address any correspondence to:

Peter J. Fowler, MD
Fowler · Kennedy Sport Medicine Clinic
3M Centre, The University of Western Ontario
London Ontario N6A 3K7

ABSTRACT

The high incidence of injuries that occur later during a session of sports or recreational activities suggest that fatigue may contribute to altered neuromuscular control of the lower limb and an individual's subsequent altered ability to dynamically stabilize the knee joint. One possible mechanism is a fatigue-mediated alteration in proprioception. This Manuscript reviews experimental evidence of fatigue-induced changes in knee joint position sense and movement sense or kinaesthesia. The possible physiological mechanisms behind these changes, including the role of joint and muscle receptors in proprioception and neuromuscular control of the knee, the role of fatigue in changes in afferent output from muscle and joint receptors will be discussed. Finally, we will explore the implications that alteration in proprioception may have for dynamic stabilization of the knee joint.

INTRODUCTION

With an increase in participation of individuals in sports and recreation, there has been an increase in the incidence of activity-related injuries. It has been estimated that in North America, 13.5 % of the population between the ages of 18 and 35 will suffer a joint injury and that roughly 15 million joint sprains and dislocations occur each year [201]. The knee is the most commonly injured major joint and serious damage to its ligaments is frequent [201, 249]. In the United States alone, approximately 80,000 anterior cruciate ligament one per 3,500 injuries occur annually [75, 141]. Several epidemiological studies have demonstrated that in sports, the most common time for an injury to occur is during the later stage of a game [122, 252, 277, 280, 343]. For example, in a prospective study of ice hockey injuries at the Junior A level, 46.2 % of injuries were sustained in the third period and 46.9% of injuries were sustained in the final 5 minutes of a period [280].

It has been demonstrated in the laboratory that the neuromuscular control of the lower limb is compromised in the fatigued state [189, 383]. Johnston et al [189] found that after fatiguing on a lower limb dynamometer to less than 50% of original strength values, subjects had a significantly decreased ability maintain balance on one or both legs. Wojtys and Huston [383] demonstrated a delay in voluntary muscle reaction time, decreased firing rates of the quadriceps and hamstrings and delayed spinal reflexes after a fatiguing protocol that led to a 50% decrease in work performed on a dynamometer.

The precise physiologic mechanisms behind the fatigue–mediated alterations in neuromuscular control of the knee have yet to be determined. One possible explanation is that neuromuscular fatigue affects knee joint proprioception with subsequent changes in motor control of the lower limb. This effect has also been studied in the shoulder [57,

[276], elbow [243, 316] and lumbar spine [346]. These studies have all demonstrated statistically significant proprioceptive deficits after a fatiguing protocol. However, the clinical significance of these has yet to be determined.

The purpose of this review is to highlight evidence where neuromuscular fatigue has been shown to influence proprioception of the knee joint, to explore the possible mechanisms behind changes in proprioception and neuromuscular control of the knee and to discuss the implications for dynamic stabilization of the lower limb.

PROPRIOCEPTION

Proprioception was originally defined by Sherrington in 1906 as, "the perception of joint and body movement as well as position of the body, or body segments, in space" [322]. It is generally divided into two elements; joint position sense and the sense of limb movement or kinaesthesia. The central nervous system receives input from several sources of stimuli that it integrates to ultimately define joint movement and position. Visual, auditory, vestibular, cutaneous, joint and muscle stimuli provide information to three distinct levels of motor control: the spine, the brain stem and the higher centers, cerebellum, basal ganglia, motor cortex [51, 141, 218-220]. Proprioceptive input may be not necessarily be consciously perceived, for example, the muscle spindle or joint afferent information involved in spinal cord reflexes. For the purposes of this review we will discuss perceived, or conscious, proprioception, which can be measured in human subjects.

Receptors can sense information that is generated from within the organism (interoception) or sense information that arises from external stimulation (exteroception).

Proprioception, in the classic sense, refers to position sense and movement sense (kinaesthesia) arising from interoceptors. In the knee, this is provided primarily by joint mechanoreceptors, Ruffini endings, Pacinian corpuscles, Golgi organs, nociceptors, bare nerve endings, muscle afferents muscle spindles and Golgi tendon organs. The function of proprioceptive sense is to allow feedback for motor control via gamma motor system, thus modulating the stiffness of muscles which control the knee [185].

Studies of conscious proprioception in humans have involved the active or passive reproduction of joint angles joint position sense, as well as the detection of passive motion of the limb kinaesthesia. These tests are conventionally performed while limiting external stimuli such as visual and cutaneous cues. Recently, variables such as movement direction [369], active versus passive positioning [43], pre-test exercise [43], weight bearing [49], and muscle contraction history [99] have all been demonstrated to influence the outcome of these tests.

FATIGUE

Fatigue is defined as the transient inability to maintain power output or force during repeated muscle contractions [17, 131]. Muscle physiology and biochemistry during exercise and fatigue has been well described [297]. Fatigue can occur anywhere along the pathway involved in muscular contraction and can effect changes in cortical input, excitatory drive to the lower motor neuron, motor neuron excitability, transmission at the neuromuscular junction, sarcolemma excitability, excitation-contraction coupling, contractile apparatus, or the metabolic energy supply [38]. Central fatigue, which involves processes above the neuromuscular junction, has been described as "a progressive exercise-induced reduction in voluntary activation of a muscle usually assessed in

maximal voluntary contraction with twitch interpolation [126]. Peripheral fatigue has been described as those mechanisms below the neuromuscular junction involving the actual muscle and contractile mechanisms.

The mechanisms or pathways by which fatigue may influence knee joint proprioception have not been elucidated. Several factors however, have been shown to influence fatigue and must be taken into consideration in any study. The physiological processes that fail may vary depending on the type of exercise performed to induce fatigue. The use of maximal versus sub-maximal contractions, concentric versus eccentric contractions, power versus endurance protocols may all incite different mechanisms of fatigue. Therefore, studies need to clearly define the type of fatiguing protocol, the definition of fatigue and the methods utilized to confirm that it has occurred.

JOINT AND MUSCLE RECEPTORS AND FATIGUE

The role of neuromuscular fatigue in altering input from the knee joint receptors is not clear; however some evidence suggests that increased knee joint laxity may play a role in proprioceptive changes following fatigue. It has been demonstrated that exercise and fatigue increases the laxity of ligaments in the knee [260, 329, 344]. It has also been shown that subjects with increased laxity have poorer proprioception [23, 296]. It is possible that exercise and fatigue-induced increases in knee joint laxity may contribute to alterations in proprioception.

The relative contribution of joint and muscle receptors to measured proprioceptive deficits following fatigue protocols also remains controversial. It is generally accepted that the greatest contribution to position sense and kinaesthesia is from muscle receptors,

primarily muscle spindles and Golgi tendon organs[94, 245]. Since fatigue protocols would presumably affect muscle tissue more than joint tissue, diminished position sense conceptually may be thought to be secondary to loss of muscle receptor input.

The potential direct role of fatigue on muscle receptors has been investigated. Several reports have demonstrated that muscle spindle and Golgi tendon organ activity may be decreased with fatigue. Graham et al [139] in the cat model have demonstrated that large diaphragmatic afferent resting discharge Golgi tendon organ, and muscle spindle was reduced under ischemia, electrically induced fatigue and local acidosis. Lagier-Tessonnier et al [213] further supported these results by demonstrating that muscle spindle and Golgi tendon organ response to high-frequency vibrations were reduced under conditions of muscle acidosis, ischemia and hypoxia in the tibialis anterior muscle. Other investigators have stimulated the cat gastrocnemius muscle until maximal contraction was reduced to 40% of maximal tension force [177]. They demonstrated a decrease in Golgi tendon organ resting discharge, static response, vibration response, peak dynamic response, and dynamic sensitivity. Pedersen et al [275] demonstrated that fatigue of the medial gastrocnemius, resulted in a decrease in the accuracy of information from the muscle spindles in the heteronymous lateral gastrocnemius. Together these studies support the direct role of fatigue in affecting muscle spindles and Golgi tendon organs and imply a role for fatigue in affecting proprioception.

In humans, several studies have demonstrated that small diameter afferents are involved in the reflex inhibition associated with fatigue. Garland demonstrated a similar decline in EMG both before and after blockade of large diameter afferents [128]. Others have shown that in the absence of a chemical stimulus, motor neuron discharge was

unaffected [39, 129]. Studies of fatigued muscles have demonstrated declines in both force production and EMG activity [38], increased muscle firing latency and less efficient neuromuscular processes when compared to non-fatigued muscles [146, 264]. Although more difficult to assess in humans, these studies support the suggestion that afferent feedback from muscle receptors is altered in the fatigued state.

EXPERIMENTAL EVIDENCE OF FATIGUE-INDUCED PROPRIOCEPTIVE DEFICITS

A number of investigators have attempted to determine the effects of neuromuscular fatigue on conscious knee joint proprioception in human subjects. Skinner et al. [328] initially studied knee joint proprioception in a group of eleven young, highly trained, healthy males. They utilized two common measures of proprioception: reproduction of passive knee joint angles in the sitting position, and threshold to detection of passive motion. Measurements were taken before and after a comprehensive lower extremity fatiguing protocol involving an initial sprint of a total of 3 ¾ miles, alternate intervals of 1 mile and ¼ mile distances with a 90 second rest between intervals and two, 2 minute, 15% uphill graded, 7 mph treadmill sessions with a 90 second rest between intervals. Fatigue was confirmed by measuring a 10% reduction in total work output utilizing a standard isokinetic exercise protocol.

These authors demonstrated a statistically significant decrease in the subjects' ability to reproduce knee joint angles after the fatigue protocol from an average of 2.9° to 3.97°. Interestingly, subjects had an improved ability to detect passive motion kinaesthesia following the fatigue protocol from an average of 1.2° to 0.84°, but this did not achieve statistical significance. The authors suggested a role for both muscular and

capsular receptors in knee joint proprioception and proposed that muscle receptors may be more important in joint position sense as the fatigue protocol may have fatigued the muscle receptors more than the joint receptors.

Marks and Quinney [237] studied the active reproduction of knee joint angles in a group of eight young sedentary women after a fatiguing protocol consisting of isokinetic quadriceps contractions. After an initial warm up, fatigue was induced by the performance of twenty consecutive maximum voluntary concentric and eccentric contractions of the quadriceps at 180°/s. Neuromuscular fatigue was confirmed by documenting a 16-23% fatigue index. This study demonstrated no significant difference in joint position sense between the experimental and control groups at baseline and immediately following exercise. It was concluded that fatigue might not induce a knee joint position sense deficit. Interestingly, in contradistinction to this, they found that the accuracy and precision of position sense in both groups improved significantly with repeated testing.

Recently, Lattanzio et al [216] measured active knee joint position sense in sixteen subjects using a weight bearing protocol. Three different cycle ergometer fatigue protocols were used, each standardized to the subject's fitness level. An initial standard ramp protocol 20/25 watts/minute to exhaustion was performed to determine the subjects' VO_{2max}, which was then used to calculate the work rates for the latter two protocols. A continuous test at 80% of VO_{2max} and interval protocol at 120%: 40% VO_{2max} were then performed to exhaustion. Each of these tests was performed on different days, separated by a week. The ability to reproduce knee joint angles was determined in the standing position using an electrogoniometer.

These authors demonstrated a statistically significant decrease in joint position sense of approximately one degree in men for all three fatigue protocols, and in women during the interval and continuous protocols. While these results support those of Skinner et al [328], they raise further questions on the effects of differing fatigue regimens on proprioception, particularly in females.

In summary, these three studies have presented conflicting information regarding changes in proprioception of the knee joint that occur during fatigue. Skinner [328] and Lattanzio [216] demonstrated a statistically significant deficit in joint position sense. The clinical significance of these changes approximately 1° of difference pre and post fatigue remains unclear. Skinner[328] tested detection of passive motion kinaesthesia and demonstrated an improvement in kinesthetic sense after a fatiguing protocol. These studies have, however, raised a number of important issues regarding testing proprioception in the knee joint. Clear definitions of the type of fatigue protocols used in a study need to be provided and compared. Measurement of joint position sense and detection of passive motion may vary depending on testing protocols static vs. dynamic testing, open vs. closed chain testing, weight bearing vs. non-weight bearing. Finally, the differences between male and female, both trained and untrained, need to be evaluated.

MECHANISM OF FATIGUE INDUCED PROPRIOCEPTIVE DEFICITS IN THE KNEE

The physiologic mechanisms behind the changes in conscious knee joint proprioception following neuromuscular fatigue are unclear [215]. It is well accepted that knee joint proprioception is mediated primarily through both joint and muscle receptors

[244, 273, 325, 336]. It is also clear that changes in the afferent input from these receptors have been demonstrated in both animals and humans to cause changes in the neuromuscular control of the lower extremity and that fatigue alters the afferent input from muscle receptors. Finally, we know that neuromuscular fatigue leads to a decrease in the body's ability to control the lower limb. What is not clearly demonstrated is the direct link between fatigue-mediated changes in proprioception and clinically significant changes in ability to dynamically stabilize the limb (Figure 1). If neuromuscular fatigue contributes to changes in position sense and kinaesthesia however, it must be mediated through joint and muscle receptors. The following sections will review joint and muscle receptors and their contribution to both proprioception and dynamic joint stabilization. The influence of fatigue on muscle receptors afferent output and efferent force production will be discussed as it relates to dynamic knee joint stabilization.

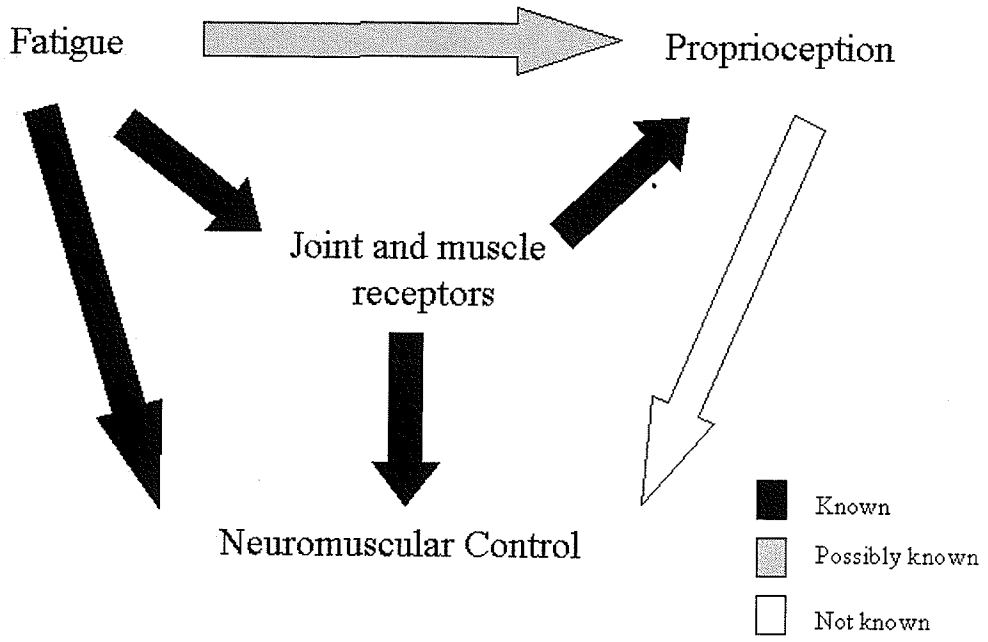

Figure 1. Relationships between proprioception, neuromuscular fatigue and neuromuscular control.

AFFERENT RECEPTORS AND PROPRIOCEPTION:

Afferent joint receptors including Ruffini receptors, Pacinian corpuscles, Golgi tendon organ-like endings, and free nerve endings are present in the anterior cruciate, and peri-articular tissues, especially the posterior capsule ligament [11, 80, 148, 156, 202, 308, 311]. The contribution of these joint receptors to position sense has been widely debated. In animal studies, two main schools of thought exist: one stating that joint receptors signal joint position sense throughout the full range of motion [106, 330] and the other, that joint receptors act as limit detectors only at the ends of range of motion [50, 62, 143, 144]. The theory

that joint receptors play a vital role in joint position sense and kinaesthesia has been strengthened by the demonstration of proprioceptive deficits in models of intra-articular knee pathology. In human research, studies have shown that aged [326], arthritic [206, 238], and post-arthroplasty [327] knees have decreased conscious kinaesthesia and position sense when compared to controls. Knee joint proprioception as it relates to anterior cruciate ligament ACL deficiency and ACL reconstruction has been more widely studied. Krauspe et al [208] found that the afferent neurons from the ACL in the cat displayed increased firing with extension and internal, external rotation of the knee, motions that stressed the ACL. They concluded that mechanoreceptors within the ligament provided information about the tension of the ACL. Consistent with this finding, decreased position sense and kinaesthesia has also been demonstrated in the ACL deficient knee [22, 35, 72, 108, 116, 271], the ACL reconstructed knee [65, 114, 266] and the PCL deficient knee [64, 219, 222, 301]. These studies however, show only small decreases ± 1° in proprioception after intra-articular derangement.

Based on studies using joint de-afferentation and vibration [243] to research muscle spindle output, it is now generally accepted that the greatest contribution to position sense and kinaesthesia is from muscle receptors, particularly the primary endings of muscle spindles [94, 245]. The resting discharge of the muscle spindles provides the brain with information about the position of the limb. Muscle spindles are complex receptors whose output is highly dependent on conditions such as joint angle and contraction history [140].

It is generally thought that both joint receptors and muscle receptors are integral to the ability to sense position and movement of the knee joint. Joint and muscle receptors likely work in a complementary fashion, each modulating the other [24]. Joint

receptors may function to help re-align altered spindle output through their effect on the gamma motor system [125]. Studies in both animals and humans suggest that situations in which the afferent output from the joint and muscle receptors is altered, can cause a change in the ability of the knee to determine both position sense and kinaesthesia.

IMPLICATIONS FOR DYNAMIC KNEE STABILIZATION

Although the extent to which joint receptors contribute to the overall ability to determine joint position sense and kinaesthesia and how much fatigue alters the output from joint receptors is unclear, in both human and animal studies the influence of afferent information from the knee joint in the neuromuscular control of the limb has been well demonstrated. Johansson postulated that the joint receptors provide information which help to modify muscle tone about the knee through the gamma motor system [185].

The ability of joint afferent information to influence motor control of the lower extremity has been well studied in animal models. In the 1970's and 1980's, a number of studies in cats demonstrated that stimulation of knee joint afferents led to a modulation in the efferent output to the muscles that control the knee [24, 111, 227, 304, 306]. Other studies have found changes in the firing patterns of joint afferents with certain movements after transection of the ACL [135, 203]. With convincing evidence that activation of joint afferents influences the firing activity of the knee extensors and knee flexors, several authors studied the effect of stimulation of the anterior cruciate ligament and found increased hamstring EMG and inhibited quadriceps EMG [153, 249, 290, 335]. Abelew et al [4] studied interjoint coordination in cats after inducing loss of spindle input. They found that coordination between the lower limbs was disrupted with loss of feedback from the

muscle spindles. These animal studies clearly show that both joint and muscle afferent information is involved in the activation of the muscles that control the knee joint.

In humans, Solomonow et al [335] demonstrated that when the ACL is placed under high loads, there is a reflex contraction of the hamstrings musculature. These findings have been supported by Sjolander [323] who observed that direct deformation of the anterior cruciate ligament intra-operatively elicited activity in the neural arcs of the muscles spindles in the hamstring muscles, presumably to decrease anterior translation of the knee. Recently, Dyhre-Poulsen [91] directly stimulated the ACL in humans and recorded contraction of the knee flexors and extensors. Although the ligamentomuscular protective reflex presumably functions during excessive high loads, other reflexes including those with receptors in the joint capsule and muscle may be relevant at lower loads [184, 336].

Beard et al [26] studied the effects of ACL deficiency on the latency of reflex hamstring contraction in 30 patients. Using a knee displacement apparatus, the reflex hamstring contraction latency was measured using surface electromyographic electrodes following the application of a 100 N postero-anterior shear force. This study demonstrated a significant increase in hamstring latency in anterior cruciate ligament deficient knees when compared to the contralateral side and to normal controls. This correlated to functional instability r=0.62 but not to KT1000 objective instability r=0.26. This suggests that the protective ligamento-muscular reflex is disrupted with anterior cruciate ligament injury. Interestingly, in a second study [25], these investigators demonstrated that a specific "proprioceptive" rehabilitation program could improve this reflex and result in functional gains. These findings raise the question of the role of

rehabilitation in limiting proprioception deficits after ACL injury and reconstruction [113, 179].

From both animal and human studies, it seems clear that the afferent input originating from the knee joint and muscles about the knee does exert an influence in the activation of the muscles that control the knee motion and thus may contribute to dynamic stability of the knee joint. Further studies to delineate the functional implications of proprioceptive deficits in the knee joint will allow a greater understanding of the interactions between fatigue, proprioception and neuromuscular control. This may eventually support changes in clinical practice for the prevention, treatment and rehabilitation of sport related injuries.

SUMMARY

Our review has highlighted experimental evidence that fatigue-mediated changes in joint position sense of the human knee occur although the clinical significance of this is unclear. Changes in kinaesthesia of the knee after fatigue have not been identified. The physiologic mechanisms behind the changes in conscious knee joint proprioception following neuromuscular fatigue are unclear. It has been well established that proprioceptive sense is mediated through joint and muscle receptors. There is both animal and human evidence that joint and muscle receptors mediate changes in the efferent output of the muscles that control the knee, potentially providing stability and stiffness to the knee. In both the state of altered afferent input as well as during fatigue, the neuromuscular control of the limb is compromised, which may lead to an inability to dynamically stabilize the knee. The influence of fatigue induced knee joint laxity on

proprioception needs to be more clearly defined. Further study is needed to determine if fatigue truly mediates changes in proprioceptive sense and what implications this may have on the ability to dynamically stabilize the knee joint.

Study 5 – The Development of Contralateral Leg Strength Deficits after Acute Anterior Cruciate Ligament Injury

The purpose of this study is to determine the effect of an acute ACL injury on the contralateral limb by documenting the time course of strength changes in the contralateral uninjured limb. This will aid in the determination of the etiology (neural or muscular) of the contralateral leg strength deficits.

This manuscript is in its final editing stages for submission to the *Journal of Clinical Biomechanics* March 2003. Ethics approval for this study has been granted by the University of Manitoba (E96:246).

A review of literature includes a discussion of de-training including immobilization, bed rest, and lower limb suspension and training-detraining.

De-training

One possible etiology of strength deficits in the contralateral uninjured limb is the period of reduced activity following ACL injury and surgery combined with varied or incomplete rehabilitation. After an acute injury, this active population undergoes a period of reduced activity; many are placed in a knee immobilizer for a period of time as well as given crutches if needed. The effects of immobilization and reduced activity on skeletal muscle have been studied in detail. There are many models of immobilization/detraining. These vary from microgravity environment, to bed rest, to cast immobilization to a training-detraining model. We believe that in this model of ACL injury and surgery, the disuse that occurs is most closely related to a model of training-detraining [391].

Bed rest

Dudley et al [86] studied knee flexor and extensor moments after 30 days of bed rest. They demonstrated an average 19% knee extensor strength loss and a 6% (not

statistically significant) knee flexor strength loss. This greater knee extensor than knee flexor loss was independent of contraction type and angular velocity. It has been demonstrated that 20 days of bed rest induced a 7-10% atrophy of the lower limb muscles [9]. Later studies demonstrated that isometric exercises prevented atrophy of the knee extensors but not the knee flexors or the plantar flexors of the calf [8]. Finally, Akima et al [7] demonstrated that a dynamic leg press, which used all muscle groups with both concentric and eccentric contraction types, prevented any atrophy of the knee extensors, partially of the knee flexors and not of the plantar flexors. This suggests that decreased activity may have varying degrees of influence on the lower limb muscles after an injury based on the amount of work performed by each muscle group.

Immobilization

Short term immobilization of an injured knee in a cast for 72 hours has been shown to have variable effects on muscle fibre size based upon muscle biopsy derived data [224]. In healthy subjects, immobilization of the knee for 21 days caused a reduction in concentric, eccentric and isometric strength by 47% [168]. Muscle fibre atrophy was only 11% after 3 weeks, the discrepancy between atrophy and strength loss is consistent with the notion that neural factors rather than muscular factors or muscular atrophy, were principally responsible for the strength losses [168]. Veldhuizen et al [363] immobilized a healthy limb in a cast for 4 weeks. They demonstrated a 53% decrease in knee extensor strength (at 60°/s), a 26% decrease in knee flexor strength (at 60°/s), and a 21 % decrease in cross-sectional area of the quadriceps. Labarque et al [212] immobilized healthy knees for 2 weeks and demonstrated a 30% decrease in knee extensor moment at 60°/s and an 11% decrease in knee flexor moment at 60°/s (KE>KF). Thom et al

[348]immobilized the knee in 60° of flexion for 10 days and noted an 11.8% decrease in cross sectional area and a 41.6% decrease in knee extensor strength. They also measured a 19.8% decrease in sarcoplasmic reticulum uptake rate at 10 days that was evident already at 3 days of immobilization[348]. Immobilization of the lower limb for longer periods of time affects both the architecture as well as the neural activation of the muscle. Five weeks of casting demonstrated that the thenar muscles exhibited a 57% decrease in maximum voluntary contraction, a 29% decrease in motor unit activation and a 45% decrease in reflex potentiation [302].

Lower Limb Suspension

Berg et al [31] performed a unique study where the lower limb was suspended by a harness so that range of motion of the hip and knee was allowed but no weight bearing was possible. After 4 weeks, they found a 7% decrease in cross sectional area of the thigh but no change in the contralateral limb. The knee extensors appeared to atrophy twice as much as the flexors. Strength testing revealed that there was a 17% decrease in knee extensor peak moment measurements of the suspended leg. A similar study [32]demonstrated decreased strength measurements after 10 days of unloading but no change in EMG activity, although the biarticular rectus femoris readings were included in the averaging. Further studies on short term (14 days) unilateral lower limb suspension demonstrated that significant decreases in KE and KF peak moment and work measurements as well as EMG activity. There was no evidence of muscle atrophy, however increased plasma cortisol measurements suggested a biochemical environment compatible with muscle atrophy [82]. Schulze et al [310] looked at 21 days of lower extremity suspension. In the non-exercising group, they found a 7% decrease in cross sectional area

and 17% decrease in MVC. Knee extensor concentric and eccentric strength decreased by 19%. These strength deficits were associated with a decrease in EMG activity in the knee extensors. There were no strength deficits if resistance training was introduced to the suspended limb. Hather et al [155] had subjects non-weight bearing for 6 weeks with movement allowed at the hip, knee and ankle. They demonstrated a 12% decrease in CSA according to magnetic resonance imaging (MRI). The knee extensors demonstrated twice the strength decrease as the knee flexors. These findings are consistent with the theory that strength deficits seen in the short term, after an acute injury, are primarily neural in origin.

Training – De-Training

The studies that have been performed looking at reduced levels of activity have mainly concentrated on training and detraining of healthy individuals. Mujika defines detraining as "the partial or complete loss of training-induced anatomical, physiological and performance adaptations, as a consequence of training reduction or cessation" [32, 254]. Large decreases in strength and EMG measurements have been demonstrated in healthy subjects after a period of training followed by a period of detraining [68, 147, 174, 341]. Strength training prior to detraining, especially combined concentric and eccentric more than concentric training alone strength training, appears to protect the muscle and slow the strength loss by more long lasting neural adaptation [68]. Hortobagyi [169] demonstrated that after 12 days of detraining of power athletes, there was a greater decrease in eccentric isokinetic strength vs. concentric. The knee extensors were more involved than the knee flexors (isolated KE eccentric strength deficits).

These studies demonstrate that there is a loss of strength following reduced activity. Most of the studies were performed on healthy individuals. There seems to be consensus that the loss in strength is the result of both peripheral controlled disuse atrophy as well as altered central neural regulation [85, 302]. These changes include alterations in the intrinsic properties of the motoneurone [123], the efficacy of synaptic transmission onto the motoneurone [124, 233] and descending control [392]. How the change in neural regulation is different after injury in addition to immobilization has not been fully elucidated. Seki et al [313] investigated the etiology of strength decreases after immobilization of the first dorsal interosseous muscle in humans. They demonstrated the there was a restriction of motoneurone firing rates and an enhancement of the voluntary force exerted when the mean firing rate was low.

The effect of immobilization or reduced activity on the contralateral leg to an injury has yet to be determined. Most studies use the contralateral leg as the control leg for both research and rehabilitation purposes. In our acute ACL injury model, one would expect a dramatic reduction in activity of the muscles of the injured leg for a period of about one week followed by a progressive increase in use of the leg. The time to return to pre-injury activity is not known; in fact it is not known whether the activity level of the injured leg ever returns to that of the pre-injury state. In this previously active population, this reduced period of activity after an injury follows most closely the training-detraining model outline in the previous literature review. This model does not take into account, however the addition of pain and knee joint effusion that would be present after an acute ACL rupture. How this nociceptive afferent input will effect the development of strength deficits in the contralateral limb has yet to be determined.

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The Time Course of the Development of Contralateral Leg Strength Deficits after Acute Anterior Cruciate Ligament Injury

Laurie A Hiemstra, MD, FRCS(C)

Dean J. Kriellaars, PhD

Sport Medicine Centre

Faculty of Medicine, University of Calgary

Calgary, Alberta T2N 1N4

School of Medical Rehabilitation

Faculty of Medicine, University of Manitoba

Winnipeg, Manitoba, R3E 0W3

Abstract

Strength deficits have been demonstrated in the reconstructed and contralateral knee flexors and extensors of patients after hamstring tendon anterior cruciate ligament (ACL) reconstruction and subsequent rehabilitation. The origin of these deficits has been attributed to be secondary to either de-training/de-conditioning or crossover inhibition secondary to nociception from the acutely injured knee. The purpose of this study was to document the time course of the development of strength deficits in the knee flexors and knee extensors of the contralateral uninjured knee after an acute ACL injury. Knowledge of the timing of these deficits will provide information regarding the neural or muscular etiology of these documented contralateral strength deficits. This may have important clinical implication for interventional rehabilitation protocols. We hypothesize that there would be an immediate strength loss (compared to control) consistent with a potent neurally mediated cross-over inhibition of motoneurone activation. We hypothesized that the initial strength loss can likely be attributed to a neural mechanism and that this strength deficit resolves as the neural inhibitory mechanism reduces in the presence of the onset of de-conditioning atrophy.

Comprehensive isovelocity strength analysis (5-95°, 5 speeds, concentric and eccentric) was performed of the knee flexors and extensors of the contralateral uninjured leg as soon as possible after ACL rupture, and at 6, 12, and 18 weeks post injury and compared to control. Physical examination and a Mohtadi Quality of Life Questionnaire were completed at each visit and correlated to strength measurements.

Results demonstrate contralateral uninjured knee extensor strength deficits of up to 39% compared to control at average 10.6 days (range 4-22) after injury ($p<0.05$).

These deficits improve by 9% but at 18 weeks the knee extensors still have a 30% strength deficit compared to control. Knee extensor deficits are consistently greater for concentric contractions than for eccentric contractions (average 5.0%). There was a contralateral uninjured knee flexor strength deficit of up to 27% initially after injury ($p<0.05$) that improved to 21% at 18 weeks. Knee flexor strength deficits are greater for eccentric contractions than for concentric contractions (11.5%).

Strength deficits in the uninjured contralateral leg after an acute knee injury are consistent with an immediate neural effect as well as subsequent de-conditioning. These findings give insight into the physiological mechanisms behind neuromuscular changes that occur after injury and will have significant implications for rehabilitation after an acute knee injury.

Introduction

Anterior cruciate ligament (ACL) reconstruction is a common orthopaedic procedure aimed at restoring antero-posterior stability to the knee joint [110]. Comprehensive strength analysis (90° range of motion, five speeds, concentric and eccentric contractions) of the knee flexors and extensors after patellar tendon and hamstring tendon ACL reconstruction has identified strength deficits when compared to control[164]. The magnitude of this deficit was, as expected, greater than has previously reported in the literature that used the contralateral leg as a control leg. It was postulated that ACL injury, reconstruction, and subsequent rehabilitation affected the strength of the contralateral uninjured leg. In a subsequent study [163], strength of the knee flexors and extensors of both the ACL reconstructed leg were compared to the contralateral uninjured leg as well as to a control group of age and activity matched peers. It was demonstrated a statistically significant reduction in knee extensor and flexor strength exist in both the ACL reconstructed and contralateral leg when compared to control.

We postulate that the strength deficits of the knee flexors and extensors that have been demonstrated in the contralateral uninjured limb after ACL reconstruction and subsequent rehabilitation are secondary to either cross-over nociceptive inhibition or to de-conditioning. In order to sort out these possible etiologies and to identify physiologically appropriate interventions, it is first necessary to identify the time course of strength changes after ACL rupture.

Methods

Nine subjects who presented with an isolated acute knee injury consistent with an ACL tear were recruited from the Fowler Kennedy Sport Medicine Centre in London, Ontario, Canada. The average age was 31.3 ±11.6 years. The average mass was 76.8 ± 18.3 kg. There were five men and four women. Seven subjects injured their dominant limb, two subjects injured their non-dominant. Three patients had surgery prior to finishing the 18 weeks follow-up but continued to participate in the study. These were managed on an intent to treat basis. Individual inspection of their strength data following their surgery revealed consistent curves to the rest of the group. ACL injury was subsequently confirmed for all subjects by either arthroscopy or MRI. Written informed consent was obtained from all subjects. This study received ethics approval by the Review Board for Health Sciences Research Involving Human Subjects at the University of Western Ontario. These were compared to a group of 12 subjects (26.6 ±6.7 years, 69.8 ± 13.8 kg) who were greater than one year post hamstring tendon ACL reconstruction and a control group (n=30, 26.3±4.8 years, 78.8±13.1 kg) with no knee injury.

Subjects were evaluated as soon as possible after injury (mean 10.6 days), at 6, 12 and 18 weeks post injury (Table 3). Each evaluation consisted of a physical examination of both the injured and uninjured legs to confirm ACL. Subjects completed the Quality of Life Questionnaire for ACL Deficiency [251] at each visit. A 10 cm visual analogue scale (VAS) was completed for pain intensity in both the injured and uninjured legs for before and during the strength testing.

Strength testing of the knee extensors and flexors of the contralateral uninjured limb was performed on a dynamometer (Kin-Com 500H dynamometer, Chattecx Corporation, Hixson, TN). Precise protocols have been previously published [164] and include 5-95° range of motion, five angular (50,100,150,200,250°/s), concentric and eccentric contractions. The resultant knee joint moment calculated by the dynamometer was corrected for the moment of the weight of the leg and foot [3, 107, 376, 380]. All resultant joint moment were normalized to body mass (Nm/kg) which has been validated for multiple velocity and isometric moment data arising from young, active individuals[59, 165, 382].

Results

Knee Extensor Strength Testing

All groups and strength tests conformed in shape to reported moment-angular velocity curves [368] for voluntary maximal contractions (Figure 1). Comparison of knee extensor strength between the contralateral limb of the acutely injured ACL group to the contralateral limb of the ACL reconstructed group demonstrated strength deficits of 19% averaged across speeds present at the initial testing session (average 10.6 days, range 4-22 days). These strength deficits were larger for concentric contractions (22%) than for eccentric contractions (16%). Over the testing period this knee extensor strength deficit decreased to 9.5% compared to the contralateral limb of the ACL reconstructed group at the 18 week point ($P<0.01$).

Comparison of knee extensor strength between the contralateral limb of the acutely injured ACL group to the uninjured control group demonstrated strength deficits of 35.7% averaged across speeds present at the initial testing session. These deficits were

larger for concentric contractions (39.1%) than for eccentric contractions (32.2%). Over the testing period, this knee extensor strength deficit decreased to 28.1% (from 35.7%) compared to the control group at the 18 week point (P<0.01).

Knee Flexor Strength Testing (Figure 2)

All groups and strength tests conformed to known moment-angular velocity curves for voluntary maximal contractions. Comparison of knee flexor strength between the contralateral limb of the acutely injured ACL group to the contralateral limb of the ACL reconstructed group demonstrated strength deficits of 11.7% averaged across speeds present at the initial testing session (average 10.6 days, range 4-22 days). These strength deficits were larger for eccentric contractions (13.9%) than for concentric contractions (9.5%). Over the testing period this knee flexor strength deficit decreased to 4.0% (from 11.7%) compared to the contralateral limb of the ACL reconstructed group at the 18 week point (P<0.01).

Comparison of knee flexor strength between the contralateral limb of the acutely injured ACL group to the uninjured control group demonstrated strength deficits of 21.9% averaged across speeds present at the initial testing session. These deficits were larger for eccentric contractions (27.1%) than for concentric contractions (16.8%). Over the testing period, this knee extensor strength deficit decreased to 15.1% compared to the control group at the 18 week point (p<0.01).

Quality of Life

A Quality of life questionnaires[251] were completed at each visit. There was a high correlation between QOL scores and time since injury (r=0.85).

Discussion

This study has demonstrated that large strength deficits are present in the knee flexors and extensors of the contralateral uninjured limb early after an acute ACL injury. The strength test is a test of the neuromuscular system, therefore changes in the moment generating capacity of the knee flexors and extensors must have a muscular or neural etiology. The most plausible etiology of strength deficits demonstrated in the first 10 days after ACL injury is nociceptive inhibition of motoneurone output to the contralateral limb.

An acute rupture of the ACL usually results in significant pain and a large hemarthrosis [217, 231] in the injured knee. Ipsilateral quadriceps inhibition has been demonstrated in humans after a knee effusion [337, 342] and a knee injury[162 315, 332, 356]. Studies performed on cats demonstrate that that increased firing of articular Group III and Group IV afferents occurs in response to experimentally induced inflammation of the knee joint [305]. In addition to increased firing of the Group III and IV afferents, the receptive field of the afferent neurons increases in response to the inflammation[305]. This increase in sensitivity occurs within hours of the experimentally induced inflammation[307]. This may be mediated via chemo sensitive receptors to inflammatory mediators such as bradykinin, serotonin and prostaglandins. The sensitization was inhibited by anti-inflammatory medications [159], capsaicin [158] and opiates [299].

Contralateral changes to an ipsilateral event have been described in both the strength and neuroscience literature. In the strength training literature, effects on the contralateral limb have been well described. Strength training of one limb has shown to cause strength gains in the contralateral untrained limb for isometric [67, 317, 372], concentric

[172, 174, 193, 370], eccentric [173, 371] and stimulated contractions [53, 171]. Contralateral strength gains are greater for eccentric contractions than for concentric contractions [170]. In the neuroscience literature, cross-over or contralateral effects have also been demonstrated in animals with movement of the contralateral limb [13-15]. Other researchers have demonstrated H-reflex facilitation in response to cutaneous stimulation of the contralateral leg [278]. Alteration of the soleus H- reflex has been demonstrated in the contralateral limb of humans [61, 69, 247, 294]. Studies of muscle inhibition after injury have demonstrated that contralateral effects to the uninjured limb are present [162, 356]. In ACL deficient subjects, decreased muscle activity in both the ACL deficient and the contralateral limb has been demonstrated [362, 382]

Specific activation of nociceptors has been demonstrated to have contralateral effects. Inflammation has been shown to have effects on the contralateral limb [40, 41]. With noxious stimulus of peripheral tissues, substances are released in the ipsilateral and contralateral dorsal horns in the spinal cord [87 334]. Nociceptive pathways have been demonstrated in both the spinal cord (ipsilateral and contralateral side) with connections to higher centres such as the hypothalamus [132]. Acute unilateral inflammation increases the effectiveness of tonic descending inhibition input from regions of the contralateral leg [60, 262, 303]. With contralateral effects well demonstrated in both animal and human studies, afferent feedback from the injured knee could affect the strength of the contralateral limb via contralateral connections in the spinal cord.

After an acute ACL injury, treatment varies however common management would include immobilization of the injured leg and decreased weight bearing for comfort purposes followed by progressive range of motion and weight bearing. Many are

unable to return to their previous levels of activity because of ongoing giving way of the knee which eventually requires surgical intervention. For this active population, the time from injury until surgical intervention represents a period of reduced activity which corresponds most closely to a model of training-detraining with the addition of an injury stimulus.

Large decreases in knee extensor strength and EMG have been demonstrated in healthy subjects after a period of training followed by a period of detraining [68, 73, 147, 174, 259, 341]. Studies using the training-detraining paradigm have found decreases in strength after longer periods of detraining of 12 weeks [147, 174] Shorter terms of unilateral limb suspension have demonstrated decrease in cross-sectional area and a decrease in strength of the suspended but not the contralateral limb [31] The extensors appeared to atrophy twice as much as the flexors. In this active population, the period of relatively decreased activity secondary to an unstable ACL deficient knee may contribute to a decrease in the strength of the contralateral uninjured leg. There seems to be consensus that the loss in strength is the result of both peripheral controlled disuse atrophy as well as altered central neural regulation [85, 302]. How the change in neural regulation is different after injury in addition to immobilization has not been fully elucidated. For shorter term immobilization, neural factors have been thought to mediate loss in strength [82]

Given this, it is reasonable to postulate that strength loss, like strength gains arise from both neural and muscular factors. As suggested by Moritani [253] the initial strength gains are generally now accepted to be mainly neural in etiology. So with de-conditioning, we hypothesize that strength loss early is neural in nature and may be enhanced by nociceptive information from an injury.

This study also demonstrated specificity of strength loss with regards to contraction type and muscle group. The knee extensors had greater concentric strength loss than eccentric strength loss while the knee flexors had greater eccentric strength loss. In this ACL deficient population return of eccentric knee flexion was clearly seen over the 16 weeks post injury. This may be due to the role of the knee flexors as an ACL agonist, protecting against anterior translation of the tibia in the presence of ACL deficiency. The etiology of differential response of concentric and eccentric contraction strength to ACL injury requires further investigation.

Note that even at 18 weeks, the uninjured contralateral leg still had a strength deficit when compared to the contralateral leg of the group which has undergone ACL reconstruction. One would speculate that a longer follow-up would demonstrate that this deficit would eventually resolve. Interestingly however, rehabilitation after ACL reconstruction did not rectify the strength deficits in the contralateral leg. Further investigation into the ability to prevent or rectify these strength deficits is necessary. It certainly appears however that current rehabilitation programs do not put emphasis on the contralateral leg strength. The large strength deficits when compared to control suggest that these patients have not returned their strength to that of their peers which may have implications for functional outcome and return to the same level of activity. In addition, this emphasizes the fact that caution should be taken when using the contralateral leg as a control.

Figure 1. Knee extensor moment for the contralateral limb of the acute ACL group, the ACL reconstructed group, and the control group. Positive angular velocities correspond to concentric contractions and negative angular velocities correspond to eccentric contractions. Moments are normalized for body mass. Standard error bars are excluded for clarity.

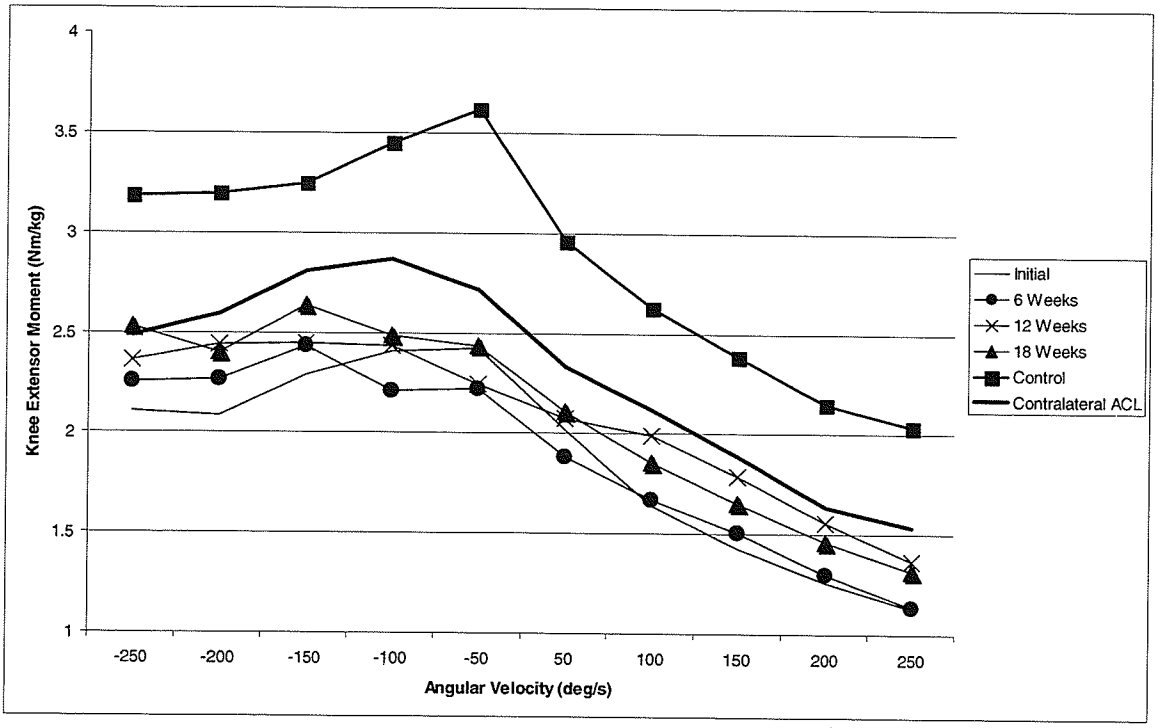

Table 1. Knee extensor strength deficit as a percentage of the control and ACL reconstructed groups.

	% (SD) Deficit Control	Concentric % (SD) Deficit Control	Eccentric % (SD) Deficit Control	% (SD) Deficit to ACL Contralateral	Concentric % (SD) Deficit ACL Contralateral	Eccentric % (SD) Deficit ACL Contralateral
Knee Extensor						
Initial	35.7 (5.1)	39.1 (4.8)	32.2 (2.3)	19.0 (5.1)	22.0 (5.0)	16.0 (3.4)
6 weeks	35.1 (5.7)	38.6 (3.4)	31.5 (5.5)	18.3 (5.1)	21.4 (2.6)	15.3 (5.3)
12 weeks	28.1 (4.5)	27.9 (3.5)	28.3 (5.8)	9.4 (4.6)	7.6 (3.0)	11.3 (5.5)
18 weeks	28.1 (5.3)	31.2 (2.7)	24.9 (5.7)	9.5 (4.7)	11.9 (1.9)	7.1 (5.7)

Figure 2. Knee flexor moment for the contralateral limb of the acute ACL group, the ACL reconstructed group, and the control group. Positive angular velocities correspond to concentric contractions and negative angular velocities correspond to eccentric contractions. Moments are normalized for mass.

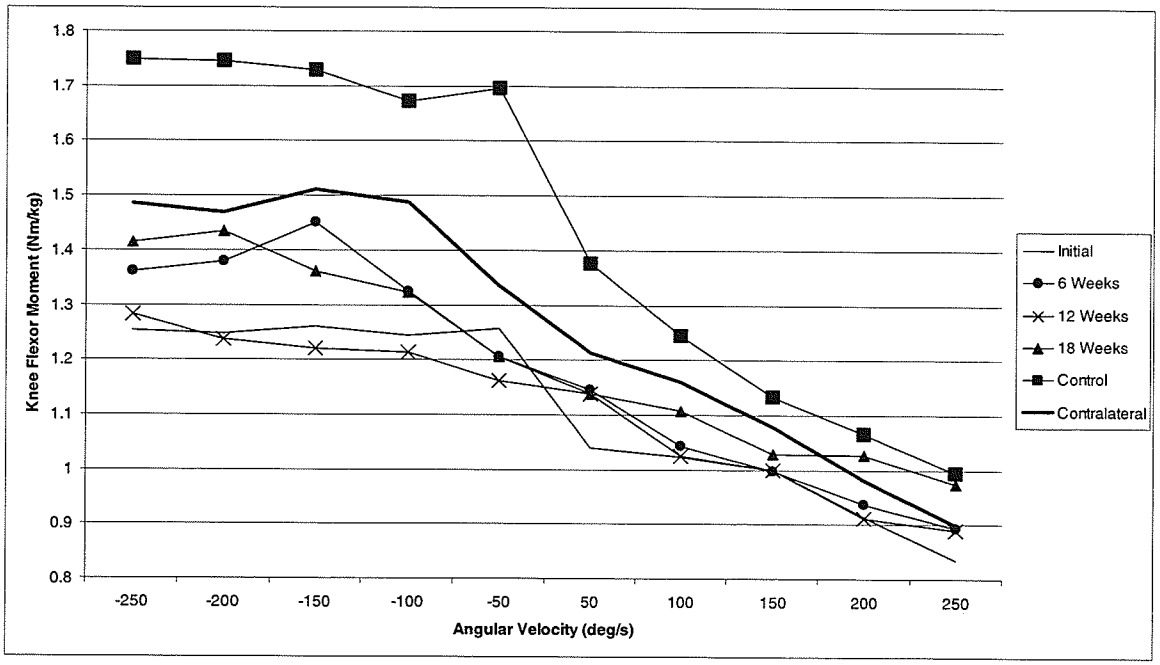

Table 2. Knee flexor strength deficit as a percentage of the control and ACL reconstructed groups.

	% (SD) Deficit Control	Concentric % (SD) Deficit Control	Eccentric % (SD) Deficit Control	% (SD) Deficit to ACL Contralateral	Concentric % (SD) Deficit ACL Contralateral	Eccentric % (SD) Deficit ACL Contralateral
Knee Flexors						
Initial	21.9 (6.3)	16.8 (4.8)	27.1 (1.3)	11.7 (4.4)	9.5 (3.4)	13.9 (4.5)
6 weeks	17.6 (5.7)	13.4 (2.9)	21.8 (4.6)	6.8 (3.2)	5.7 (3.5)	7.8 (2.8)
12 weeks	21.6 (8.0)	14.3 (3.2)	28.8 (1.9)	11.4 (5.8)	6.7 (3.9)	16.0 (2.8)
18 weeks	15.1 (8.5)	8.6 (6.1)	21.6 (4.3)	4.0 (6.2)	0.5 (4.5)	7.6 (3.8)

Table 3. Time from injury to testing sessions. QOL – Quality of Life with ACL deficiency questionnaire average scores.

	Time since injury – days (SD)	QOL Average (SD)
Initial Visit	10.6 (6.7)	31.2 (3.8)
Second Visit	45.4 (6.6)	29.3 (6.3)
Third Visit	91 (4.4)	37.5 (2.8)
Fourth Visit	132.4 (4.7)	42.7 (1.7)

Study 6 – Hip Strength Following Hamstring Tendon Anterior Cruciate Ligament Reconstruction

This study was designed to evaluate hip extensor and hip adductor strength of subjects who have had a hamstring tendon ACL reconstruction and subsequent rehabilitation. We hope to determine whether an insult to a bi-articular muscle, such as the semitendinosus and gracilis has direct (muscular) or indirect (neural) effects at the hip joint.

This Manuscript has been submitted for publication to the *Clinical Journal of Sport Medicine*. Ethics approval for this study has been granted by the University of Western Ontario 07856E. A review of literature includes a discussion of bi-articular muscles.

Bi-articular Muscles

Both the semitendinosus and gracilis muscles are bi-articular in that they cross both the hip and the knee joint. The effect of contraction of two-joint muscles is never limited to one joint [120]. Muscle tendon complexes of bi-articular muscles change differently at different joints and therefore may have more action at a particular joint [365]. They will, therefore, have a differential functional role in the control of multi-joint movements.

The function of bi-articular muscles has been widely researched. The general consensus is that bi-articular muscles aid in the transfer of force through the body segments during complex movements [288, 360]. van Ingen Schenau [361] proposed that mono-articular muscles are primarily responsible for the generation of force and work and that

bi-articular muscles function more to control the direction of external forces by regulating the distribution of the net moments that act across the joints [42, 83, 180, 361]. This theory is supported by the finding of more complex neural circuitry to motoneurones innervating bi-articular muscles [283]. Thus, force generation during dynamic movements is more a function of bi-articular muscles while during isometric movement the force is generated more by the mono-articular muscles [359]. Some have reported that the activation of two-joint muscles is dependent on the demands of the two joints that the muscle crosses [120, 286]. Regulation of two-joint muscles also may come from central pattern generators which can be modified by afferent input [181, 182]. Others have suggested an energy saving hypothesis in that motor control strategies governing one and two-joint muscles function to minimize energy expenditure [285].

The neural coordination strategies governing control of mono-articular and bi-articular muscles during different motor tasks continues to be widely debated [160, 285]. It has been demonstrated that mono-articular and bi-articular muscles behave differently during fatiguing tasks [93]. Studies during walking and running suggest that the control of bi-articular muscles activation appears to be independent of contraction type (concentric, eccentric and isometric) [180, 286, 287]. Mono-articular muscles exert force only in the phase in which these muscles shorten, whereas this appears not to be the case for the bi-articular muscles [42, 361].

There have been many studies looking at the interactions between the hip and knee in particular. It has been demonstrated that there are obvious interactions between moment generating capacity of the hip and knee based on the state of the hip or knee angles [152, 274]. Other studies however, have found that knee angle had no effect on hip

extensor strength [261]. Hip angle has been shown to have implications for the generation of internal tibial rotation, with increased rotational strength generated at large hip flexion angles [269].

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Hip Extensor and Adductor Strength after Hamstring Tendon Anterior Cruciate Ligament Reconstruction

Laurie A Hiemstra, MD, FRCSC

Wade T. Gofton, MD

Dean J. Kriellaars, PhD

Sport Medicine Centre
Faculty of Medicine, University of Calgary
Calgary, Alberta T2N 1N4

Fowler Kennedy Sport Medicine Centre
University of Western Ontario
London, ON, Canada

School of Medical Rehabilitation
Faculty of Medicine, University of Manitoba
Winnipeg, Manitoba, R3E 0W3

ABSTRACT

The purpose of this study was to determine the effects of semitendinosus and gracilis tendon harvest for anterior cruciate ligament (ACL) reconstruction on the resultant joint moment generated by the hip extensors and hip adductors.

Fifteen subjects greater than one-year post-op ACL reconstruction with semitendinosus and gracilis tendons underwent strength testing of the hip extensors and hip adductors of the operated limb and contralateral limb. In addition to physical examination, evaluation using a modified Mohtadi Quality of Life Questionnaire was completed. The subjects were compared to fifteen age, sex and activity matched controls with no lower limb injury. Isokinetic hip extensors strength tests were performed at 50 and 150 °/s, and isometric hip adductors strength tests were performed at 15 and 30-° of adduction.

Isometric resultant joint moment of the hip adductors deficits of up to 28% are demonstrated in the ACL reconstructed subjects compared to control. For hip extension, there is a significant difference in strength between dominant and non-dominant hip extensors ($P<0.05$) in the control subjects. In the ACL reconstructed subjects, there is a trend toward increased eccentric hip extensor strength that approaches but does not reach significance.

The identification of hip adductor strength deficits after hamstring harvest for ACL reconstruction may have important implications for both graft harvest site selection, as well as post-operative rehabilitation protocols. Possible increases in eccentric hip extensor strength may contribute to increased lower limb strength imbalances given that

knee strength deficits have been identified. This may have implications for graft protection.

INTRODUCTION

Anterior cruciate ligament (ACL) injuries of the knee are common in active individuals and the current standard of care for symptomatic patients is the arthroscopically-assisted intra-articular autograft ACL reconstruction [110]. The most common autograft selections for ACL reconstruction include the central third of the patellar tendon and the quadruple stranded semitendinosus and gracilis tendons [248]. Donor site morbidity for the patellar tendon autograft for ACL reconstruction has been well documented and includes anterior knee pain, knee extensor strength deficits, and arthrofibrosis[6, 19, 300]. Documented morbidity for the hamstring tendon graft includes damage to the saphenous nerve and knee flexor strength deficits [195 34, 164, 234, 258]. These knee flexor strength deficits were previously unrecognized due to methodological differences in dynamometry techniques and data analysis. In addition, studies have demonstrated that hamstring harvest results in strength deficits of tibial internal rotation [312, 364]. The semitendinosus and gracilis tendons, however, are biarticular or two-joint muscle, therefore given that there are knee flexor strength deficits secondary to semitendinosus and gracilis harvest, it would follow that there would also be a concomitant reduction in hip extensor and hip adductor strength. There are no published studies examining hip strength after ACL reconstruction. This study was designed to evaluate hip extensor and hip adductor strength of subjects who have had a STG ACL reconstruction and subsequent rehabilitation. We hypothesize that significant strength deficits will exist in the hip extensors and hip adductors in this population.

MATERIALS AND METHODS

Fifteen subjects (age 28.7 years ± 9.05; mass 81.1 kg ± 15.4; 10 males and 5 females) who were greater than one year post STG ACL reconstruction were recruited from a tertiary care sport medicine centre for comparison to fifteen controls (age 29.6 years ± 2.6; mass 73.3 kg ± 9.19; 9 males and 6 females) with no previous knee injury. Both groups consisted of active recreational athletes, with no elite athletes included. The study was approved by the Review Board for Health Sciences Research Involving Human Subjects at the University of Western Ontario. All subjects completed an informed consent and subjective questionnaire.

Sample size was calculated using conservative estimates of mean expected difference and variance as well as a power index of 2.92 (α= 0.05 one-tailed; β = 0.1), the minimum number of subjects required in each group was determined to be fifteen.

All surgical subjects had undergone quadruple-looped semitendinosus and gracilis tendon ACL reconstruction greater than one year previously. The standardized arthroscopically-assisted technique used an Endobutton (Smith and Nephew Inc, Andover, MA) for femoral fixation, and a belt buckle staple configuration for tibial fixation. Subjects were allowed to participate in post-operative rehabilitation with the physiotherapist of their choice however standardized guidelines were given to each subject and physiotherapist.

All subjects underwent physical examination followed by strength evaluation of the hip extensor and hip adductors of both limbs. The surgical subjects completed a modified Quality of Life Questionnaire for ACL Deficiency [251].

The study of strength about the hip is less widely performed [54, 56, 235, 282 10, 331].

Dynamometry Strength Evaluation

A dynamometer (KinCom 500H, Chattecx Corporation, Hixson, TN) was used to evaluate concentric and eccentric hip extensor strength (50°/s and 150°/s) and isometric hip adductor strength (15° and 30°). Strength evaluation was performed bilaterally in both the ACL reconstructed and control groups. Prior to strength testing, subjects warmed up on a cycle ergometer (35 watts, 0.75 kilopound at 70 revolutions per minute) for five minutes.

Hip Extensor Tests Isovelocity hip extensor strength tests were performed in the standing position with the subject leaning slightly forward, stabilizing the torso on the dynamometer. The half seat of the dynamometer was removed to allow for hip flexion. The centre of rotation of the hip joint, as determined by palpation of the greater trochanter, was lined up with the centre of rotation of the actuator arm of the dynamometer. The actuator arm was strapped in a comfortable position to the posterior aspect of the subject's thigh. Subjects were familiarized to the strength testing protocol by performing 3 to 6 sub-maximal effort (less than 50% effort) concentric and eccentric repetitions at each speed prior to maximal effort testing. Testing consisted of three maximal effort concentric and eccentric contractions at the two speeds. Strength was measured over a 50° range of motion (20° hip flexion to 30° hip extension) with neutral corresponding to the thigh being in line with the torso. A five second pause was provided between successive concentric and eccentric contractions at each velocity. A two minute rest was given between test velocities.

Hip Adductor Tests

Isometric hip adductor strength testing was performed with the subject standing facing the actuator drive of the dynamometer. Subjects stood on a box to elevate them to avoid the foot hitting the floor. The centre of rotation of the hip joint was lined up with the centre of rotation of the actuator arm. Subjects were familiarized to the isometric strength testing protocol by performing 3 to 6 sub-maximal effort (less than 50% effort) at each angle prior to maximal effort testing. Isometric hip adduction strength tests were performed hip joint angles of 15 and 30° of adduction. Care was taken to maintain the centre of rotation of the hip at the centre of rotation of the actuator arm. Three maximal effort isometric contractions, held for 5 seconds, were performed at each joint angle, alternating between angles. Average moment data was collected for the three repetitions.

Data Collection, Reduction and Analysis

The dynamometer's "gravity correction" software feature was used to correct for the moment of the weight of the leg, foot and resistance pad for all isovelocity and isometric strength tests. The raw data from the dynamometer was exported to a computer and analyzed using Excel Software (Version 2000). The average of the three repetitions was calculated and used in the data analysis.

RESULTS

Hip Extensors

Comparison of hip extensor strength in the control group demonstrated statistically significant difference in hip extensor strength between the dominant and non-dominant limbs ($P<0.05$) with the dominant limb being stronger (Table 1). In the ACL

reconstruction group, comparison of dominant and non-dominant limbs demonstrated no significant difference in strength ($P>0.05$) (Table 1).

When dominance of the injured limb was factored in there was a trend suggesting a strength difference between dominant and non-dominant limbs. When the ACL reconstruction was performed on the dominant limb, the dominant limb was stronger at 50°/s both concentrically and eccentrically but not at 150°. When the ACL reconstruction was performed on the non-dominant limb there was no difference in strength between groups (Table 2).

Comparison of the control and ACL reconstructed groups demonstrate no significant strength deficits between groups for either the dominant or the non-dominant limb (Table 3). When you factor in which limb is injured, the ACL reconstructed group trends towards higher hip extensor resultant joint moment however this did not attain statistical significance. There was no correlation between hip extensor strength and Quality of Life scores.

Hip Adductors

Comparison of isometric hip adduction strength between the 15° adducted and 30° adducted positions demonstrate a statistically significant difference between the two angles for both the control group and the ACL reconstructed group ($P<0.01$). (Figure 2)

Comparison of hip adduction strength between dominant and non-dominant limbs demonstrated that in the control group there was a statistically significant strength difference between the limbs ($P<0.05$) at 15° with the non-dominant limb being stronger. This difference only approaches significance at 30° ($P=0.08$). In the ACL group there is

no significant difference between dominant and non-dominant limb strength ($P>0.05$) at both 15° and 30° adduction. If you factor in limb dominance, there was no difference in strength between the dominant and non-dominant limbs regardless of which limb was injured.

Comparison of the control group vs. the ACL reconstructed group hip adductor strength demonstrated significant strength deficits for both limbs at 15 ° ($P<0.05$). At 30°, the strength difference was significant for the non-dominant limb ($P<0.05$) and approached significance for the dominant limb ($P=0.07$). These strength deficits were up to 43.7% of control in the non-dominant limb and up to 36.8% in the dominant limb.

If limb dominance was separated, the group that had the ACL performed on the dominant limb no difference was found between the groups. If the ACL reconstruction was performed on the non-dominant limb, there was a larger difference between limbs that were significant for the non-dominant limb and approached significance for the dominant limb. There was no correlation between isometric hip adductor strength and Quality of Life scores.

DISCUSSION

The morbidity associated with autograft reconstruction using semitendinosus and gracilis tendons of ruptured ACL has not been fully elucidated. Strength changes have been demonstrated for knee flexors, knee extensors and the muscles controlling internal rotation and external rotation of the tibia [164, 312]. Given the observed effects of surgical intervention on the ST and G neuromuscular performance about the knee, it is important

to evaluate the effect on hip strength based upon the biarticular characteristic of the ST muscle, as well as the direct effect of G about the hip.

Studies investigating hip strength have described hip strength in normal, healthy controls [54, 56, 235] and in specific athletic populations[10, 331, 351]. These studies suggest that hip weakness or various types of imbalances may predispose these athletes to subsequent injury[10, 355]. Hip strength has been studied in some pathologic conditions. After arthroscopic knee surgery, Jaramillo et al demonstrated a 25.3% isometric hip extensor strength deficit when compared to the contralateral limb[183]. In the ACL deficient population, statistically significant isometric hip adduction, abduction and flexion strength deficits were demonstrated compared to the contralateral limb prior to any ACL reconstruction surgery[246]. This study has been the first to investigate strength of the hip extensors and hip adductors in a subject group who has undergone ACL reconstruction using the semitendinosus and gracilis tendons and compare it to an uninjured control group.

The resultant joint moment for hip extension is accomplished by the action of many muscles including gluteus maximus, biceps femoris, semimembranosus, and semitendinosus. The gluteus maximus crosses only the hip joint and has a larger physiological cross-section than the semitendinosus. One would not expect a large deficit in the hip extension resultant joint moment as a direct result of harvest of the semitendinosus tendon given its small cross-sectional area of the hip extensor muscles. This study has demonstrated that in a healthy uninjured population, significant strength differences exist between dominant and non-dominant limbs for hip extension. These findings are consistent with those described by Nadler et al.[257] This side to side strength

imbalance seen in the control group is no longer evident in the ACL reconstructed subject group. No statistically significant strength difference was found between limbs in the ACL group and the strength had equalized at the stronger moment value. When dominance of the injured limb is taken into account, the reconstructed limb increases in strength, balancing limbs when the non-dominant limb is injured and maintaining the strength "imbalance" when the dominant limb is injured, at least at the slower angular velocities. This suggests that rehabilitation programs for ACL reconstruction are successful in maintaining and perhaps improving the strength of the ACL reconstructed limb. Another explanation is that the hip extensors compensate and adapt secondary to altered knee kinematics which arise from strength deficits of knee flexion, extension, and internal rotation.

Hip extension strength changes also may have implications for knee/hip strength imbalance. Strength deficits have been identified in the knee flexors and extensors after hamstring ACL reconstruction[164]. The lack of corresponding strength changes in the hip extensors may lead to a knee-hip strength imbalance. Hip strength and knee strength have been shown to be related. It has been demonstrated that varying the hip angle will affect knee resultant joint moment and knee extensor muscle excitation[48, 88, 152]. The functional consequences of altered hip-knee strength balance have yet to be determined. The finding that graft re-rupture rate has been shown to be higher in skiers after hamstring ACL reconstruction compared to patellar tendon reconstruction [265] may be a function of altered neuromuscular balance in the lower limb [79]. Strength changes in the hip extensors and adductors may have implications for rehabilitation and return to previous levels of sport. In addition, these strength imbalances may predispose to graft rupture or subsequent

injury. The resultant joint moment for hip adduction demonstrates large and significant strength changes between hip adduction at 15° and 30°. This is true for both the control and the ACL reconstruction groups. This finding justifies the testing of both joint angles in this study. Comparison between dominant and non-dominant limbs in the healthy uninjured group demonstrates a statistically significant strength difference at 15° which approaches significance at 30° in the control group. In the ACL group, there was no difference between index and contralateral limb hip adduction strength and thus a change from the normal dominant non-dominant strength imbalance. If limb dominance of the ACL reconstructed group is analyzed separately, the group who had the ACL performed on the dominant limb demonstrated no right- left strength difference but the group who had the ACL performed on the non-dominant limb did demonstrate side to side strength difference similar to the control group. Comparison of the control group and the ACL reconstructed group demonstrates an equalization of limb strength in the ACL reconstructed group. This hip adductor strength deficit is large and statistically significant with strength deficits of up to 42% of control.

These large adductor strength deficits would seem to have both a mechanical and a neural etiology. The small cross-section area of the gracilis and the semitendinosus would seem inadequate to account for the large strength deficits demonstrated. This large adductor strength deficit is concerning however, given that the adductors are important for pivoting and deceleration activities, those which put the most strain on the ACL.

This study increases our knowledge of the morbidity of semitendinosus and gracilis tendon harvest for ACL reconstruction. This information must be taken in context with information on changes in knee strength as well as other complications of autograft

harvest. The precise etiology of the changes in strength about the hip after hamstring ACL reconstruction has yet to be determined. Pure mechanical factors are not enough to account for the strength changes that have been demonstrated, especially those in the contralateral uninjured limb. The process therefore, of ACL injury, de-training in the post-injury pre-surgical period, ACL reconstruction surgery and subsequent rehabilitation may all play a role in the neuromuscular changes that occur about the hip. The functional significance of the demonstrated hip strength changes have yet to be determined.

Table 1. Comparison of dominant and non-dominant limb between the control group and the ACL reconstructed group. *** statistically significant different in hip extensor strength. SD = standard deviation.

Angular Velocity	Dominant limb Nm/kg (SD)	Non-Dominant limb Nm/kg (SD)	P-Value (paired t-test)
	Control	**Control**	
150°/s eccentric	*** 1.78 (0.85)	1.53 (0.67)	0.014
50°/s eccentric	*** 1.65 (0.81)	1.46 (0.68)	0.019
50°/s concentric	*** 1.51 (0.83)	1.24 (0.65)	0.026
150°/s concentric	*** 1.66 (0.71)	1.45 (0.61)	0.023
	ACL Reconstructed	**ACL Reconstructed**	
150°/s eccentric	2.06 (0.69)	1.98 (0.56)	0.570
50°/s eccentric	1.94 (0.60)	1.79 (0.59)	0.214
50°/s concentric	1.45 (0.57)	1.45 (0.55)	0.957
150°/s concentric	1.70 (0.59)	1.62 (0.56)	0.176

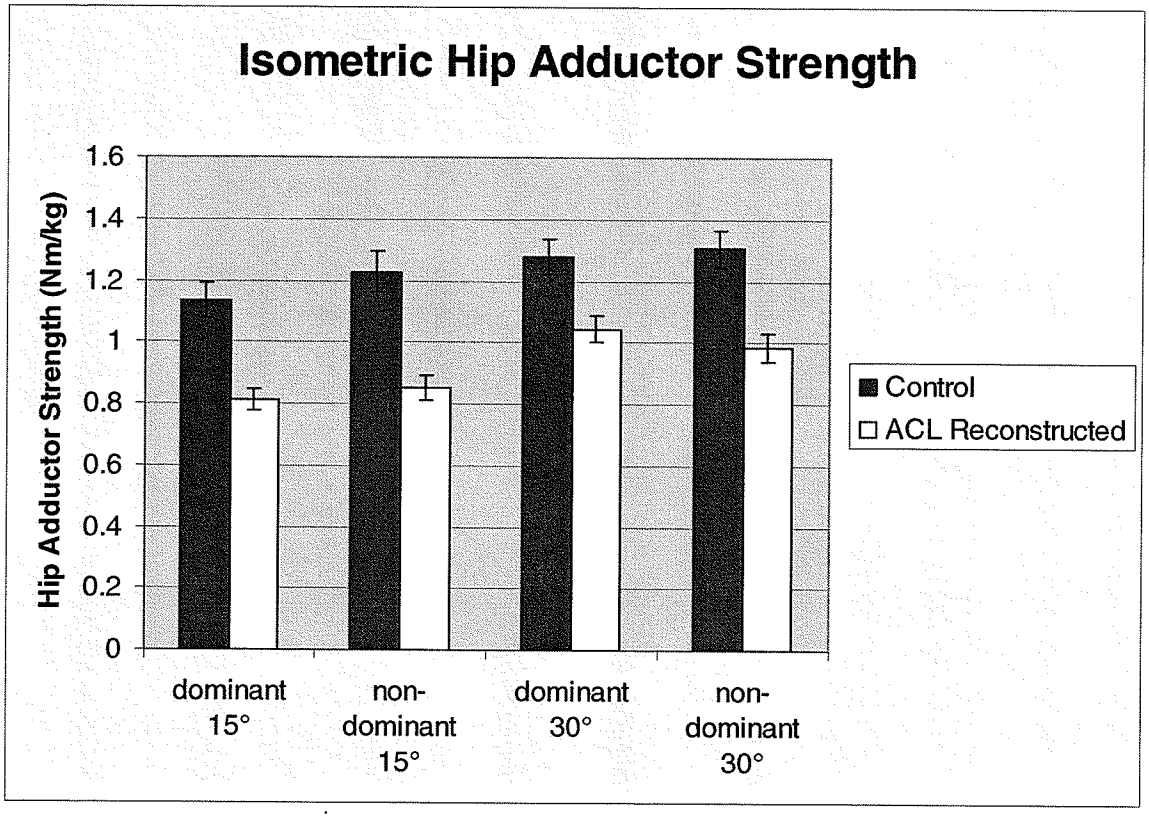
Table 2. ACL reconstructed group divided to factor in dominance of ACL reconstructed limb. *** P<0.05. SD = standard deviation.

Angular Velocity	Dominant limb Nm/kg (SD)	Non-Dominant limb Nm/kg (SD)
	ACL on Dominant limb	**ACL on Dominant limb**
150°/s eccentric	2.25 (0.86)	1.97 (0.54)
50°/s eccentric	***2.11 (0.77)	1.81 (0.63)
50°/s concentric	***1.65 (0.62)	1.50 (0.44)
150°/s concentric	1.92 (0.60)	1.69 (0.49)
	ACL on Non-dominant limb	**ACL on Non-dominant limb**
150°/s eccentric	1.97 (0.46)	2.00 (0.63)
50°/s eccentric	1.81 (0.42)	1.78 (0.59)
50°/s concentric	1.50 (0.48)	1.39 (0.67)
150°/s concentric	1.69 (0.55)	1.56 (0.65)

Table 3. Comparison of ACL reconstructed group to the control group. *** statistically significant difference. SD = standard deviation.

Angular Velocity	Control Nm/kg (SD)	ACL Reconstructed Nm/kg (SD)	P-Value (paired t-test)
	Dominant	**Dominant**	
150°/s eccentric	1.78 (0.85)	2.06 (0.69)	0.263
50°/s eccentric	1.65 (0.81)	1.94 (0.60)	0.067
50°/s concentric	1.51 (0.83)	1.45 (0.57)	0.826
150°/s concentric	1.66 (0.71)	1.70 (0.59)	0.476
	Non-Dominant	**Non-Dominant**	
150°/s eccentric	1.53 (0.67)	1.98 (0.56)	0.056
50°/s eccentric	1.46 (0.68)	1.79 (0.59)	0.200
50°/s concentric	1.24 (0.65)	1.45 (0.55)	0.299
150°/s concentric	1.45 (0.61)	1.62 (0.56)	0.399

Figure 1. Isometric hip adductor strength comparison between control and ACL reconstructed group. * = statistically significant difference.



Summary of Results

Study 1 - Graft Site Dependent Knee Strength Deficits after Patellar Tendon and Hamstring Tendon ACL Reconstruction

1. All subjects who had undergone ACL reconstruction, independent of autograft donor site, demonstrated a significant 25% **global** knee extensor strength deficit compared to an active control group which was greater than previously reported in the literature. This strength deficit was up to 50% during eccentric contractions compared to controls. Since this knee extensor strength deficit appears independent of graft type, it represents a 'specificity of injury', analogous to the concept of specificity of training.
2. The hamstring autograft ACL reconstruction group demonstrated a graft-site dependent, **regional** knee flexor strength deficit that was not observed in the BPB group. This deficit was greatest during high speed eccentric contractions over all joint angles. This represents a neuromuscular adaptation that is specific to the subsequent surgery and rehabilitation.
3. The BPB ACL reconstruction group did not have a knee flexor strength deficit.
4. Subjects who had undergone a BPB autograft had an additional regional knee extensor strength deficit that was not present in the H group. This deficit was located during low speed concentric contractions from 65-85° of knee flexion.
5. Strength deficits in this study that compared to an uninjured control group were greater than previous studies that used the contralateral leg as the control. This is consistent with the theory that substantial **contralateral** leg deficits are also present in this population.

6. These findings suggest both a neural (regional strength deficits) and a muscular (global strength deficits) etiology of knee extensor and flexor deficits as the existence of regional strength deficits which are specific to joint angle, angular velocity or contraction type could only exist through a neural process and can not be explained by alterations in muscular or mechanical properties.
7. Strength deficits demonstrated using comprehensive modern dynamometry techniques suggest that previous studies may have had inadequate strength testing protocols and were therefore unable to fully elucidate strength deficits.

Study 2 - Hamstring and Quadriceps Strength Balance in Normal and Hamstring ACL Reconstructed Subjects

1. There is a systematic variation in the H/Q ratio and DCR dependent on knee joint angle, contraction type (eccentric and concentric) and angular velocity. Representing these as a single numerical value such as the well-published H/Q ratio, gives an incomplete portrayal of neuromuscular balance.
2. Balance is actually a state of inequity in strength between the two muscle groups, whereby the H is stronger near flexion and Q is stronger in extension.
3. Angle and velocity matched H/Q ratio maps and dynamic control ratio maps are reproducible for control and ACL reconstructed subjects.
4. Regional changes occur in agonist-antagonist ratio with ACL reconstruction. The ACL reconstructed group showed a tendency toward a higher H/Q ratio at angles of knee extension where the strain on the ACL is the greatest and reflex pathways may be activated resulting in inhibition to quadriceps and excitation of hamstrings.

Study 3 - Contralateral Leg Strength Deficits after Hamstring Tendon ACL Reconstruction

1. No statistically significant difference was detected comparing knee extensor peak moments between the ACL reconstructed and contralateral uninjured limbs at any of the velocities tested. Bilateral strength normalization was achieved for the knee extensors defined as strength within 10% of contralateral limb.
2. Significant strength deficits are identified when knee extensor strength of the contralateral and ACL reconstructed legs are compared to an age and activity matched control group. This strength deficit is bilateral and is up to 30% in some regions (knee joint angle 55-90°, eccentric, high speed).
3. A statistically significant strength deficit is identified during eccentric contractions when comparing knee flexor strength in ACL reconstructed leg compared to the contralateral leg. This deficit measures up to 19.9% in certain regions (low velocity eccentric contractions, through all joint angles); as such bilateral strength normalization is not achieved.
4. When compared to an age and activity matched control group, the knee flexor strength deficit is statistically significant across all angular velocities. This strength deficit is up to 31.7% in certain regions (angle 5-50°, high angular velocities, eccentric>concentric).
5. For both the knee flexors and knee extensors, the strength deficits between the ACL reconstructed group and the control group are greatest during eccentric contractions. This variability in strength deficits according to contraction type suggests a neural etiology to strength changes. Eccentric knee flexor strength

deficits may have implications for the ability to control knee joint stability during knee extension.

6. A significant, positive correlation was found between the Quality of Life score and concentric knee extensor peak moments ($r=0.61$, $p=0.027$).

Study 4- Effect Of Fatigue On Knee Proprioception: Implications For Dynamic Stabilization

1. Afferent feedback from muscle receptors is altered in the fatigued state.
2. There is conflicting information regarding changes in proprioception of the knee joint that occur during fatigue.
3. Afferent input originating from the knee joint and muscles about the knee does exert an influence in the activation of the muscles that control the knee motion and thus may contribute to dynamic stability of the knee joint.

Study 5 - The Development of Contralateral Leg Strength Deficits after Acute Anterior Cruciate Ligament Injury

1. Knee flexor and extensor strength of the contralateral limb is immediately reduced relative to control after an acute ACL injury consistent with neurally mediated inhibition.
2. Strength of the contralateral limb after ACL injury returns slowly to levels just below that of the contralateral limb of subjects greater than one year post ACL reconstruction.

3. Significant strength deficits exist in the contralateral uninjured limb relative to control at 16 weeks after ACL injury (15% knee flexor; 28% knee extensor). These strength deficits are consistent with the magnitude of the contralateral limb strength deficits demonstrated in Study 3.

Study 6 - Hip Extensor and Adductor Strength Following Hamstring Tendon Anterior Cruciate Ligament Reconstruction

1. There is a hip extensor strength difference between dominant and non-dominant limbs in healthy active subjects with the dominant limb stronger than the non-dominant.
2. After hamstring ACL reconstruction, strength between limbs equals out at the strength of the stronger dominant limb (strength gain).
3. Comparison of isometric hip adduction strength between the 15° adducted and 30° adducted positions demonstrate a statistically significant difference between the two angles for both the control group and the ACL reconstructed group.
4. There is an isometric hip adductor strength difference between dominant and non-dominant limbs in healthy active subjects with the dominant limb stronger than the non-dominant.
5. In hamstring ACL reconstructed subjects significant hip adductor strength deficits exist for both limbs at 15° which approach significance at 30°. These strength deficits were up to 43.7% of control in the non-dominant limb and up to 36.8% in the dominant limb.

Discussion

This goal of this series of studies was to elucidate and characterize the changes in neuromuscular control after injury in humans using ACL rupture and reconstruction as a model. Strength deficits, both global and regional, have been identified in the knee flexors and extensors after ACL injury and after ACL reconstruction and subsequent rehabilitation (Study 1,5). Strength changes in the hip extensors and hip adductors have also been identified after hamstring ACL reconstruction (Study 6). These changes are seen in both the ipsilateral injured limb as well as the contralateral uninjured limb (Study 3). These modifications in neuromuscular control must result from alteration of the neural control of motoneurone activation or the intrinsic muscular or mechanical properties of the sensorimotor system [96]. Neural alterations of motor unit activation could arise from changes in sensory integration arising from the injury or surgical intervention, changes at the spinal cord level, modification by higher centres or volitional control. Muscular or mechanical changes can result from changes in the force producing capability of the muscles secondary to muscular atrophy, change in the force-length relationship of the muscle or change in the moment arm of the muscle-tendon unit. These studies represent important early investigations to differentiate the contributing roles of neural and muscular/mechanical factors to changes in neuromuscular control that has been demonstrated after injury in humans during voluntary contractions.

A largely neural etiology for the alteration in the moment producing capabilities of the muscles which control the hip and knee after injury and surgery is supported throughout the series of studies. The existence of regional strength deficits which are specific to joint angle, angular velocity or contraction type could only exist through a

neural process and can not be explained by alterations in muscular or mechanical properties. If the demonstrated strength deficits were secondary to a muscular or mechanical etiology, the deficit should be largely maintained across angular velocities and contraction types. A neural mechanism is also supported by the fact that the regional strength deficits were graft site specific. Alteration of the afferent information from both the flexor (hamstring) and extensor (patellar tendon) mechanisms by graft harvest led to site specific alteration in the neuromuscular activation patterns during maximum voluntary contraction strength (Study 1). A neural etiology is also supported by the presence of contralateral leg strength deficits which are also regional in nature (Study 3). Detraining or muscle atrophy alone could not account for regional strength deficits as the strength deficit would be consistent across angular velocities, joint angles and contraction types. These changes in motor output in the contralateral limb are evident immediately after an injury giving further support to a neural etiology as there would not have been enough time for muscular atrophy to take place (Study 5). Finally, alterations in bi-articular muscles are shown to have differential effects on the two joints that they cross with large knee strength deficits and no hip extensor strength deficits seen after hamstring tendon harvest (Study 6). This is only possible through neural mediation of motor output. Thus, just as it has been well established in strength literature that there is specificity of strengthening resulting from a combination of neural and muscular changes. In the same way, we propose that de-conditioning also would involve both neural and muscular contributions with specificity of injury.

There is evidence that changes in the muscular and mechanical properties contribute at least in part to the demonstrated strength deficits. In addition to the observed

regional strength deficits, a global knee extensor strength deficit was identified regardless of graft type. This subject population undergoes a period of decreased activity after their injury. This period of decreased training or incomplete rehabilitation could account for the ACL injury dependent global strength deficits identified in this population. Strength deficits demonstrated in the contralateral limb were not completely accounted for by nociceptive crossover in the first 16 weeks after ACL injury (Study 5). Reduced volume of activity and secondary muscular atrophy could account for a portion of the contralateral leg strength deficits. Some of the graft-site specific strength deficit could be due to alteration in the cross-sectional area or moment arm with harvest of the semitendinosus and gracilis tendons. Although further studies are required to establish this effect, some preliminary (unpublished) work from this lab suggests that at least part of the deficit is secondary to muscular or mechanical causes.

The neural basis of these alterations in motor output appears established (Study 1-3,5,6) however the exact sensorimotor mechanisms by which this takes place is still unclear. Further studies are needed to establish the precise mechanisms by which motoneurone activation is influenced however, certain observations as to the nature of the changes in neuromuscular control can be made. It is clear that the regulation of eccentric contractions is unique when compared to the regulation of concentric contractions [95, 368]. After injury or surgery, a larger magnitude strength deficit is demonstrated during eccentric contractions compared to concentric contractions (Study 1,3,5). Study 5 demonstrates selective recovery of eccentric knee flexor strength after an acute ACL injury.

The exact physiological mechanisms behind the observed changes in voluntary muscle contraction after injury and surgery are still to be determined. The most likely hypothesis is that nociceptive afferent information from the injury or the knee joint effusion causes inhibition of the contralateral motor pool. This is consistent with findings that nociceptive input has contralateral effects [40, 41]. In addition, mechanoreceptor afferent information from the ACL is no longer provided. Another possible mechanism for a neural etiology for altered motor output is the change in information from mechanoreceptors within the ruptured ACL. Once ruptured, the ligament will no longer develop tension with anterior translation of the tibia on the femur and thus the afferent input to the spinal cord and higher centres will be altered. With increased antero-posterior laxity, mechanoreceptors within the joint and muscle spindles in the muscles about the joint may have altered afferent output. This is consistent with findings that in humans with ACL deficiency, alteration in the motor output from the knee extensors and knee flexors has been documented [27, 350, 382]. The spinal cord may control motor output through spinal cord reflexes as well as through neural crossover. Finally higher centres may modulate motor output through a feedback mechanism or through volitional control. From one or multiple of these mechanisms, we hypothesize that altered afferent feedback from the knee joint and muscles surrounding the knee results in changes in motor unit recruitment/activation patterns leading to regional strength deficits.

Some limitations to the preceding series of studies exist. The measurement of maximum voluntary strength is different than the measurement of motor output during functional tasks. Strength assessment on a dynamometer is not a weight bearing assessment and therefore may not be directly predictive of functional outcomes. In

addition, strength is an indirect measure of neuromuscular function. No direct measurement of motoneurone activation was performed in this series or measurement of sensory systems. Finally, identification of strength deficits in this population has not been correlated to objective functional outcome measurements. Although strength deficits relative to peers would suggest poorer function, it has not been demonstrated that amelioration of these deficits would improve function or reduce the risk of further injury.

Future studies should continue to try to characterize the changes in neuromuscular control that occur after injury and surgery. In addition, systematic investigation will help to elucidate the physiological mechanisms behind the described changes. The institution of maintenance strengthening programs may help prevent contralateral leg strength deficits. Specific strengthening programs designed to target regional strength deficits will provide insight into whether these are mutable. The application of a local anaesthetic into the joint space of an acutely injured knee with pre and post strength testing of the contralateral leg will help elucidate the role of nociceptive afferent information for contralateral leg strength deficits. Quantification of the amount of decrease in activity following acute ACL injury may help to determine the role of de-conditioning in contralateral leg strength deficits after an acute injury. Finally, the functional consequences of the described strength deficits need further quantification in an objective manner. This could be accomplished by using emerging technologies (GPS, triaxial accelerometry, heart rate monitoring) to devise objective functional outcome measures. Finally, the functional consequences of the regional strength deficits could be determined by investigating the differential acceleration of limb segments dynamic task. The attachment of triaxial accelerometers to the thigh and the shank during functional

activities will determine whether strength deficits lead to a decreased ability to control acceleration of the lower limb.

These studies provide data which support the hypothesis that alteration in motor unit activation after injury and surgery is largely under neural control. The most plausible explanation for altered motor output after injury and surgery is alteration of neural activation strategies leading to modification of motoneurone recruitment/activation during different speeds, contraction types and ranges of motion.

Clinical Application of Findings

The findings in these studies have direct clinical applications with the potential for immediate implementation. Directed knee extensor rehabilitation protocols should be implemented to address the global knee extensor strength deficits identified after ACL injury and reconstruction (Study 1). In addition, a greater emphasis is required on eccentric contractions for both the knee flexors and the knee extensors. Rehabilitation protocols specific to procedure type (hamstring or patellar tendon ACL reconstruction) should be implemented to ameliorate strength deficits that are specific to graft site. (Study 1) A more potent knee flexor and hip adductor strengthening protocol is necessary after hamstring tendon autograft reconstruction (Study 1 and 6). Use of bilateral strength normalization should be one progression in the process of rehabilitation with the endpoint being the restoration of strength to the level of their peers (Study 3). The restoration of strength balance (both agonist-antagonist and right-left) should be included as an endpoint of rehabilitation (Study 2). After an acute ACL injury preventative maintenance strengthening programs for the contralateral limb may help prevent contralateral leg strength deficits (Study 5). Finally, these studies have demonstrated that we should not be

complacent in our knowledge of the neuromuscular changes that occur after injury. Continuous vigilance is necessary to maintain a high level of excellence in our management of musculoskeletal injuries. The concepts discussed here can be extrapolated to other injuries and surgeries and will have implications in the fields of medicine, rehabilitation, and physiology.

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Appendix A – Modified Quality of Life Questionnaire

Quality of Life Assessment in Anterior Cruciate Ligament Reconstruction

Symptoms and Physical Complaints:

1- With respect to your overall knee function. How troubled are you by giving way episodes? (Make a slash at the extreme right, i.e., 100, if you are experiencing no giving way episodes in your knee. Please note that this question has two parts. It is concerned with both the severity (1a) and frequency (1b) of the giving way episodes.)

1a- 0 _____ 100

Major giving way episodes — Minor giving way episodes

1b- 0 _____ 100

Constantly giving way — Never giving way

2- With any kind of prolonged activity (i.e., greater than half an hour) how much pain do you get in your knee?

0 _____ 100

Severe pain — No pain at all

3- With respect to your overall knee function, how much are you troubled by stiffness of loss motion in your knee?

0 _____ 100

Severely troubled — Not troubled at all

4- Consider the overall function of your knee and how it relates to the strength of your muscles. How weak is your knee?

0 _____ 100

Extremely troubled — Not weak at all

Work-Related Concerns:

The following questions are being asked with respect to your job of vocation. The questions are concerned with your ability to function at work and how your knee has affected your current working situation, i.e., you work-related concerns. If you are a full-time student or homemaker consider this and any part-time work together. Consider the last three months.

If you are currently not employed for reasons other than your knee then place a check on this line. _____

5- How much trouble do you have, because of your knee, with turning or pivoting motions at work? (Make a slash at the extreme left, i.e., 0, if you are unable to work because of the knee.)

0 ——————————————————— 100

Severely troubled No trouble at all

6- How much trouble do you have, because of your knee with squatting motions at work? (Make a slash at the extreme left, i.e., 0, if you are unable to work because of the knee.)

0 _____ 100

Severely troubled No trouble at all

7- How much of a concern is it for you to miss days from work due to problems or re-injury to your knee? (Make a slash at the extreme left, i.e., 0, if you are unable to work because of the knee.)

0 _____ 100

An extremely significant concern No concern at all

8- How much of a concern is it for you to lose time from "school" or work because of the treatment of your ACL-reconstructed knee?

0 _____ 100

An extremely significant concern No concern at all

Recreational Activities and Sport Participation or Competition:

The following questions are concerned with your ability to function and participate in these activities as they relate to your ACL-reconstructed knee. Consider the last three months.

9- How much limitation do you have with sudden twisting and pivoting movements or changes in direction?

0 _____ 100

Totally limited — No limitations

10- How much of a concern is it for you that your sporting or recreational activities may result in the status of your knee worsening?

0 _____ 100

An extremely significant concern — No concern at all

11- How does your current level of athletic or recreational performance compare with your pre-injury level?

0 _____ 100

Totally limited — No limitations

12- With respect to the activities or sports that you currently desire to be involved with, how much have your expectations changed because of the status of your knees?

0 _____ 100

Exceptions totally lowered — Expectations not lowered at all

13- Do you have to play your recreation or sport under caution? (Make a slash at the extreme left, i.e., 0, if you are unable to play recreation or sport because of your knee.)

0 _____ 100

Always play under caution — Never play under caution

14- How fearful are you of your knee giving way when playing recreation or sport? (Make a slash at the extreme left, i.e., 0, if you are unable to play recreation or sport because of your knee.)

0 _____ 100

Extremely fearful — No fear at all

15- Are you concerned about environmental conditions such as wet playing field, a hard court, or the type of gym floor when involved in your recreation or sport? (Make a slash at the extreme left, i.e., 0, if you are unable to play recreation or sport because of your knee.)

0 _____ 100

Extremely concerned — Not concerned at all

16- Do you find it frustrating to have to consider your knee with respect to your recreation or sport?

0 _____ 100

Extremely frustrated — Not concerned at all

17- How difficult is it for you to "go full out" at your recreation or sport? (Make a slash at the extreme left, i.e., 0, if you are unable to play recreation or sport because of your knee.)

0 _____ 100

Extremely difficult Not difficult at all

18- Are you fearful of playing contact sports? (Circle the "N/A" at the right of the scale if you do not play contact sport for reasons other than the knee.)

0 _____ 100 N/A

Extremely fearful No fear at all

The following questions are specifically asking about the two most important sports or recreational activities that you do or that you wish to do. Please write them in order.

1. _____

2. _____

19- How limited are you in playing the number "1" sport or activity? (Make a slash at the extreme left, i.e., 0, if you are unable to play the recreation or sport because of your knee.)

0 _____ 100

Extremely limited Not limited at all

20- How limited are you in playing the number "2" sport or activity? (Make a slash at the extreme left, i.e., 0, if you are unable to play the recreation or sport because of your knee.)

0 _____ 100

Extremely limited Not limited at all

Life Style:

The following questions are concerned with your life style in general and should be considered outside of your work and recreational or sport activities as they relate to your ACL reconstructed knee.

21- Do you have to concern yourself with general safety issues (e.g., carrying small children, working in the yard) with respect to your ACL-reconstructed knee?

0 _____ 100

Extremely concerned No concern at all

22- How much has your ability to exercise and maintain fitness been limited by your knee problem?

0 _____ 100

Totally limited Not limited at all

23- How much has your enjoyment of your life been limited by your knee problem?

0 _____ 100

Totally limited Not limited at all

24- How often are you aware of your knee problem?

0 _____ 100

All of the time None of the time

25- Are you concerned about your knee with respect to life style activities that you and your family do together?

0 _____ 100

Extremely concerned No concern at all

26- Have you modified your life style to avoid potentially damaging activities to your knee?

0 _____ 100

Totally modified No modifications

Social and Environmental:

The following questions are about your attitudes and feelings as they relate to your ACL-reconstructed knee.

27- Does it concern you that your competitive needs are no longer being met because of your knee problem? (Make a slash at the extreme right, i.e., 100, if your competitive needs are being met.)

0 _____ 100

Extremely concerned Not concerned at all

28- Have you had difficulty being able to psychologically "come to grips" with your knee problem?

0 _____ 100

Extremely difficult Not difficult at all

29- How often are you apprehensive about your knee?

0 _____ 100

All of the time None of the time

30- How much are you troubled with lack of confidence in your knee?

0 _____ 100

Severely troubled Not troubled at all

31- How fearful are you of re-injuring your knee?

0 _____ 100

Extremely fearful No fear at all

Modified from:

Nick Mohtadi: Quality of life Questionnaire for ACL Deficiency. The American Journal of Sports Medicine. 26 (3): 350-359, 1998