UNIVERSITY OF CINCINNATI

February 8, 2002

I, Jason T. Shearn, hereby submit this as part of the requirements for the degree of:

Doctor of Philosophy

in:

Engineering Mechanics

It is entitled:

POSTERIOR CRUCIATE LIGAMENT: STUDIES OF ONE AND TWO-BUNCLE RECONSTRUCTIONS

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POSTERIOR CRUCIATE LIGAMENT: STUDIES OF ONE AND TWO-BUNDLE RECONSTRUCTIONS

A dissertation submitted to the

Division of Research and Advanced Studies
of the University of Cincinnati

In partial fulfillment of the
requirements for the degree of

DOCTOR OF PHILOSOPHY (Ph.D.)

In the department of Aerospace Engineering and Engineering Mechanics
Of the College of Engineering

2002

by

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ABSTRACT

The posterior cruciate ligament (PCL) is the primary restraint to posterior translation and is a secondary restraint to varus, valgus and external rotation.[12, 32, 43, 60] PCL injuries increase posterior translation and impair a person’s ability to perform daily activities.[59, 67, 100] If left untreated, degenerative changes will appear earlier in the injured knee than in the contralateral knee.[17, 79, 82] A variety of surgical procedures have been used in an attempt to restore normal posterior translation. Some procedures have been unable to restore posterior translation.[48, 77, 111, 127] while others have been unable to maintain normal posterior translation[65, 85, 90]. The failure to control posterior translation has been attributed to graft elongation.[48, 81, 85] In an attempt to prevent graft elongation, two-bundle reconstructions have been investigated[65, 81, 119] and have shown promising initial results. However, the resistance to posterior translation is unknown. The purpose of this research was to investigate the ability of one and two-bundle PCL reconstructions to resist posterior translation during cyclic fatigue testing.

The femoral attachment of the one-bundle graft and one bundle (AL₂) of all two-bundle grafts were located within the PCL’s anterolateral band, which is named for its anterior femoral insertion and its lateral tibial insertion. The second bundle was placed posterior to the AL₂ bundle in one of three locations that varied in their depth within the notch: shallow (S), intermediate (I), and deep (D). The specimens were cycled from near full extension to 120° of flexion with 100 N applied posterior force. The knees were cycled until the tension in both bundles was 50 N or less.

The two-bundle reconstructions did not provide better resistance to posterior translation than the one-bundle reconstruction for a 2.5-mm posterior translation increase. The AL₂-I
reconstruction resisted the return of posterior translation for significantly more cycles than the one-bundle reconstruction, but the two-bundle reconstructions did not reduce the rate of posterior translation return as the posterior translation increased to 7.5 mm. However, the rate of return for the AL2-I reconstruction decreased as the posterior translation increased beyond 2.5 mm.

This study did not provide a reconstruction that replicated the intact knee’s resistance to posterior translation, but several areas were discovered that might lead to the improvement of current PCL reconstructions.
ACKNOWLEDGEMENTS

I am grateful to the many individuals that I have had the pleasure to meet during the course of my dissertation. I cannot thank them enough for their help and contribution to this work and the hours of distraction that made the work bearable.

First, my wife, Rebecca, without you the completion of this dissertation would have been significantly more difficult because you were always there to help with testing and to read any of my abstracts, posters and manuscripts. You took care of the things outside of my research to free my time and effort. You were always there for me no matter what. I love you with all of my heart.

To my mentor, Edward Grood, you are the reason I came here, I choose this work, and I stayed at the University of Cincinnati. You were always available to help me with any problems, but it was the respect you gave me and trust you had in me that drove me to improve as an individual and as a researcher. I will never be able to repay the wisdom that you have bestowed to or the friendship that we have.

To my committee members, David Butler, I owe you a debt of gratitude for the knowledge you have imparted to me in the classroom and for including me in research discussions outside of my work. Marty Levy, your ability to convey statistics has been invaluable. Ala Tabiei, you expanded my intellectual horizons through your caring about teaching. I took as many classes of yours that I could fit in because you were a wonderful professor.

To my parents, without your love, support and encouragement I would not be the person that I am today. To my brother, your love and friendship has meant a great deal to me over the
years. To my grandparents, you watched over me as your own, and I cannot begin to express how I feel about you.

To Frank Noyes, I am grateful for the time and the expertise that you have provided to me throughout to the completion of this work. To the staff of Cincinnati Sports Medicine Research and Education Foundation, I thank you for the support you have provided to me throughout my tenure at UC. To the Cincinnati Sports Medicine Research and Education Foundation, I would not have been able to complete this work without your financial support.

To the Musculoskeletal Tissue Foundation, I am grateful for your generous gift of samples because without the donations this work could not have been completed.

To my fellow graduate students, past and present, you have helped with my work, have become my friends, and have shown me that completing a doctoral degree is possible. I would like to thank you (Hani Awad, Dave Holtzclaw, Bala Harridas, Eric Schantz, Shawn Hunter, Matt Dressler, John West, Shun Yoshida, and Natalia Juncosa) for your contributions. To Dale Knochenmuss, I thank you for providing me with a knowledge base in mechanical drawing and with the testing system. To the former and current staff of the Department of Aerospace Engineering and Engineering Mechanics, I am grateful to you (especially Stacy Webber and Stanley Rubin) for your guidance during my early years at UC. To the staff of the Biomedical Engineering Department, I thank for your support and friendship. To my co-op students, Erik Petersen, Daniel Marsh, and Gwendolyn Perez, I thank you for your contributions to this dissertation.

To my friends outside of the university, I am grateful to you for providing an outlet that had absolutely nothing to do with school or research.

I dedicate this work to my wife, Rebecca, and to my family. I love you all so much.
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CHAPTER 1

INTRODUCTION

Investigators have found the posterior cruciate ligament (PCL) to be important to normal knee function.[29, 31] The PCL serves as the primary restraint to posterior\textsuperscript{1} tibial translation by providing over 90\% of the resistive force,[3, 12, 23, 31, 76, 93, 137] and the PCL also serves a secondary restraint to external, varus, and valgus rotations.[12, 32, 43, 60] The PCL, in combination with the ACL, helps produce the screw home motion of the knee as it goes into extension.[43, 88, 89] The PCL has also been identified as the primary stabilizer of the knee [29, 41, 74, 76, 78, 111, 135, 137, 139, 140].

PCL injuries alter knee kinematics,[31, 41] which can lead to short\textsuperscript{2} and long term disability.[4, 36] The frequency of PCL injury is low, approximately 7\% of all knee ligament injuries.[91] PCL injuries cause pain,[4, 29, 36-38, 64, 67, 100, 105] swelling[4, 37, 38, 64, 67, 105] and instability\textsuperscript{2} [36, 41, 64, 79, 82, 105] in the first few weeks following a PCL injury. These symptoms can affect the quality of life of the person by limiting his/her ability to perform daily activities such as ascending and descending stairs and walking long distances.[59, 67, 100]. In an athlete, a PCL injury can be career threatening [24, 44, 59].

If the PCL injury remains untreated, changes to other structures in the knee will occur in as little as three months after injury[4, 36] with some changes being irreversible.[83] As a result of the injury, the tibia will translate to a more posterior position. This posterior tibial position will lead to changes in the anterior cruciate ligament (ACL) and the knee extensor mechanism.

\textsuperscript{1} definition found in APPENDIX A
\textsuperscript{2} A condition of a joint characterized by an abnormal increased range of motion (mobility) due to an injury to the ligaments, capsule, menisci, cartilage or bone.”[98]
The new tibial position shortens the distance between the femoral and tibial attachment sites of the ACL, thus reducing the tension in the ACL. The ACL will remodel to accommodate the new mechanical environment. The remodeled ACL has been shown to have a lower tensile strength, but the stiff was not altered.

In addition to weakening the ACL, a posteriorly positioned tibia alters the knee extensor mechanism, which can lead to osteoarthritis. These alterations result from the posterior tibial position, which changes the angle the patellar tendon makes with the patella resulting in increased contact pressure between the patella and the femoral condyles. Over time the resulting degenerative changes can produce permanent knee pain. The increased contact pressure will cause the articular cartilage on the surfaces of the patella and the femoral condyles to soften and pit. With additional time, the softened cartilage will begin to erode exposing the underlying bone and induce scar tissue formation.

Many approaches to treating PCL injuries have been studied in an attempt to restore posterior translation to normal. Non-surgical (conservative) approaches have not been successful because the abnormal posterior translation is not eliminated. Conservative treatment focuses on strengthening the muscles surrounding the knee and maintaining the knee’s range of motion. The early subjective results are good, but the objective results are poor. With time, both the subjective and objective results worsen, and degenerative joint changes appear. The inability of the conservative treatment to restore the knee to normal has lead clinicians to pursue surgical reconstruction.

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3 definition found in APPENDIX A  
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Advances in PCL reconstruction have been based on concepts and techniques developed for achieving good results with ACL reconstruction,[71] in particular the placement of a grafts to behave isometrically[35, 71]. Unfortunately, the use of isometric\(^6\) placement has not been successful for the PCL.[44, 105, 106] The isometric reconstruction is able to control posterior translation from 0 to 45° of flexion, but not at higher flexion angles[20, 48, 105, 111] where the PCL provides over 90% of the resistive force[23]. The failure of isometric reconstruction could be due to the fact that only a small fraction of PCL fibers (5 to 15%) are believed to behave isometrically.[35, 108, 125] The flawed isometric reconstruction focused research into finding a femoral attachment site that was representative of the native PCL.

The anatomic one-bundle reconstruction placed the femoral attachment site within the anterolateral band of the native PCL for several reasons.[2, 60, 75, 108] First, the AL band of the PCL has been shown to be taut in flexion, [45, 55] which correspond to the region where the native PCL resists over 90% of the force needed to resist posterior tibial translation[23, 115, 137]. The AL band has also been shown to have increasing fiber length with tension, which has also been found for a majority of the PCL [34, 42, 123]. Initially, the anatomic one-bundle reconstruction controlled the posterior translation over the entire range of knee flexion,[3, 12, 27, 48, 108] but with time, the abnormal posterior translation frequently returned [63, 65, 85, 90]. The return of posterior translation has been attributed to improper graft pretensioning,[48, 81, 85, 87] improper graft location [48, 81] and/or graft elongation [48, 81, 85]. The failure of the anatomic reconstruction is believed to be caused by graft elongation, [48, 81, 85] which results from high graft forces and/or loss of strength due to graft remodeling [81, 85]. Bergfeld et al.[8] showed that after only 72 cycle of a 150 N anterior-posterior force at 90° of flexion that the 10 mm patellar tendon graft would elongate and produce a 2 mm posterior translation increase.

\(^6\) definition found in APPENDIX A
Mehalik[87] reported that after 1000 cycles of flexion-extension with a 50 N applied posterior force the graft would elongate and produce a less than 2 mm posterior translation increase. The failure of the anatomic one-bundle PCL reconstruction to maintain posterior translation control prompted clinicians and researchers to investigate methods to reduce the graft tension, which could prevent graft elongation.

More recently, investigators have studied the biomechanics of two-bundle PCL reconstructions in the hope that two bundles would reduce the local graft tension by improving the nonuniform load distribution and improve long-term control of posterior translation. The studies showed that a two-bundle reconstruction could control posterior translation over the entire range of flexion.[65, 81, 119] The attachment location of second bundle did not affect posterior translation control, but it did influence the load distribution between the bundles.[81] Mannor and colleagues[81] showed that the relationship between bundle tension and flexion angle depended on femoral bundle placement. Mannor et al.[81] produce a reconstruction that exhibited cooperative load sharing (shallow\textsuperscript{7}-shallow) and reciprocal load sharing (shallow-deep\textsuperscript{8}), Figure A.1. The study of Mannor et al. [81] showed that high forces occurred in the bundles at the extremes of motion. The initial results from the two-bundle PCL reconstruction showed promise to increase the reconstruction’s ability to resist the return of posterior translation over time. The purpose of this research was to investigate the ability of one and two-bundle PCL reconstructions to resist the return of posterior translation during a cyclic fatigue test.

\textsuperscript{7} definition found in APPENDIX A
\textsuperscript{8} definition found in APPENDIX A
CHAPTER 2

LITERATURE REVIEW

2.1 ANATOMY

The PCL attaches to the lateral\textsuperscript{9} wall of the medial\textsuperscript{10} femoral condyle, Figure 2.1, and to the posterior surface of the proximal\textsuperscript{11} tibia, Figure 2.2. The femoral attachment of the PCL has a tear drop shape oriented in the anterior\textsuperscript{12}-posterior direction.[32, 45, 51, 64, 68] The femoral anterior fibers attach to the roof of the intercondylar notch [34, 51, 64, 68, 123] while the posterior fibers attach approximately 5 mm anterior to the posterior articular cartilage margin.[123] The distance from the anterior to posterior border of the PCL is 20.9 to 33.0 mm.[34, 51, 68, 88, 114] The distal\textsuperscript{13} boundary of the PCL lies adjacent to the articular cartilage margin [34, 51, 64, 68] until it reaches the center of the femoral footprint,[123] and then the distal border proceeds posterior and proximal until it intersects with the proximal boundary at the point of the tear drop.[34, 45, 51, 64] The proximal edge of the PCL is almost horizontal when the knee is near full extension.[34, 45, 51, 68] The distance from the distal to proximal boundary of the PCL is 12.2 to 14.0 mm.[34, 68, 114]

\textsuperscript{9} definition found in APPENDIX A
\textsuperscript{10} definition found in APPENDIX A
\textsuperscript{11} definition found in APPENDIX A
\textsuperscript{12} definition found in APPENDIX A
\textsuperscript{13} definition found in APPENDIX A
The tibial insertion of the PCL is located posterior and proximal to the tibial plateau, Figure 2.2. The PCL inserts into a sloped recess that is centered between the medial and lateral tibial plateaus.[2, 12, 32, 34, 38, 45, 51, 64] The shape of the insertion site has been described as a rectangle with rounded corners.[45, 64, 68] The distance from the medial to lateral boundary is 13.0 to 15.3 mm,[51, 68] and the distance from the proximal to distal boundary is 10.1 to 14.6 mm.[34, 68]

Figure 2.2: Posterior view at full extension (left) and sagittal view at approximately 90° (right)[1]

The PCL travels posteriorly and inferiorly with a slight lateral deviation from the femoral to tibial insertion, Figure 2.2.[32, 36, 58, 102, 137] A sagittal plane projection of the PCL reveals that the PCL elevates 57° off the anterior-posterior direction at full extension.[102] The anterior fibers of the femoral footprint attach to the lateral aspect of the tibial footprint, and the posterior fibers of the femoral footprint attach to the medial aspect of the tibial footprint.[68, 75,

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13 definition found in APPENDIX A
14 definition found in APPENDIX A
The average length of the fibers is 38 mm [34, 51, 64, 102, 114] with the longest fibers being 41 mm. The longest fibers are found in the posterior proximal corner of the PCL and have a more oblique path, more medial lateral deviation.[42]

The cross sectional area of the PCL varies with position along the length. The midsubstance of the PCL is 13 to 14 mm wide [34, 51, 64, 102, 114] with a thickness of 6 mm.[102] From the midsubstance of the PCL, the cross sectional area increases in both the superior and inferior directions with the increase being more pronounced in the superior direction.[51, 64] The cross sectional area of the attachment sites are approximately three times the cross sectional area of the midsubstance (approximately 50 mm² [62]) with the femoral attachment area being the largest.[64]

Within the PCL, prior studies have found different fiber bundles. The classification method used to identify the bundles has varied from anatomic to functional. The anatomic classification is based on a distinct separation within the femoral footprint producing the anterolateral (AL) and the posteromedial (PM) bundles, but this distinct division has not been identified in all knees.[3, 35, 38, 51, 75, 89, 94, 123, 126, 137] The functional classification bases the bundle distinction on what range of the flexion-extension cycle the fiber region is taut. Fibers in the PCL can tense in flexion, tense in extension or remain at a constant tension (isometric bundle) throughout the entire range of flexion.[3, 9, 35, 43, 47, 55, 60, 64, 65, 68, 75, 78, 88, 89, 95, 117, 140]

### 2.1.1 Synovial Membrane

The PCL is located within the intercondylar notch of the femur, but the PCL is considered an extraarticular ligament because it is surrounded by a synovial sheath, which separates the PCL from the joint space.[34, 36, 64, 89, 137] The sheath on the medial, lateral
and anterior aspects of the ligament originates from the posterior capsule,[34, 36, 64, 137] and the inferior, posterior aspect of the PCL integrates into the posterior capsule as well as the periosteum (the membrane surrounding bone).[64, 137]

2.1.2 Vascular Supply

The vascular supply for the PCL passes through the surrounding synovial sheath. Transverse vessels from the sheath connect to vessels within the PCL that are parallel to the long axis of the ligament.[34, 137] The vascular network of the PCL innervates all but three regions of the ligament. The area near each attachment site and the mids substantive of the ligament are avascular.[113, 137] The vascular supply for the synovial membrane mainly originates from the middle genicular artery [34, 88, 89, 113] with auxiliary supply from the supreme genicular artery, medial and lateral superior genicular arteries, the anterior and posterior tibial recurrent, the descending branches of the lateral femoral circumflex, and the recurrent fibular arteries.[137] The vascular supply of the PCL is larger than that of the ACL,[34] which could lead to better natural healing in the PCL as compared to the ACL.

2.1.3 Lymphatic Supply

The lymphatic system aids in maintaining the homeostasis of the tissue. The lymphatic system has architecture similar to that of the vascular system including three regions with no lymphatic ducts. The lymphatic system helps to maintain a local pressure distribution by moving water and proteins out of the interfibrillar space as the PCL becomes taut. In addition to helping with the local pressure distribution, the lymphatic system removes proteins that cannot be absorbed and used by the cells or venules.[113]
2.1.4 Neural Supply

Nerves can be found throughout the entire PCL, but the majority of the nerves are concentrated at the superior portion of the ligament.[137] As with the vascular supply, the nerves that innervate the PCL pass through the synovial sheath. The posterior articular nerve, the largest nerve supplying the knee, not only has branches within the PCL,[34, 88, 137] but it also has branches in the oblique popliteal ligament, the posterior capsule, and the menisci.[137] With the posterior articular nerve innervating these structures, one may speculate that the neural connectivity may provide a signal pathway between the structures so that when an injury occurs, a compensatory response may begin. In addition to the posterior articular nerve, the posterior portion of the PCL has branches from the posterior tibial nerve as well as the obturator nerve.[33, 137] The anterior portion of the PCL is innervated by branches from the femoral, common peroneal and saphenous nerves.[137]

The neural supply provides many receptors that send information to the central nervous system (CNS). Within the PCL, Golgi-like receptors have been found. The Golgi-like receptors, which are located primarily near the femoral attachment, resemble receptors found in tendon, and they provide tension information to the CNS. The Golgi-like receptors are found on the surface of the ligament beneath the synovial sheath, and they are oriented along the long axis of the ligament.[34, 137] The Golgi-like receptors are responsible for reporting the motion of the knee joint to the CNS by monitoring the degree of the stretch of the ligament.[83] Additional information about knee motion is also supplied by Ruffini corpuscles (pressure receptors), Vater-Pacini corpuscles (velocity receptors), and free nerve endings (pain receptors).[34, 89]
2.1.5 Ultrastructure

The PCL is a composite structure containing water and several types of protein. The water residing within the PCL accounts for about 60 to 80% of the wet weight, and the main dry component of the PCL is collagen, comprising 70 to 80% of the dry weight. The collagen fibrils provide the major restraint to tensile loading in the ligament. The primary type of collagen found in the PCL is Type I. In addition to Type I, there are several other types of collagen (Type III and Type V) that are responsible for ligament healing and for fibril diameter regulation. In addition to collagen, other proteins, such as elastin, reticulum, and core proteins of the proteoglycan (PG) are found in the PCL. The elastin fibers are used to restore the spatial orientation of the collagen fibrils when the tension on the ligament is removed. The reticular fibers are chemically similar to collagen, but they exhibit different mechanical properties. The reticular fibers form a network that supports the blood vessels within the ligament.

The majority of water present in the PCL is held by glycosaminoglycans (GAG) molecules. The GAG molecules attach to core proteins forming PG molecules. The GAG content in ligaments is larger than that of tendons. The higher GAG content in ligaments leads to a greater amount of water, which affects the viscoelastic properties. The viscoelastic properties are attributed to the presence of both fluid and solid properties in the tissue. The water also provides a shock absorbing property when a compressive force is applied to the ligament. This property is important to the PCL because as the knee nears full extension the anterior surface of the PCL and the posterior surface of the ACL are forced together.

The collagen fibrils and the elastin fibrils form a three dimensional matrix with the GAGs filling the interfibrillar space. The collagen fibrils are oriented along the long axis of the PCL.
while the elastin fibrils are oriented to connect one collagen fibril to the adjacent collagen fibrils. The spatial relationship between the collagen fibrils is regulated by the elastin fibrils [83] and the GAGs [13, 15]. The circular collagen fibrils, 0.89 aspect ratio (minor to major axis) in a transverse section, occupy approximately $\frac{1}{2}$ of the total area of the PCL [6]. The percentage area (transverse section) occupied by collagen fibrils decreases slightly with increasing distance from the femoral attachment of the PCL, superior: 0.49; middle: 0.48; inferior: 0.46. The average diameter of the collagen fibrils also decreases with increasing distance from the femoral attachment of the PCL, superior: 90.4 nm; middle: 75.1 nm; inferior: 60.8 nm [6].

Researchers have hypothesized that the diameter of the collagen fibril will determine the role of the fibril in the ligament. In the superior portion of the PCL, the fibril diameters are larger, and the inferior portion has smaller diameters fibrils [64]. The larger diameter fibrils provide the resistance to tensile loading because of the greater number of intrafibrillar crosslinks [13, 14]. The number of intrafibrillar crosslinks is greater because larger diameter fibrils are composed of more collagen molecules, and the molecules are held together by the intrafibrillar crosslinks. The smaller diameter fibrils have been associated with creep resistance and ligament stiffness due to the increased number of interfibrillar crosslinks and crosslinking with GAGs [13, 14].

2.1.6 Properties

The mechanical properties of the PCL are correlated to the ultrastructure of the ligament. Historically, the PCL was believed to be twice as strong as the ACL [24, 43, 64, 89] but the PCL has been found to be only slightly stronger than the ACL [43, 44, 89]. The ultimate strength of the ACL has been reported to be 2160 N [43], and the ultimate strength of the PCL has been reported to be between 1500 and 2000 N [7, 32, 64, 86, 118]. The discrepancy in values may be
attributed to the age of the specimen and the methods used to test the ligament. Within the PCL, the ultimate strength of the two bundles is significantly different. The AL bundle (1120 to 1620 N) is more than twice as strong as the PM bundle (258 to 450 N).\[64, 88, 118\] The strength of the two bundles added together is greater than that of the PCL. The greater strength is due to the difference in orientation used during testing. When the PCL is tested as a whole, neither of the two bundles are oriented optimally to resist the tensile loads, but when the bundle are tested separately, they are oriented to optimally resist the tensile loads.

The PCL is also more compliant than the ACL because the PCL has less small diameter fibrils. The stiffness of the PCL ranges from 145 to 254 N/mm.\[64, 118\] The stiffness of the two bundles of the PCL is also significantly different. The AL bundle (120 to 347 N/mm) is more than twice as stiff as the PM bundle (57 to 77 N/mm).\[64, 92, 118\]

2.2 FUNCTION

Historically, the PCL has been viewed as a relatively unimportant ligament with little disability as a result of injury.\[31, 135\] With more recent study, however, the PCL has been identified as one of the most important structures in the knee,\[29\] and normal knee function requires the presence of the PCL\[31\]. The primary role of the PCL is to resist posterior tibial translation at all knee flexion angles.\[3, 12, 20, 23, 27, 31, 32, 40, 43, 53, 56, 58, 60, 68, 72, 76, 78, 86, 88-90, 111, 114, 115, 126, 137\] The PCL also plays a secondary role in resisting varus-valgus and external rotation.\[12, 32, 43, 60\] The PCL in combination with the ACL provides the screw home mechanism near extension, or guides the motion of the knee.\[43, 88, 89\] The PCL has also been identified as the primary joint stabilizer \[29, 41, 74, 76, 78, 89, 111, 135, 137, 139, \]
because of its tensile strength, cross sectional area and location near the central axis of the knee.

Posterior tibial translation is resisted by many structures within the knee, but the primary restraint is the PCL, which provides over 90% of the force needed to restrain posterior translation. With the PCL intact, no other structure in the knee provides more than 2% of the restraining force. With a PCL deficient knee, the posterolateral capsule and the popliteus tendon provide the primary restraint to posterior translation (58.2%). The importance of the popliteus tendon as a secondary restraint may be due to its orientation in the knee, which is parallel to the PCL in the sagittal plane. The other secondary restraints after PCL disruption are the medial collateral ligament (15.7%), the posteromedial capsule (6.9%), the lateral collateral ligament (6.3%), and the mid-medial capsule (6.2%). Other studies have identified additional structures such as the meniscofemoral ligament (MFL) and the bony geometry of the knee as possible secondary restraints. The restraint attributed to the MFL is due to its position adjacent to the anterior and/or the posterior aspect of the PCL and its orientation, which follows a path similar to PCL, in the knee. The role of the bony geometry has been controversial. The articulating surface of the femur is round while the articulating surface of the tibia is flat. The incongruity would provide little resistance to posterior translation, but the menisci, which provide a transition from one surface to the other, produce a more stable geometry by increasing the amount of contact area between the tibia and the femur. Even with the increased contact area, the knee geometry would provide little resistance to posterior translation.

The fibers in the PCL change length as the knee is flexed and extended. The femoral attachment location (footprint) of a fiber has the greatest effect on its length through the flexion-
Changes within the tibial footprint produce little to no change in fiber length behavior. Within the femoral footprint, the direction that provides the greatest change in fiber length has been disputed. A number of researchers have shown that a change in the proximal-distal direction causes the greatest length change, but there are others who have shown that it is the anterior-posterior direction. Some of the studies that advocate the anterior-posterior direction have used a coordinate system that is rotated 90°, and they were actually finding that the proximal-distal direction was the most important. The studies have also shown that distal fibers lengthen with flexion, and proximal fibers shorten with flexion. In addition to the fibers that change length, an isometric region, where little or no change occurs, has been identified.

The region of the flexion-extension cycle in which the fibers are functional is dependent on the fiber length. One theory on fiber function is that the length between the femoral attachment site and the tibial attachment site has to be at or beyond the just-taut length. Another theory is that the fiber length has to be within 5% of the maximum length of the fiber to be functional. Despite the differing theories on the relationship of fiber function and fiber length, most researchers feel that the bulk of the PCL is functional in flexion. The AL bundle of the PCL is also functional in flexion, but the functionality of the PM bundle is not readily agreed upon. Historically, the PM bundle has been believed to function in extension. However, some studies have found that the PM bundle is functional in flexion. The differences in reported functionality are due to different locations for the center of the PM
bundle. If the center of the PM bundle is more distal within the femoral footprint, the functional region of the PM bundle would shift to a higher flexion angle because the fibers in a more distal position will be at their maximum later in the flexion cycle.

2.3 PCL INJURY

Injuries to the PCL are less common than those of the ACL. Approximately 500,000 significant injuries to the ACL occur in the US, annually.[64] The ACL is involved in more than 60% of all knee ligament injuries[91] in the general population while the PCL is involved in only 7% of all knee ligament injuries.[91] One possible reason that the PCL may be injured less than the ACL is that the PCL is biomechanically stronger[24, 43, 44, 60, 64, 76, 88, 89] and anatomically larger than the ACL [60, 62, 88]. Another possible reason for the lower injury rate is the number of misdiagnosed PCL injuries.[4, 27, 37, 64, 78, 127] Several clinical studies have reported that a correct diagnosis for a PCL injury was found less than 20% of the time.[37, 78]

2.3.1 Injury Mechanism

Until recently, motor vehicle accidents were the leading cause of PCL injuries with sports being the second leading cause.[30, 38, 78, 139] It was believed that 50% of PCL injuries were caused by motor vehicle accident, 40% sports and 10% other,[30] but recent studies have shown that sports related injuries are becoming the leading type of injury [17, 38]. An injury to the PCL is a result of the PCL fibers being elongated beyond their yield point[38] leading to serial failure of the tissue. The elongation happens when the knee motion, typically posterior translation in case of PCL, is forced past the normal limits.[105]

Several mechanisms, which produce elongation of the PCL, have been identified in sports related injuries. One mechanism is a fall on the flexed knee with the foot plantar flexed, toes
pointing inferiorly, with the impact near the tibial tubercle.[4, 30, 40, 86] This mechanism results in an interstitial tear with the severity of the tear depending on the level of impact.[4, 30] Another mechanism producing interstitial tears is when the knee is forced into hyperflexion.[4, 40, 86, 90, 137, 139] In addition, an athlete that plants his/her foot and quickly rotates the tibia in an external, varus or valgus direction could rupture the PCL after the primary restraint was ruptured.[4, 36] Knee dislocations during athletic participation are rare occurrences, but they are extremely debilitating injuries.[36, 58, 90] A dislocated knee will tend to have ACL, PCL, and capsular damage with the possibility of other ligamentous injury and neural and/or vascular damage.

The common mechanism for a PCL injury for a passenger of motor vehicles is a dashboard injury.[4, 30, 33, 40, 76, 139] With the knee flexed, a posterior force is applied to the proximal tibia as the knee contacts the dashboard.[4, 10, 30, 86, 89] This mechanism will result in either an isolated PCL disruption or a tibial avulsion,[4, 10, 30, 76] where the ligament pulls away from the attachment site, or a piece of the bony attachment will be displaced from the attachment site.[76]

Several other injury mechanisms have been identified. A sudden hyperextension of the knee results in a tear in the PCL [4, 30, 36, 38, 89, 90, 105, 137, 139] only after the posterior capsule and the ACL have been torn [4, 30, 38, 105]. Two other mechanisms can cause injury to the PCL, but the amount of the externally applied load must be high. An inferiorly directed force applied to the distal femur while the knee is flexed can injury the PCL because the femur is forced anteriorly while the tibia remains stationary.[33] This is the same as forcing the tibia posterior in which the PCL provides the primary restraint to posterior tibial translation.
2.3.2 Injury Classification

Injuries of the PCL are classified based on the extent of damage. Two clinical examinations are often used to diagnosis the presence of a PCL injury. The posterior drawer test is performed by applying a posterior force to the flexed knee, and the sag test is a passive test performed in flexion. The tests are performed in flexion because the PCL provides approximately 95% of the force required to resist posterior translation in flexion. A grade I injury is indicative of a partial tear. The posterior drawer test reveals less than 5 mm of posterior translation compared to the uninjured knee, and the sag test shows that the anterior surface of the tibial plateau is still anterior to the anterior surface of the femoral condyles. A grade II injury is indicative of a more severe partial tear. The posterior drawer test reveals between 5 and 10 mm of posterior translation compared to the uninjured knee, and the sag test shows that the anterior surface of the tibial plateau is now even with the anterior surface of the femoral condyles. A grade III injury is indicative of a complete tear. The posterior drawer test reveals more than 10 mm of posterior translation compared to the uninjured knee, and the sag test shows that the anterior surface of the tibial plateau has moved posterior to the anterior surface of the femoral condyles.

2.3.3 Effects of PCL injury

With the importance of the PCL to normal knee function, an injury can affect the ability of a person to perform activities of daily life. In addition to affecting daily life, athletes may have their careers ended or significantly reduced. The symptoms that appear after PCL injury include pain, instability, “giving way”, and locking. Pain develops posterior to the patella after the person walks long distances, descends stairs, and/or squats.
The development of pain behind the patella may be caused by the change in the extensor mechanism of the leg. The extensor mechanism is composed of the quadriceps muscles on the anterior aspect of the femur, the quadriceps tendon, the patella, and the patellar tendon (PT). With the PCL injured, the tibia is posterior to its normal anatomic position. This puts the extensor mechanism at a disadvantage, which leads to increased contact pressure between the patella and the femur producing knee pain. Another early symptom of PCL injury is a feeling of knee instability. People feel that their knee is “giving out” when they are walking on uneven ground, running, descending stairs, and/or changing directions. People may also experience locking of the knee and swelling.

As time passes after the PCL injury, additional symptoms can develop anywhere from 3 months to 5 years. PCL injury affects the joint kinematics, which can lead to progressive damage to other knee ligaments. Researchers have shown that the ultimate strength of the ACL decreases after PCL rupture. The loss in the ultimate strength of the ACL is attributed to a change in physiological tension. With the PCL ruptured, the tibia is posterior to its normal anatomic position, and the distance between the attachment sites and the physiological tension of the ACL decreases. With the reduction in tension, the ACL will remodel to accommodate the new knee geometry. The number of collagen fibrils will decrease lowering the ultimate strength. In addition to altering the ACL, the secondary restraints to posterior translation will also undergo modification because they are required to control posterior translation.

The abnormal joint kinematics not only damages other ligaments, but it also increases the contact pressure in the tibiofemoral and patellofemoral joints. The increased
contact pressure in the two joints leads to damage of the articular cartilage.[64, 79] The amount of damage seen in radiographs increases with time from injury,[17, 73, 82] but the actual articular cartilage damage has been shown to be more extensive than seen in the radiographs [29, 49, 73]. These degenerative cartilage changes are found in the patellofemoral joint[4, 36, 38, 64, 70, 74, 82] as well as in the medial [4, 29, 36, 64, 74, 77, 79, 82] and lateral [36, 74, 79] compartments of the tibiofemoral joint with the degenerative changes in the tibiofemoral joint being more prevalent in the medial compartment.[29, 49, 77, 82] The medial femoral condyle is more rounded than the lateral femoral condyle, and the medial meniscus is also less mobile than the lateral meniscus. With the tibia in a more posterior position, the medial meniscus is unable to slide anteriorly and increase the contact area, thus increasing the contact pressure in the medial compartment.[79] With time, the increased contact pressure erodes the articular cartilage leading to permanent joint pain[38, 41] requiring intervention (total knee replacement) to alleviate the symptoms.

2.4 PCL TREATMENT

The treatment of PCL injuries has ranged from non-surgical to surgical treatment. Non-surgical (conservative) treatment was first used because some clinicians felt that an injured PCL did not adversely affect the knee.[31, 72] Since that time, the PCL has been recognized as an important knee structure, but conservative treatment has still been advocated in certain situations. Conservative treatment has been used to treat isolated injuries,[16, 38, 60, 73, 90, 133] chronic injuries, and grade I or II injuries [60, 75, 90, 114]. Surgical repair of the PCL has been advocated because of the poor outcome for the conservative treatment [16, 38, 65, 71, 73, 77, 90, 121, 129] and the success of ACL surgical treatment[35, 71]. Surgical repair has been used to
treat combined PCL injuries,[27, 110, 114, 133] acute injuries,[27, 60, 73, 100, 103, 110] chronic injuries,[27, 75] and grade III injuries [27, 60, 75, 110, 114].

Conservative treatment has been used to treat many types of injuries because the patients reported that their knees felt better after treatment.[40, 71, 77, 126, 127, 129, 135] However, the main reason for the use of conservative treatment is because the success rate for surgical intervention has been poor [28, 48, 65, 71, 75, 76, 85, 89, 95, 106, 111, 131, 132]. Historically, conservative treatment was nothing more than immobilization of the joint to allow time for the PCL to heal.[59, 60] With the vascular supply, healing of the PCL is not impossible, but the quality of the healing has been poor.[25, 43] The poor healing could be attributed to avascular regions within the PCL,[113] which would prevent factors necessary for healing (nutrients, growth factors, etc.) from reaching the injury site. To compensate for the poor quality of the healed PCL, patients were fitted with braces to compensate for the lost function.[59]

Conservative treatment has evolved a great deal over time. The treatment now requires maintaining a full range of knee motion,[3, 72, 110, 114] which was necessitated by several studies that found a decrease in the mechanical properties of collagenous tissue with long-term immobilization.[99, 141-143] In addition to maintaining motion, the muscles around the knee, especially the quadriceps muscle, are strengthened to protect the injured PCL.[3, 60, 71, 73, 114, 126, 127] The quadriceps muscle is the focus of the strengthening protocol because when the quadriceps muscle is taut, the patellar tendon, which has an anteriorly directed force component, also becomes taut. The anteriorly directed force component of the taut patellar tendon can resist some of the abnormal posterior translation.

Most patients report that they feel the injured knee is better after conservative treatment than before the treatment began, but the outcome of the clinical quantitative examinations are
typically poor. Patients with isolated injuries usually do well and can continue with their level of activity, but other patients return to a lower level of athletic participation. However, athletic participation is accompanied by significant symptoms. Some researchers believe that the return to athletic participation and the improved subjective results for isolated injuries are attributed to the fact that the PCL heals with some residual laxity that is invariant over time. The healing has been shown by performing a posterior drawer test one day post injury and two weeks post injury, and the soft endpoint at day one changed to a firmed endpoint two weeks later. Grade III isolated injuries, however, tend not to do well because the injured knee develops medial and patellofemoral compartment chondrosis. A report from the Swiss workman’s compensation board has shown that patients treated conservatively had better subjective and objective results compared to those patients treated operatively, but these findings are not what is normally found with long term study of conservative treatment. The initial reports of good knee function by patients tends to deteriorate with time as the functionality of the knee is hampered by joint degeneration that can be identified as early as three months post injury. The objective findings that are initially poor also deteriorate with time. Even though patients initially feel that their knees have improved, the conservatively treated PCL injuries will follow a similar path as untreated PCL injuries resulting in the accelerated onset of osteoarthritis.

Surgical repair of the PCL, primary repair and reconstruction, has been advocated to stop or slow the onset of osteoarthritis. The short-term goals of surgical treatment are to restore the normal tibiofemoral stability and to return the patient to their preinjury level of activity without pain. If these short-term
goals are achieved, the long-term goal of limiting degenerative changes should also be accomplished.

The primary repair of an interstitial tear failed to fulfill the goals of surgical repair.[28, 78, 89, 121, 132] The premise of the primary repair is to suture the ends of the ruptured PCL together [121] so that the ends can reattach during the healing process. Injuries that occur near the attachment sites respond better to primary repair than do injuries that are located in the midsubstance of the tissue.[78, 121] The site dependence may be due to the regional vascular supply. The increased vascular supply near the attachment sites as compared to the midsubstance of the PCL may explain why the primary repair near the attachment areas are better, but even primary repair in these locations has still resulted in poor outcomes with a failure to restore functional stability.[28, 78, 121, 132] One reason for the poor outcomes is the lack of initial strength of the repair, which requires knee immobilization to protect the repair,[121] but joint immobilization has been shown to lead to tissue degradation.[99, 141-143]

2.5 PCL RECONSTRUCTION

Surgical reconstruction of the injured PCL has the potential to provide the most successful outcome. The advantage of a reconstructive treatment is that the damaged PCL is replaced with a graft that requires no interstitial healing to be functional so early knee motion would not endanger the repair. Graft material used for reconstructions has ranged from autografts (from the patient’s body) to allografts (from a different individual of the same species). The patellar tendon (PT),[3, 27, 60, 74, 100, 135] hamstrings tendon [27, 60, 74, 100, 135] and quadriceps tendon [60] are some of the tissues harvested as autografts. Since the graft is taken from the same body, there will be no immune response, where the immune system
attacks and attempts to destroy the foreign body. The PT graft also has the advantage of having bone at both ends, which allows for secure fixation and facilitates the fastest healing for the bone tunnel because of the bone-bone contact. The PT graft also provides greater strength than the PCL, however, the PT graft is not without its disadvantages. The PT graft is difficult to position in the knee, the bony ends cause a large amount of donor site morbidity, and the rectangular shaped graft does not fill the circular tunnel with collagenous fibers.

Other autologous materials present different sets of advantages and disadvantages. The hamstrings tendon and the quadriceps tendon produce less donor site morbidity because the grafts only have bone at one end, but their fixation is less secure because the soft tissue end must heal to bone. The autologous grafts will undergo a remodeling process, including a necrosis phase to adapt to the new set of demands. The remodeling process weakens the graft, which makes the graft more susceptible to elongation and failure during rehabilitation.

Allografts provided an alternative source of graft material for PCL reconstruction. Allografts are readily available and most importantly reduce the amount of trauma to the patient because an otherwise healthy structure is not damaged to provide the graft. Many allografts have been used for reconstruction, but the most popular allograft is the Achilles tendon. The Achilles tendon has sufficient length, is initially stronger than the PCL and is easily passed from tibia to femur. However, the Achilles tendon allograft also has a down side. To prepare the allograft for implantation, it has to be sterilized to prevent disease transmission. The common method used for sterilization is gamma irradiation. The gamma irradiation needed to completely
prevent the disease transmission and the immunogenic response causes damage to the tissue and reduces the initial strength to a level below that of the PCL.[19, 39, 43, 50, 120, 124, 138, 142] However, researchers have recently found that a 2 Mrad gamma irradiation level would significantly reduce the immunogenic response and the likelihood of disease transmission, but the strength of the allograft is not significantly reduced.[39, 50, 120, 138]

2.5.1 Isometric reconstruction

An isometric reconstruction was first advocated because of the success of ACL reconstruction. The theory behind ACL reconstruction is that the central fibers are isometric.[35, 71] In the last 15 years, isometric ACL reconstructions have produced results that are predictable and reproducible with several clinicians reporting a good to excellent outcome in over 90% of the patients.[71] The success of the isometric reconstruction is based on the lack of length change for the fibers as the knee is cycled through flexion-extension.[42, 68, 108, 112] This lack of length change results in the generation of forces that are below the yield point of the graft fibers.[18, 20, 42, 68] These forces will not compromise the graft fibers [18, 42] and the graft fixation. Therefore, early postoperative mobilization, which aids in healing, is allowed.[42]

The problem with an isometric PCL reconstruction is that the precise location of the isometric region is not known. An isometric region is defined as “a pair of points on opposite sides of the joint that remain equidistant as the knee goes through a normal range of motion”,[12] but this strict definition is not used experimentally. Researchers feel that a change of 2 mm or less is indicative of an isometric region.[12, 35, 112] Given this criterion, many researchers have attempted to define the isometric region of the PCL. The majority of the studies have placed the isometric region along the proximal border of the PCL, but the anterior-posterior location is disputed.[2, 12, 35, 44, 46, 55, 68, 71, 105, 107, 108, 111, 112, 123, 125, 131] The exact
location may depend on the types of loads used during testing [55, 108] and the state of the knee (intact, PCL deficient, or reconstructed).[48]

The results of an isometric reconstruction are poor in comparison to ACL reconstructions. Clinicians have found that most of the subjective and objective results would be placed in the good or fair categories.[44, 105, 106] However when an isometric ACL reconstruction is performed, 90% of the patients were rated in the good or excellent categories.[71] One of the reasons that the results were below expectation is that normal kinematics were not restored. The strain patterns found in the reconstructed knee were different than those found in the intact knee,[12] and the reconstructed knees did not return control of posterior translation beyond 45° of flexion. However for flexion angles between 0 and 45°, posterior translation was not significantly different than the intact knee.[20, 48, 105, 111] Some patients also lost range of motion in flexion, which varied from as little as 5° to as much as 30°.[105, 106] In addition to the abnormal knee kinematics, the majority of patients could not return to their preinjury level of athletic participation, and degenerative changes were found at follow up.[105]

One problem with isometric PCL reconstruction is that the bulk of the PCL fibers change length as the knee is flexed and extended.[3, 35, 75, 107, 108, 125] In addition, the isometric region of the PCL constitutes only 5 to 15% of the bulk of the ligament.[35, 108, 125] In addition to replacing only a small portion of the native PCL, the isometric reconstruction places the graft in a more vertical orientation and is less capable of resisting posterior translation.[125]

2.5.2 Anatomic Reconstruction

Clinicians and researchers have advocated an anatomic reconstruction technique in an attempt to alleviate the problems found using an isometric reconstruction. This technique is
aimed at restoring a portion of the normal PCL anatomy that will reproduce the length behavior of the native PCL fibers.[2, 3, 35, 75, 107, 108, 125] The location of the fibers that produce this behavior has been a topic of extensive research. The research has shown that a graft centered in the anterolateral (AL) portion of the PCL will lengthen with flexion as does the majority of the PCL.[2, 60, 107, 108] In addition to having the appropriate length behavior, the AL bundle also exhibits several other characteristics that make it a more appropriate graft location. The AL bundle is nearly double the size of the posteromedial (PM) bundle.[41, 64, 88] The AL bundle is also stiffer [41, 60, 64, 88] and stronger [60, 64, 88] than the PM bundle. With these attributes, researchers and clinicians have identified the AL bundle as the most important functional component of the PCL.[60, 75]

The results of an anatomic reconstruction have been better than those of an isometric reconstruction initially, but with time after surgery, the results have deteriorated. Clinicians have found that 75% of the overall results would be placed in the excellent and good categories for short follow up periods,[27] but with a longer follow up period, only 23% of the overall ratings were in the excellent or good categories.[75] The initial encouraging results can be attributed to the improved knee kinematics. The strain patterns found in the reconstructed knee were not different than those found in the intact knee,[12] and the reconstruction also returned control of posterior translation to the knee at all flexion angles,[3, 12, 27, 48, 108] and only minor reductions in the range of motion were found.[27] In addition to the return of the nearly normal knee kinematics, the majority of patients returned to their preinjury level of athletic participation initially, but this number was reduced to about 50% with a longer follow up.[75]

The problem with anatomic reconstructions is that the results deteriorate with time after surgery. The deterioration in the results has been linked to the recurrence of the abnormal
posterior translation [63, 65, 85, 90] which maybe caused by improper tensioning initially,[48, 81, 85, 87] improper graft placement,[48, 81] , and/or graft elongation.[48, 81, 85] Graft elongation has been identified as the primary mechanism for failure, which has been linked with high graft forces. The high graft forces may be attributed to improper protection of the graft during the initial healing stage, to nonuniform load distribution, or both.[81, 85]

2.5.3 Two-bundle Reconstruction

A two-bundle reconstruction has recently been advocated because it can overcome some of the problems seen with the one-bundle reconstructions (isometric and anatomic). The two separate grafts allow the surgeon to fill more of the native femoral footprint with collagenous tissue,[60, 71, 95] and the reconstructions could restore the normal kinematics of the knee.[65, 95, 122, 133] A two-bundle reconstruction can pair an isometric graft, which some feel is critical to the success of a reconstruction,[35, 107, 112] with an anatomic graft, which can control posterior translation in the flexed knee.[3, 12, 27, 48, 108] A two-bundle reconstruction can also pair two anatomic grafts together. Both of the combinations should reduce the demand on each bundle,[65, 81] and the forces should not exceed the yield point of the graft fibers, which causes graft elongation.

The current biomechanical studies of the two-bundle reconstruction have revealed several important themes. First, posterior translation control is independent of the combination, shallow-shallow or shallow-deep, used, Figure A.1.[65, 81, 119] Second, the force in the bundles is dependent on the femoral tunnel location. The force in the shallow bundles increases with increasing flexion angle, and the force in the deep bundles decreases with increasing flexion angle.[65, 81, 119] These studies have shown that a two-bundle reconstruction can initially control abnormal posterior translation and reproduce the loading patterns seen in the native PCL.
The loading patterns seen in the normal PCL are determined by the state of the PCL. If the PCL is tested as a single entity, the force increases with increasing flexion angle[26, 86, 136, 139] similar to the shallow-shallow configuration,[81] but if the PCL is separated into the AL and PM bundles, the loading pattern is reciprocal, one increases as the other decreases,[9, 34, 117, 118] which is similar to the shallow-deep configuration [81, 119]. The long-term effects of these different loading patterns have not been determined.

The clinical results of a two-bundle reconstruction have appeared only once in the literature.[140] The femoral tunnels for the two grafts were located to replace the AL and PM bundles. These locations were chosen because “both show different tension behavior in the knee movements and only in their combined function can stability in all joint positions be achieved”[140]. The grafts were obtained by releasing the tibial insertions of the semitendinosus and the gracilis tendons, but their attachment to the muscles, which are located around the femur, were maintained. This method of reconstruction is considered dynamic because the tension in the graft is a function of muscular activity. The success of this method requires that the muscle retains its strength so the graft can resist posterior translation. Subjectively, all of the patients were satisfied with the outcome, but the objective results were not as good. All of the knees had an increased posterior drawer compared to the contralateral knee, but the postoperative posterior drawer was less than the preoperative test.[140] The appearance of a positive posterior drawer may be misleading because of how the posterior drawer test is performed. The test is performed with the muscles relaxed, which allows slack in the graft, so the graft cannot resist the abnormal posterior translation.

With the exception of the abnormal posterior translation, the two-bundle graft seems to provide an adequate repair. The clinicians found minor limitations in the amount of knee motion.
in flexion, no more than 15°. All of the patients were able to return to sports, but 33% of the patients decided not to return to competitive athletics. This may signify that some of the abnormal posterior translation found in the drawer test was not caused by the testing method but was caused by an inadequacy in the reconstruction.

2.6 SUMMARY

PCL injuries impair a person’s ability to perform daily activities and can lead to long-term disability. However, repair of the injured PCL has not been successful because the repairs have not been able to restore and maintain the normal posterior translation limit, to return the preinjury level of athletic participation, and to slow or stop the onset of degenerative changes. Anatomic PCL reconstruction can restore the normal posterior translation limit and return athletic participation, but with time, the abnormal posterior translation frequently returns. Many factors such as improper graft pretension, improper graft location and graft elongation have been hypothesized as the mechanism for reconstruction failure, but graft elongation is believed to be the major contributing factor. Graft elongation is produce by applying high loads to a small portion of the graft (nonuniform load distribution) and/or loss of graft strength during remodeling. In an attempt to provide a more uniform load distribution, two-bundle PCL reconstruction has been pursued. The biomechanical study of the two-bundle reconstruction has shown that regardless of the bundle combination posterior translation is controlled over the entire range of motion, but some of the bundles have been shown to have high loads in the extremes of motion. However, there is no information about
how the different combinations and loading distributions can resist the return of posterior translation over time.
CHAPTER 3

HYPOTHESES

The goal of this study was to find a two-bundle PCL reconstruction that provides better restraint to posterior translation than the traditional one-bundle PCL reconstruction. The three hypotheses listed are the null hypotheses that serve as the basis for the three subsequent chapters. The first two hypotheses focus on characterizing the initial and long-term behavior of both one and two-bundle reconstructions. The last hypothesis was aimed at determining if the acute angle at the exit of the tibial tunnel is the primary cause for PCL reconstruction failure.

Several studies[65, 81, 119] have focused on two-bundle PCL reconstruction in an attempt to improve the nonuniform graft load distribution, which is believed to cause the failure of anatomic PCL reconstruction. Biomechanical studies have shown that a two-bundle reconstruction can control posterior translation over the entire range of knee motion regardless of the femoral attachment site of the second bundle when combined with the AL bundle, but the tension in the AL bundles is influenced by the position of the second bundle.[65, 81, 119] These studies have also shown high tension in the second bundle in deep flexion and near full extension.[81, 119]

**Hypothesis 1:** The addition and location of a second bundle paired with an AL bundle will be unable to reduce the tension in the AL bundle.

The failure of the anatomic reconstruction to resist posterior translation in the long-term has prompted researchers to examine the cyclic behavior of PCL reconstruction. Bergfeld and colleagues[8] examined the change in posterior translation for a one-bundle reconstruction, and
they found that after only 72 loading-unloading cycles at 90° of flexion that 2 mm of posterior translation had returned. Mehalik[87] also examined the cyclic behavior of a one-bundle PCL reconstruction. Mehalik[87] found an increase of approximately 2 mm of posterior translation while the knee was cycled from near full extension to 105° of flexion with an applied posterior load.

In addition to the cyclic behavior of PCL reconstructions researchers have also examined two-bundle PCL reconstructions in an attempt to improve graft load distribution. The studies have exhibited that two-bundle PCL reconstructions can return normal knee kinematics[65, 81] and more evenly distribute graft load,[81] but the long-term behavior of these reconstructions has not been studied.

**Hypothesis 2:** No two-bundle PCL reconstruction will resist the return of posterior translation for more cycles than the one-bundle PCL reconstruction during cyclic fatigue testing.

The tibial tunnel approach for PCL reconstruction has been questioned in the last decade because as the graft wraps around the tunnel exit, the graft can abrade and fail.[7, 89] This concern has lead to the naming of the tibial tunnel exit as the “killer turn”. [7, 90] The tunnel exit has also been theorized to hamper graft incorporation,[89, 130] to inhibit proper graft fiber orientation,[8, 130] and to alter the cellular differentiation and viability.[130] In addition, the tibial tunnel requires a large portion of tibial bone stock to be removed, which can also hamper graft incorporation.[7] Investigators feel that the tibial tunnel approach leads to poor placement,[8] makes graft passage difficult,[7] and shields the applied pretension from the intraarticular portion of the graft.[7, 8]
**Hypothesis 3:** The graft failure location will not exhibit a preferential region in which failure occurs.
CHAPTER 4

TWO-BUNDLE POSTERIOR CRUCIATE LIGAMENT RECONSTRUCTION: A STUDY OF HOW BUNDLE TENSION DEPENDS ON FEMORAL PLACEMENT

ABSTRACT

This study was designed to determine how the addition of a second bundle to an anterolateral (AL) one-bundle posterior cruciate ligament (PCL) reconstruction affects the tension in the AL bundle and to determine the effects of femoral location on the tension in the second bundle. The one and two-bundle reconstructions were performed on 19 cadaveric knees. The femoral attachment of the one-bundle (AL1) graft and one bundle of all two-bundle (AL2) grafts was located within the native PCL’s AL band. The second bundle of the two-bundle grafts was placed posterior to the AL2 bundle in one of three locations that varied in their depth within the notch: shallow (S), intermediate (I), and deep (D). The tibial end of the one and two-bundle grafts was placed at the center of the native PCL’s tibial attachment. Bundle tension and knee motion were measured as the knee was cycled from near full extension to 120° of flexion while a 100 N posterior force was applied. Placement of the second bundle in either the S or the I location significantly reduced the AL bundle tension. As the distance for the second bundle increased from the cartilage edge, the peak bundle tension and the rate of tension generation decreased. The S bundle required tension that was three times the applied force in deep flexion in order to reduce the AL bundle tension. The AL2-S and the AL2-I reconstructions exhibited load sharing (both bundles function together) while the AL2-D reconstruction exhibited reciprocal loading (one bundle functioning in flexion and one functioning in extension). The
load sharing characteristics for the AL2-I reconstruction were better than those for the AL2-S reconstruction because of the high tension in the S bundle after 90° of flexion.

**INTRODUCTION**

Between 5 and 20% of all knee ligament injuries are to the posterior cruciate ligament (PCL).[30, 64, 86] PCL injuries alter knee kinematics [4, 34, 64], which can inhibit activities of daily living such as climbing stairs and walking on inclined or declined slopes.[67, 100] Several treatment options have been used in an attempt to restore the normal knee kinematics with little success. Conservative treatment has been shown to improve the patient’s perception of the injury without improving the knee kinematics.[40, 77, 127] Osteoarthritic changes have also been reported to appear in as little as 3 months with conservative treatment.[4, 36] PCL reconstruction has been used because reconstruction has the potential to restore normal knee kinematics and to slow or stop the onset of the osteoarthritic changes.

PCL reconstructive procedures were originally based on the techniques used in anterior cruciate ligament (ACL) reconstruction because the ACL reconstruction was predictable and successful (over 90% of the patients were rated as good or excellent).[71] The reconstruction placed the femoral tunnel in the isometric region of the native ACL.[71] but when the femoral tunnel for the PCL reconstruction was placed in the isometric region, normal knee kinematics were not restored, particularly with flexion beyond 45°.[48, 111] The problem with isometric reconstruction is that the majority of the PCL fibers change length during the flexion-extension cycle with only 5 to 15% of the femoral footprint being isometric.[35, 108, 125] The inability of isometric reconstruction to restore the normal limit of posterior translation[48, 111] has lead researchers to study nonisometric reconstructions.
Typically, the nonisometric femoral tunnel was placed in the anterolateral (AL) region of the PCL. The AL bundle tightens with flexion,[2, 60] and the AL is larger and stronger than the posteromedial bundle of the PCL.[64, 88] The use of the nonisometric reconstruction initially restores normal knee kinematics.[12, 48] However with time, the abnormal posterior translation frequently returns,[65, 85, 90] which has been attributed to poor initial graft placement,[48, 81] inadequate graft pretension, and/ or graft elongation.[48, 81, 85] We hypothesize that graft elongation, which we attribute to a highly nonuniform distribution of force among graft fibers, is the primary mechanism of PCL reconstruction failure.

In an attempt to improve the success of the nonisometric reconstruction, several researchers have studied two-bundle PCL reconstructions.[65, 81, 119] The use of the second bundle could provide a more uniform load distribution and prolong the life of the graft because the orientation of the native PCL is better replicated.[60, 71] Biomechanical studies have shown that a two-bundle reconstruction can control posterior translation over the entire range of knee motion regardless of the femoral attachment site of the second bundle when combined with the AL bundle, but the tension in the AL bundles was influenced by the position of the second bundle.[65, 81, 119] Mannor et al.[81] has shown that the second bundle generate high tension in the extremes of motion. We feel that if a second bundle is placed in the midsubstance of the native PCL the peak tension in the bundle will be reduced. The purpose of this study was to determine how the femoral attachment location of the second bundle of a two-bundle PCL reconstruction affects the bundle tension in the AL bundle and the load distribution.
METHODS

Study Design

The change in bundle tension with knee flexion was investigated using both one-bundle and two-bundle patellar tendon (PT) grafts. The PCL reconstructions were performed on 19 unembalmed cadaveric lower limbs from 14 donors (4 male, 10 female) with a mean age of 70.4 years (age range: 50 - 88). The posterior motion limit for the intact and reconstructed knees was determined as the knee was cycled from near full extension to 120° of flexion while a 100 N posterior force was applied. The knee configurations tested included the intact knee, the PCL deficient knee (includes removal of the ligaments of Humphrey and Wrisberg when present), a one-bundle reconstruction, and three different two-bundle reconstructions, Figure 4.1. The femoral attachment of the one-bundle (AL1) graft and one bundle of all two-bundle (AL2) grafts were within the PCL’s anterolateral band, Figures 4.2 and 4.3. The second bundle of the two-bundle grafts was placed posterior to the AL2 bundle in one of three locations that varied in their depth within the notch: shallow (S), intermediate (I), and deep (D), Figures 4.2 and 4.3. The tibial end of the one and two bundle grafts was placed at the center of the PCL’s tibial attachment. The reconstructions were pretensioned to restore the posterior translation, which was measured by an instrumented spatial linkage, to within ± 1 mm of the intact knee at 90° while a 100 N posterior force was applied to the proximal tibia. Bundle tension was measured at the femoral end of the graft, during bundle tensioning and subsequent knee motion limit testing, using strain gage load cells. Both the AL2-S and AL2-I reconstructions were tensioned to share load, and the AL2-D reconstruction was tensioned so the AL2 bundle carried 80% of the resistive force. The tests were performed on each specimen and a group average was calculated based on the reconstruction used for the specimen.
Detailed Methods

The specimen preparation and testing used in this study follows the protocol outlined in a previous publication.[81]

Reconstructions: Bone-patellar tendon-bone grafts were used because the bone ends facilitated graft gripping and prevented graft slippage. One-bundle grafts were comprised of the central 10 mm section of the patellar tendon along with 10 mm x 20 mm bone ends. Two-bundle, Y-shaped, grafts were formed by cutting the one-bundle grafts into medial and lateral halves. The tibial bone was not divided leaving the two bundles attached. The graft bone ends were fixed within cylindrical stainless-steel grips using polymethyl methacrylate (PMMA). Two size grips were used, one with a 10 mm inside diameter (ID) for the large bone ends, and one with a 5 mm ID for the small bone ends.

The graft ends were attached to the tibia and femur by cylindrical fixtures that were placed within holes drilled through the bone.[81] The fixture design and the method of attachment to bone prevented axial rotation of the graft bone ends while allowing for adjustment of graft tension. Graft bundle tension was measured at the femoral end by strain gage load cells with sensitivity of 0.9 mV/lb.

The tibial fixture was placed within a 16 mm drill hole that started just lateral to the tibial tubercle and exited at the center of the PCL’s tibial attachment. Within the tibial fixture, the bone grip was oriented to place the collagenous material along the proximal edge of the tunnel. The femoral fixture for the one-bundle AL₁ grafts was placed within a 16 mm drill hole that started medial to the trochlear groove and exited within the AL band of the PCL. The center of the femoral hole was chosen so that the shallow edge of the graft was 3 mm proximal to the articular cartilage margin. The femoral fixtures for the two-bundle grafts were placed within a
10 mm drill hole that started on the medial femoral condyle and ended within the substance of the PCL. The center for the AL₂ hole and the S hole was chosen so that the shallow edge of the graft was 2 mm proximal to the articular cartilage margin. The center for the I hole was chosen so that the shallow edge of the graft was 5 mm proximal to the articular cartilage margin, and the center for the D hole was chosen so that the center was on or just shallow to the proximal edge of the native PCL. For all of the bundles, the graft fibers were located along the shallow edge of the grip with the transverse axis of the graft in the anterior-posterior direction of the femur.

**Graft Tensioning:** The grafts were tensioned to restore posterior translation to within ± 1 mm of the intact knee at 90° while a 100 N posterior force was applied to the proximal tibia. The graft was implanted in the knee, and each bundle was tensioned to 45 N. The knee was then cycled five times from near full extension to 120° of flexion to stabilize graft behavior, and the knee was returned to 90° of flexion. The bundles were then tensioned to return the posterior translation to normal. After each subsequent half turn of the tensioning nut, Figure D.1, the knee was cycled once from near full extension to 120° of flexion to ensure that the applied tension reached the intraarticular portion of the graft. The average tension needed for the bundles in the one and two-bundle reconstructions to restore the posterior translation is shown in Table 4.1. The two-bundle AL₂-S and the AL₂-I reconstructions were tensioned to share the load equally at 90°, Table 4.1. The two-bundle AL₂-D reconstruction was tensioned so the AL₂ bundle would carry approximately 80% of the load at 90°. This load distribution was used so that the maximum tension in each bundle (AL₂ in flexion and D in extension) would be similar as shown previously.[81]
Table 4.1. Posterior Translation After Graft Tensioning¹

<table>
<thead>
<tr>
<th>Reconstruction</th>
<th>n</th>
<th>Δ Post. Trans. (mm)</th>
<th>AL Bundle Tension (N)</th>
<th>2nd Bundle Tension (N)</th>
</tr>
</thead>
<tbody>
<tr>
<td>One-bundle AL₁</td>
<td>5</td>
<td>-0.7 ± 0.3</td>
<td>230.3 ± 23</td>
<td>n.a.</td>
</tr>
<tr>
<td>Two-bundle AL₂-S</td>
<td>5</td>
<td>-0.1 ± 0.3</td>
<td>123.3 ± 6</td>
<td>152.3 ± 14</td>
</tr>
<tr>
<td>Two-bundle AL₂-I</td>
<td>5</td>
<td>-0.1 ± 0.4</td>
<td>148.0 ± 12</td>
<td>155.1 ± 15</td>
</tr>
<tr>
<td>Two-bundle AL₂-D</td>
<td>4</td>
<td>0.6 ± 0.5</td>
<td>246.9 ± 20</td>
<td>40.8 ± 16</td>
</tr>
</tbody>
</table>

¹Data are for 90º flexion with a 100 N posterior force applied to the proximal tibia. Values are mean ± standard error of the mean. Δ Post. Trans. is the change in posterior translation from the intact knee.

**Testing:** Posterior motion limits were determined while a 100 N posterior force was applied to the proximal tibia via two pneumatic actuators. To avoid constraining tibial rotation, a linear bearing oriented in the medial-lateral direction was placed at the effector end of the actuators, and the actuator force was applied to a 73-mm polyvinyl chloride hemicylinder attached anteriorly to the proximal tibia. The hemicylinder was attached to the proximal tibia with two screws and PMMA. During each motion limit test, the knee was cycled from near full extension to 120° of flexion while the knee motions as described by Grood and Suntay[57] were recorded using an instrumented spatial linkage accurate to 0.5 mm and 0.5°[134]. The PCL and the meniscofemoral ligaments were then severed approximately 5 mm from their femoral attachment sites through an anterior incision, which was used to remove the patella and patellar tendon. The portion of the fibers that remain attached to the femur were used as a guide for graft bundle placement. The tibial attachment of the PCL was resected through a posterior midline incision. The knee was then reconstructed with either a one or two-bundle reconstruction. The posterior motion limit was determined for the reconstructed knee while the tension in each bundle was measured.
Statistical Analysis

The specimens were assigned to groups based on the reconstruction performed on the knee. Each group contained 5 specimens, except for the AL2-D reconstruction group which had 4 specimens. The bundle tension residuals were normal and homoscedastic (equal variance between and within groups). Analysis of variance (ANOVA) was performed with reconstruction modeled as a fixed factor. The data at flexion angles of 5, 10, 15, 30, 45, 60, 75, 90, 105, and 120° were treated as repeated measures. If the ANOVA detected a difference, post hoc testing was performed to find the difference in the group means using the Fischer’s protected LSD method for multiple comparisons. A one way, between subjects ANOVA was performed on the peak tension in each group. If a difference was detected by the ANOVA, the post hoc testing was performed as described before. A regression analysis (bundle tension vs. flexion angle) was then performed for each bundle. Another one way, between subjects ANOVA was performed to determine if the slopes produced by the regression analysis were significantly different. Once again if significance was found, the post hoc tests was performed as before. A one way, within subjects ANOVA was performed for the two-bundle reconstructions to determine if the averaged bundle tension, the peak bundle tension, and the slope produced by the regression analysis were significantly different between the bundles. If significance was found, the post hoc tests were performed as before. Power was calculated for the tests that were found to be insignificant even though the data would suggest a difference exists. The level of significance used was $P < 0.05$, and the analyses were performed using SPSS version 10.1 (SPSS Inc., Chicago, Illinois).
RESULTS

ANTEROLATERAL BUNDLE

Table 4.2. Anterolateral Bundle Tension

<table>
<thead>
<tr>
<th>Reconstruction</th>
<th>Average Tension(^1)</th>
<th>Peak Tension(^1)</th>
<th>Angle @ Peak</th>
<th>Slope(^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>One-bundle AL(_1)</td>
<td>149.3 ± 11</td>
<td>236.2 ± 23</td>
<td>105°</td>
<td>49.9 ± 4</td>
</tr>
<tr>
<td>Two bundle AL(_2)-S</td>
<td>74.7 ± 7</td>
<td>133.9 ± 12</td>
<td>75°</td>
<td>18.9 ± 4</td>
</tr>
<tr>
<td>Two bundle AL(_2)-I</td>
<td>88.6 ± 9</td>
<td>148.0 ± 12</td>
<td>90°</td>
<td>25.7 ± 5</td>
</tr>
<tr>
<td>Two bundle AL(_2)-D</td>
<td>133.2 ± 14</td>
<td>246.9 ± 20</td>
<td>90°</td>
<td>60.0 ± 6</td>
</tr>
</tbody>
</table>

\(^1\) mean ± standard error of the mean in newtons. \(^2\) mean ± standard error of the mean in newtons/30° of flexion

The average AL bundle tension significantly decreased with the addition of a second bundle placed in a shallow (S) or in an intermediate (I) location (\(P < 0.001\) and \(P = 0.002\), respectively), Table 4.2 and Figure 4.4, but not when the second bundle was placed deeper within the notch. The average AL bundle tension was not significantly different when the second bundle was in either the S or the I location, but the average AL bundle tension was significantly greater when paired with the D bundle than when paired with either the S or the I bundles (\(P = 0.003\) and \(P = 0.018\), respectively).

The peak AL bundle tension significantly decreased with the addition of a second bundle placed in the S or the I location (\(P < 0.001\) and \(P = 0.002\), respectively), Table 4.2 and Figure 4.4, but the D bundle did not affect the peak AL bundle tension. The peak AL bundle tension was not significantly different when the second bundle was either the S or the I bundle, but the peak AL bundle tension was significantly greater when paired with the D bundle than when paired with either the S or the I bundles (\(P < 0.001\) and \(P = 0.001\), respectively).

The AL bundle tension slope significantly decreased with the addition of a second bundle placed in either the S or the I location (\(P = 0.006\) and \(P = 0.026\), respectively), Table 4.2 but not with the addition of the D bundle. The AL bundle tension slope was not significantly different when the second bundle was either the S or the I bundle, but the AL bundle tension slope was
significantly greater when paired with the D bundle than when paired with either the S or the I bundle \((P = 0.002\) and \(P = 0.006\), respectively).

**SECOND BUNDLE**

<table>
<thead>
<tr>
<th>Reconstruction</th>
<th>Average Tension(^1)</th>
<th>Peak Tension(^1)</th>
<th>Angle @ Peak</th>
<th>Slope(^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Two bundle AL(_2)-S</td>
<td>93.2 ± 15</td>
<td>309.1 ± 27</td>
<td>120(^\circ)</td>
<td>68.4 ± 6</td>
</tr>
<tr>
<td>Two bundle AL(_2)-I</td>
<td>124.5 ± 8</td>
<td>210.1 ± 29</td>
<td>120(^\circ)</td>
<td>31.8 ± 4</td>
</tr>
<tr>
<td>Two bundle AL(_2)-D</td>
<td>74.2 ± 8</td>
<td>119.8 ± 29</td>
<td>5(^\circ)</td>
<td>-19.0 ± 5</td>
</tr>
</tbody>
</table>

\(^1\) mean ± standard error of the mean in newtons. \(^2\) mean ± standard error of the mean in newtons/30\(^\circ\) of flexion

The average I bundle tension was significantly greater than in either the S or the D bundles \((P = 0.028\) and \(P = 0.003\), respectively), Table 4.3 and Figure 4.5. The average tension for either the S or the I bundle was not significantly.

The peak S bundle tension was significantly greater than in either the I or the D bundle \((P = 0.028\) and \(P = 0.001\), respectively), Table 4.3 and Figure 4.5. The peak I bundle tension was greater than the peak D bundle tension, but the difference was not significant. The power with \(\alpha = 0.05\) to determine the difference of 90.3 N was approximately 77%.

The slope of the second bundles significantly decreased as the bundle location was deeper in the notch. The S bundle tension slope was significantly greater than for either the I or the D bundle \((P = 0.021\) and \(P < 0.001\), respectively), and the I bundle tension slope was significantly greater than the D bundle tension slope \((P = 0.005\).

**DISCUSSION**

In this study, we found that the position of the second bundle not just the addition of a second bundle was critical for the reduction of tension in the AL bundle. When the AL bundle was combined with either the S or the I bundle, the average bundle tension, the peak bundle tension and the slope of the bundle tension curve was less than the values found for the one-
bundle reconstruction. The combination of the AL bundle with the D bundle did not provide a reduction in the loading characteristics when compared with the one bundle reconstruction.

We also found that the femoral attachment site of the second bundle was critical to the loading characteristics of the second bundle. The peak S bundle tension and the S bundle tension slope were significantly greater than in the other two bundles. The I bundle had the largest average bundle tension because the I bundle carried higher loads in extension when compared to the loads carried by the S bundle in extension and the D bundle in flexion. The D bundle possessed the lowest average bundle tension, peak bundle tension and slope for the bundle tension curve. The drastic change in the bundle tension was found for femoral tunnels that differed in proximal-distal position by only 7-8 mm.

The two-bundle reconstructions exhibited two different methods for graft load distribution. The AL2-S and the AL2-I reconstruction distributed the load evenly, Figures 4.8 and 4.9. The peak bundle tension and the slope for the bundle tension curves were not significantly different between the AL2 and the I bundles, but the average bundle tension was significantly greater in the I bundle ($P = 0.020$). The power with $\alpha = 0.05$ to determine a 62.1 N difference in the peak bundle tension was approximately 76%. The inability to find the difference in the peak tension can be attributed to the high variability in the peak I bundle tension. The AL2-S reconstruction exhibited an even load distribution for 90° of flexion, but then the tension in the S bundle ramped to over 300 N while the tension in the AL2 bundle declined to approximately 50 N. The difference in the average bundle tension for the AL2 and the S bundles were not significantly different, but the peak S bundle tension and the S bundle tension slope was significantly greater than the values in for the AL2 bundle ($P < 0.001$ and $P = 0.004$, respectively). The AL2-D reconstruction exhibited a reciprocal load distribution in which
one bundle (AL\textsubscript{2}) functioned in flexion and the other bundle (D) functioned in extension, Figure 4.10. The average bundle tension, the peak bundle tension and the bundle tension slope for the AL\textsubscript{2} bundle were significantly greater than the values for the D bundle ($P = 0.001$, $P = 0.001$, and $P < 0.001$, respectively).

The ability of the reconstructions to control posterior translation independent of the presence of the second bundle or the location of the second bundle was intriguing, Figure 4.6. Galloway et al.,[48] Stone et al.[133] and Harner et al.[65] have also shown that a high and shallow femoral bundle location is capable of controlling posterior translation. The tension recorded in the Stone et al.[133] and Harner et al.[133] studies was much lower than the forces found in this study because posterior translation remained after reconstruction in these studies. The presence of posterior translation can be attributed to their tensioning technique. Harner et al.[65] and Stone et al.[133] tensioned their reconstructions to a predetermined level, but in this study the reconstructions were tensioned to match the posterior translation found in the intact knee at 90°. Markolf et al.[85] performed a study on PCL reconstructions in which they employed an AL graft and a tensioning protocol similar to the one used in this study. Markolf et al.[85] recorded tension that was slightly greater than the applied posterior load at 90°, but we feel that the force in the graft should be nearly twice the applied load at 90° based on the anatomical orientation of the PCL.

The shallow-shallow combination required a larger force to control posterior translation in flexion than did the one-bundle reconstruction, Figure 4.7. The tension in the AL\textsubscript{2}-S reconstruction increased to a maximum of 361 N over 125 N more than the one-bundle reconstruction. We have also shown with this reconstruction that we can significantly reduce the
peak AL bundle tension. However to achieve this reduction, the second bundle had to carry extremely high loads in deep flexion, which could lead to graft elongation.

The shallow-intermediate combination also required a larger force to control posterior translation over the entire range of knee motion than did the one-bundle reconstruction, Figure 4.8. In this combination, the peak AL bundle tension was significantly reduced in comparison with the one-bundle reconstruction, and the peak I bundle tension was significantly less than the peak S bundle tension that may lead to better resistance to posterior translation for than for the AL2-S reconstruction.

The larger forces in the shallow-shallow and the shallow-intermediate reconstructions in comparison to the one-bundle reconstruction could be a potential problem. The increased force can be attributed to the orientation of the second bundle. For the one-bundle reconstruction, the graft is oriented in an anterior-posterior direction. In the AL2-S reconstruction and the AL2-I reconstruction, one bundle has the same orientation, but the second bundle has a greater medial-lateral deviation so a smaller proportion of the graft force is resisting posterior translation, Figures 4.8 & 4.9. Even though there is an equal load in each bundle, the bundles are not resisting the same amount of posterior translation so should the bundle distribution be based on force or on contribution to posterior translation resistance?

The shallow-deep combination also required a larger force in comparison to the one-bundle reconstruction over the entire range of knee motion, but the shallow-deep reconstruction became unstable beyond 105° of flexion, Figure 4.7. In this reconstruction, the addition of the second bundle did not reduce the peak AL bundle tension so we believe this combination would not improve the long-term success of PCL reconstruction.
In recent literature, several authors have examined the biomechanical nature of two-bundle PCL reconstructions.\cite{65, 81, 119} The two-bundle reconstructions controlled posterior translation regardless of the position of the second bundle. We believe that the second bundles used in these studies were positioned shallow,\cite{81} intermediate\cite{65} and deep\cite{81, 119} based upon descriptions, figures and loading patterns. Race and Amis\cite{119} produced loading patterns similar to our shallow-deep combination, but the magnitude of the force was not comparable because their data was obtained with no applied posterior load. Harner et al.\cite{65} used a shallow-intermediate reconstruction, but the position of their intermediate bundle was located between our intermediate and deep bundles based upon the loading pattern. They reported PCL force levels that were 80\% of the applied posterior force at 90°.\cite{65} The tension appears low based on the research of Butler et al. who found that at 90° the PCL resists almost 95\% of the posterior translation in the knee.\cite{23} In addition to this, the PCL is not oriented parallel to the anterior-posterior axis of the tibia; therefore only a percentage of the intraarticular PCL force is resisting posterior translation. However, Harner et al.\cite{65} still produced a knee that was not significantly different than the intact knee suggesting that another structure or an interaction of structures in the knee provided the posterior translation restraint. Mannor et al.\cite{81} studied both a shallow and deep positioning for the second bundle, and the bundle tension agrees with the results found in this study.

The selection of graft material and size should not be viewed as a recommendation for clinical use. The selection of the patellar tendon was necessitated by our method of holding and tensioning the graft and measuring the bundle tension. The patellar tendon allowed us to grip bone at both end to ensure adequate fixation. The 5-mm bundle width was needed for several reasons. First, the size of the graft bundle determined the tunnel size to be drilled in the femur.
The tunnel not only has to hold the bone grip, but the tunnel also had to accommodate the fixturing that tensioned the bundle and measured the bundle tension. A larger tunnel would inhibit our ability to accurately place these tunnels, and two large femoral tunnels could have weakened the femoral condyle and caused a fracture. The second reason was that a 10-mm wide graft has been used clinically as a single bundle to reconstruct the PCL. We wanted to show that any changes in the reconstruction were caused by the position of the second bundle and not the addition of collagenous material. The goal of this study was to determine the affects of altering femoral tunnel placement, and not graft selection. We feel that the results obtained from this study are applicable to the clinical environment regardless of our graft choice because Mehalik[87] found that a thin wire cable and a 10-mm Achilles tendon graft produced the same behaviors as long as the center of the tunnel holding the construct was the same.

This study provided insight into the biomechanics of two-bundle PCL reconstructions, which could produce better long-term resistance to posterior translation. The reconstructions, which placed the second bundle in either the S or the I location, significantly reduced the AL bundle tension. However for the AL2-S reconstruction, the reduced AL bundle tension was accompanied by high tension in the S bundle in deep flexion. The reduction in AL bundle tension improves the nonuniform load distribution, which we believe is the major contributor to graft elongation. Even though both the AL2-S and the AL2-I reconstructions reduced the AL bundle tension, the total graft tension was greater than the tension in the AL1 reconstruction at all flexion angles. The increased total graft tension can be attributed to the orientation of the second bundle. The second bundle has a larger medial-lateral deviation so less of the intraarticular bundle tension is resisting the posterior subluxing force. Therefore, the AL bundles in the AL2-S and AL2-I reconstructions are resisting more of the posterior subluxing force, even though the
bundle tensions are not significantly different. If a method to optimize the relationship between the functional distribution and the tension distribution is devised, the reconstructions could provide better resistance to posterior translation.
### Configurations Tested

<table>
<thead>
<tr>
<th>I.</th>
<th>II.</th>
<th>III.</th>
<th>IV.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intact Knee (N = 14)</td>
<td>PCL and MFL Sectioned (N = 14)</td>
<td>One-Bundle PCL Reconstruction AL1 (N = 5)</td>
<td>Two-Bundle PCL Reconstruction AL2-S (N = 5)</td>
</tr>
<tr>
<td></td>
<td>Same as I.</td>
<td>Flexion Angle, A/P Translation, Bundle Tension</td>
<td>Same as III.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>AL2-I (N = 5)</td>
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<td></td>
<td></td>
<td></td>
<td>AL2-D (N = 4)</td>
</tr>
</tbody>
</table>

**Figure 4.1.** Knee configurations tested and measurements made.
Figure 4.2. Sagittal view of the femur at 90° summarizing the center for each graft bundle
Figure 4.3. Sagittal view of the femur at 90°; A. AL₁ reconstruction (AL₁: 6.7 mm from trochlear groove, 10.6 mm from cartilage edge) B. AL₂-S reconstruction (AL₂: 5.2 mm from trochlear groove, 6.3 mm from cartilage edge; S: 14.5 mm from trochlear groove, 6.3 mm from cartilage edge) C. AL₂-I reconstruction (I: 14.8 mm from trochlear groove, 9.5 mm from cartilage edge) D. AL₂-D reconstruction (D: 12.1 mm from trochlear groove, 13.3 mm from cartilage edge)
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CHAPTER 5

POSTERIOR CRUCIATE LIGAMENT RECONSTRUCTION: POSTERIOR CREEP DURING CYCLIC FATIGUE TESTING

ABSTRACT

This study was designed to determine the ability of one and two-bundle posterior cruciate ligament (PCL) reconstructions to resist the return of posterior tibial translation. The one and two-bundle reconstructions were performed on 19 cadaveric knees. The femoral attachment of the one-bundle (AL1) graft and one bundle of all two-bundle (AL2) grafts were located within the PCL’s anterolateral band. The second bundle of the two-bundle grafts was placed posterior to the AL2 bundle in one of three locations that varied in their depth within the notch: shallow (S), intermediate (I), and deep (D). The tibial end of the one-bundle and two bundle grafts was placed at the center of the PCL’s tibial attachment. The specimens were cycled from near full extension to 120° of flexion while a 100 N posterior force was applied. The knees were cycled until the tension in each bundle was 50 N or less. Posterior translation and bundle tension were recorded periodically throughout the tests, and a data set was collected after graft failure. For a 2.5 mm increase in posterior translation, the two-bundle reconstructions did not provide better resistance to posterior translation than the AL1 reconstruction. For a 7.5 mm increase in posterior translation, the AL2-I reconstruction resisted the return of posterior translation for significantly more cycles than the AL1 reconstruction, but the two-bundle reconstructions did not reduce the rate of posterior translation return. However, we found that the rate of return for the AL2-I reconstruction decreased beyond 2.5 mm of posterior translation.
INTRODUCTION

In 1917, Hey Groves[59] stated that an injury to a cruciate ligament or ligaments “is an extremely serious one, which produces permanent total disablement for active pursuits and which necessitates a very prolonged treatment by immobilising apparatus.” In the same report, Hey Groves described a method for surgically reconstructing both cruciate ligaments. However, the importance of the PCL has not been widely accepted, historically. Little research was aimed at understanding how to repair an injured PCL because the frequency of injury was low in comparison to injury of other ligaments, and the effects associated with PCL injury were believed to be minor. The incidence rate for PCL injury has ranged from between 3.4 and 20% of all knee ligament injuries depending on the patient population,[38] but the belief is that the true incidence rate is approximately 7% of all knee ligament injuries[91] while the incidence rate for anterior cruciate ligament (ACL) injury is over 60% of all knee ligament injuries[91]. In addition to the infrequent occurrence, clinicians feel that patients with isolated PCL injuries would be unaffected and would fair well without surgery.[67]

However in the last decade, there has been an increase in research on the PCL function and the clinical treatment of PCL injuries. The primary reason is that the PCL has been found to be vital to normal operation of the knee.[29, 31] The PCL resists greater than 90% of posterior subluxing forces over the entire range of knee motion,[23] and the PCL is also a secondary restraint to external, varus and valgus rotations.[32, 43, 60] The PCL, in association with the anterior cruciate ligament (ACL), also contributes to the screw home mechanism.[43, 88, 89] Along with our increased knowledge of PCL function, there has been a growing appreciation that PCL injury is not as innocuous as once thought. Shelbourne and Rubinstein[128] found that isolated PCL injuries that are conservatively treated do not fair well over the long-term with
Injuries to the PCL often lead to difficulty in performing activities of daily living.[100] Patients report symptoms of pain, the feeling of giving way and locking while performing activities such as descending stairs, raising from a seated position and walking long distances. Over time, symptoms persist and progress. The development of patellofemoral pain is due to the posterior position of the tibia, which increases the patellofemoral contact pressure. The increased contact pressure begins to break down the cartilage on the patellar and femoral surfaces,[36] starting degenerative joint change. In addition to the break down of the cartilage, the posterior position of the tibia also leads to changes in other ligaments in the joint. For example, the ACL becomes weaker because the length between the ACL attachment sites decreases which leads to decreased physiological tension in ACL.[101]

The goal of PCL treatment is to return the knee to the preinjury state and to prevent or delay degenerative changes. Conservative treatment,[128] primary repair,[121] and isometric reconstruction[48, 104] do not restore the knee to normal because they are unable to return the normal posterior translation limit. In an effort to improve the aforementioned treatments, an anatomic reconstruction, which replicates the normal length tension behavior of the PCL,[35, 125] has been studied. Although this reconstruction initially restores the normal posterior translation limit,[12, 48] over time abnormal posterior translation frequently returns.[65, 85] The recurrence of abnormal posterior translation has been linked to improper femoral graft location,
excessive graft pretension and/or graft elongation.[48, 81] We believe that graft elongation is the key event and that graft elongation can occur at normal levels of PCL force due to a nonuniform distribution of force among graft fibers resulting from bending at the intraarticular entrance of the drill holes. While the graft bend at the tibia can be alleviated by performing a tibial inlay,[8, 89] no similar procedure exists to alleviate the effect of the femoral graft bend. It is our hypothesis that these bends in the graft cause only a small minority of graft fibers to transmit the majority of the PCL tension. As these fibers fail the graft permanently elongates, causing a decrease in PCL force along with recruitment of additional fibers. This process continues until the graft fails or until the newly recruited fibers are sufficient to transmit the remaining force.

The failure of the anatomic reconstruction to resist posterior translation in the long-term has prompted researchers to examine the cyclic behavior of PCL reconstruction. Bergfeld and colleagues[8] examined the change in posterior translation for a one-bundle reconstruction and reported that after only 72 loading-unloading cycles at 90° of flexion that 2 mm of posterior translation had returned. Mehalik[87] also examined the cyclic behavior of a one-bundle PCL reconstruction. Mehalik found an increase of approximately 2 mm of posterior translation while the knee was manually cycled from near full extension to 105° of flexion with an applied posterior load.[87] In addition to the cyclic behavior of PCL reconstructions researchers have also examined two-bundle PCL reconstructions in an attempt to improve the graft load distribution. Several studies have exhibited two-bundle PCL reconstructions that return the normal posterior translation limit[65, 81] and more evenly distribute load within the graft,[81] but the long-term behavior of these reconstructions has not been studied.

The study of two-bundle PCL reconstructions presents possibilities to increase graft longevity. In previous studies[65, 81], two methods of load distribution were found. An even
load distribution has the advantage of reduced peak tensions, but both bundles are required to work over a wide range of knee motion. A reciprocal load distribution allows each bundle to be unloaded for a portion of the range of motion, but the peak tensions are much higher. We questioned if load sharing or a reciprocal load distribution of a two-bundle reconstruction was better than the other, and the comparison of a two-bundle reconstruction with an anatomic one-bundle reconstruction. If the two-bundle configurations do not improve graft longevity is there a position within the PCL femoral footprint that can increase the graft longevity? The purpose of this study was to determine the ability of one and two-bundle PCL reconstructions to prevent the return of abnormal posterior translation during cyclic fatigue testing.

**METHODS**

**Study Design**

Cyclic fatigue tests were performed on knees reconstructed with one and two-bundle patellar tendon (PT) grafts. The reconstructions were performed on 19 cadaveric specimen from 14 donors (4 male, 10 female). The mean age of the specimens was 70.4 years with an age range of 50 - 88 years. The posterior motion limit for the intact knee, for the reconstructed knee and for the cycled knee was determined as the knee was moved from near full extension to 120° of flexion while a 100 N posterior force was applied, Figure 5.1. After the posterior motion limit was determined for the intact knee, the knee was cycled 2048 times while a 100 N posterior force was applied to determine the posterior creep for the knee. The knee motions were recorded at every 2th cycle until cycle number 256, and then, data was collected at every 128th cycle.

The femoral attachment of the one-bundle (AL₁) graft and one bundle of all the two-bundle (AL₂) grafts was at the site of the PCL’s anterolateral band, Figure 5.2. The second
bundle of the two-bundle grafts was placed posterior to the AL2 bundle in one of three locations that varied in their depth within the notch: shallow (S), intermediate (I), and deep (D), Figure 5.2. The tibial end of the one-bundle and two bundle grafts was placed at the center of the PCL’s tibial attachment. Prior to implanting the graft, a digital image was taken of each bundle to determine the initial length; no axial load was applied. The reconstructions were pretensioned to restore the posterior translation to within ± 1 mm of the intact knee at 90° with a 100 N applied posterior force. Bundle tension was measured at the femoral end of the graft, during bundle tensioning and subsequent knee motion limit testing, using strain gage load cells. Both the AL2-S and AL2-I reconstructions were tensioned to share load, and the AL2-D reconstruction was tensioned so the AL2 bundle carried approximately 80% of the resistive force. After the reconstruction, the knee was cycled to failure while 100 N of posterior force was applied. The posterior translation and bundle tension were recorded at every 2nth cycle until cycle number 256, and then, data was collected at every 128th cycle until failure. Failure occurred when the tension in each bundle was less than 50 N. After failure occurred, another posterior motion limit test was performed. The graft was then removed from the knee, and a second digital image was taken of the graft under the same conditions as before. The data collected after an abrasion or an avulsion failure was discarded because we wanted to represent midsubstance graft elongation. The failure modes were determined using the photographs taken both before and after cycling and the recess depth of the femoral bone grip.
Detailed Methods

The preparation of the specimen followed the protocol published in the Mannor et. al. study.[81]

Reconstructions: Bone-patellar tendon-bone grafts were used because the bone ends facilitated gripping the graft and prevented slippage. One-bundle grafts were comprised of the central 10 mm section of the patellar tendon along with 10 mm x 20 mm bone ends. Two-bundle, Y-shaped, grafts were formed by cutting the one-bundle grafts into medial and lateral halves. The tibial bone was not divided leaving the two bundles attached at one end. The graft’s bone ends were fixed within cylindrical stainless-steel grips using polymethyl methacrylate (PMMA). Two size grips were used, one with a 10 mm inside diameter (ID) for the large bone ends, and one with a 5 mm ID for the small bone ends.

The graft ends were attached to the tibia and femur by cylindrical fixtures that were placed within holes drilled through the bone.[81] The fixture design and the method of attachment to bone prevented axial rotation of the graft bone ends while allowing for adjustment of graft tension. Graft bundle tension was measured at the femoral end by strain gage load cells (ELW-B1-200L, Entran Devices, Inc., Fairfield, NJ) with sensitivity of 0.9 mV/lb.

The tibial fixture was placed within a 16 mm drill hole that started just lateral to the tibial tubercle and exited at the center of the PCL’s tibial attachment. Within the tibial fixture, the bone grip was oriented to place the collagenous material along the proximal edge of the tunnel. The femoral fixture for the one-bundle AL1 grafts was placed within a 16 mm drill hole that started medial to the trochlear groove and exited in the AL band of the PCL. The center of the hole was chosen so that the shallow edge of the graft was 3 mm proximal to the articular cartilage margin. The femoral fixtures for the two-bundle grafts were placed within a 10 mm
drill holes that started on the medial femoral condyle and exited in the substance of the PCL. The center for the AL₂ hole and the S hole was chosen so that the shallow edge of the graft was 2 mm proximal to the articular cartilage margin. The center for the I hole was chosen so that the shallow edge of the graft was 5 mm proximal to the articular cartilage margin, and the center for the D hole was chosen so that the center of the hole was on or just shallow to the proximal edge of the native PCL. For all of the bundles, the graft fibers were located along the shallow edge of the bone grip with the transverse axis of the graft in the anterior-posterior direction in the femur.

**Graft Tensioning:** The grafts were tensioned to restore posterior translation to within ±1 mm of the intact knee at 90° while a 100 N posterior force was applied to the proximal tibia. The graft was implanted in the knee, and each bundle was tensioned to 45 N. The knee was then cycled five times from near full extension to 120° of flexion to stabilize graft behavior, and the knee was returned to 90° of flexion. The bundles were then tensioned to return posterior translation to normal. After each subsequent half turn of the tensioning nut, Figure D.1, the knee was cycled once from near full extension to 120° of flexion to ensure that the applied tension reached the intraarticular portion of the graft. For the two-bundle reconstructions, two different tension distributions were employed. The two-bundle AL₂-S and the AL₂-I reconstructions were tensioned to share the load equally at 90°, Table 5.1. The two-bundle AL₂-D reconstruction was tensioned so the AL₂ bundle would carry approximately 80% of the load at 90°. This load distribution was used so that the maximum tension in each bundle (AL₂ in flexion and D in extension) would not be different from each other.[81]

**Cyclic Fatigue Testing:** During testing, the knees were cycled from near full extension to 120° of flexion and back in steps of 0.09° at a rate of one cycle every five seconds with a 100 N applied posterior force. The flexion and extension motions were produced by a stepper motor.
(M112-FF-206, Warner Electric, Bristol, Connecticut) with a 1.8° step size, which was attached to a planetary gear head (PL42-020-M001, Warner Electric, Bristol, Connecticut) with a gear ratio of 20:1.

To establish the endpoints of knee motion, the specimen was stepped to a position near 90° of flexion, a data point was collected, and the flexion angle was verified using a goniometer. If the flexion angle was not 90°, the knee was stepped to the appropriate flexion angle. Once a 90° flexion angle was obtained, the knee was extended to between 1 and 2° of flexion, and a limit switch was placed adjacent to the leading edge of the trigger, Figure 5.3. The knee was then stepped through 123.75° of flexion, and a second limit switch was placed adjacent to the leading edge of the trigger.

**Testing:** Posterior motion limits were determined while a 100 N posterior force was applied to the proximal tibia via two pneumatic actuators. To avoid constraining tibial rotation, a linear bearing oriented in the medial-lateral direction was placed at the effector end of the actuators, and the actuator force was applied to a 73-mm polyvinyl chloride hemicylinder attached anteriorly to the proximal tibia. The hemicylinder was attached to the proximal tibia with two screws and PMMA. During each motion limit test, the knee was cycled from near full extension to 120° of flexion while the knee motions as described by Grood and Suntay[57] were recorded using an instrumented spatial linkage accurate to 0.5 mm and 0.5°[134]. The intact knee was then cycled 50 times with a 50 N applied posterior force. When the cycling was complete, the knee was wrapped in medium saturated gauze[81] and stepped to approximately 90° of flexion, and then a 100 N anterior force was applied to the knee for one hour. After the hour, the knee was returned to the zero set point, and the knee was then cycled 2048 times from near full extension to 120° of flexion with a 100 N applied posterior force. The joint motions
were recorded every 2\textsuperscript{nd} cycle for the first 256 cycles, and then the data was collected every 128\textsuperscript{th} cycle. The knee was placed in the refrigerator (4°C) for a day and a half to allow the knee to recover.

The knee was removed from the refrigerator the morning of testing and allowed to warm to room temperature. A posterior motion limit test was performed again as a 100 N posterior force was applied. The PCL and the meniscofemoral ligaments were then severed approximately 5 mm from their femoral attachment sites through an anterior incision, which was used to remove the patella and patellar tendon. The portion of the fibers that remain attached to the femur were used as a guide for graft bundle placement. The tibial attachment of the PCL was resected through a posterior midline incision.

The knee was then reconstructed with either a one or two-bundle graft. The posterior motion limit was determined while tension in each bundle was recorded. The knee was then cycled 30 times with a 50 N posterior force applied. When the cycling was complete, the knee was wrapped in medium saturated gauze and stepped to approximately 90° of flexion, and then a 100 N anterior force was applied to the knee for one hour. After the hour, the knee was returned to the zero set point, and the knee was cycled from near full extension to 120° of flexion with a 100 N applied posterior force until graft failure. The knee motions and the bundle tension were recorded every 2\textsuperscript{nd} cycle for the first 256 cycles, and then the data was collected every 128\textsuperscript{th} cycle until failure. After failure occurred, another posterior motion limit test was performed.

**Statistical Analysis**

The residuals from the change in posterior translation for the intact knee from the first to second day of testing were normal and homoscedastic (equal variance between and within the groups). This data was then tested to determine if the change in posterior translation was
significant using a paired student t-test at each of the flexion angles. The residuals from the change in posterior translation for the intact knee due to cycling were also normal and homoscedastic. The posterior translation was then tested to see if the changes were significantly different than at the beginning of cyclic testing. The significance was tested with a paired student t-test.

For the cycling of the reconstructed knee, the specimens were separated into 4 groups (N = 5, for AL₁, AL₂-S and AL₂-I; N = 4 for AL₂-D). Four values were calculated from the posterior translation data. The cycle numbers, which represented a 2.5 mm and a 7.5 mm increase in posterior translation, were determined. A regression analysis (posterior translation vs. cycle number) was also determined for a 2.5 mm and a 7.5 mm increase in posterior translation. The residuals from the calculated values were found to be normal and homoscedastic. A one way, between subjects analysis of variance (ANOVA) was used to compare the four calculated values. If the ANOVA determined a difference, post hoc testing using the Fischer’s protected LSD method found where the difference existed. Power was calculated for the tests that were found to be insignificant even though the data would suggest there should have been a difference. The level of significance used was $P < 0.05$, and the analyses were performed using SPSS version 10.1 (SPSS Inc., Chicago, Illinois).
RESULTS

SPECIMEN EXCLUSION

Table 5.1. Reasons for excluding specimen from results

<table>
<thead>
<tr>
<th>Data Set</th>
<th>N</th>
<th>Reason</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intact Knee: first to second day changes</td>
<td>3</td>
<td>Large decrease in posterior translation, specimen were outliers (a value that is three and a half standard deviations of more different than the group mean)</td>
</tr>
<tr>
<td>Intact Knee: cycling</td>
<td>2</td>
<td>Specimen were outliers</td>
</tr>
<tr>
<td>Reconstructed Knee: cycling</td>
<td>3</td>
<td>2 – Bundle damage during the 30 cycle precondition stage</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 – Early abrasion failure in the AL2 bundle</td>
</tr>
<tr>
<td>Reconstructed Knee: 2.5 mm</td>
<td>1</td>
<td>Qualified as an outlier</td>
</tr>
<tr>
<td>Reconstructed Knee: 7.5 mm</td>
<td>2</td>
<td>Abrasive failure before 7.5 mm posterior translation increase</td>
</tr>
</tbody>
</table>

INTACT KNEE

The posterior translation for the remaining 11 specimens did not change significantly from the first to the second day of testing, Figure 5.4. Cycling the remaining intact knees 2048 times while a posterior force was applied produced a small, less than 1 mm of posterior translation increase, but the increase in posterior translation was significant at all of the cycle numbers except for cycle 4, 16, and 384, Figure 5.5.
### RECONSTRUCTED KNEE

#### Table 5.2. Cyclic data for the reconstructed knees

<table>
<thead>
<tr>
<th>Reconstruction</th>
<th>n</th>
<th>Cycle Number to 2.5 mm</th>
<th>Regression Slope for 2.5 mm&lt;sup&gt;1&lt;/sup&gt;</th>
<th>Cycle Number to 7.5 mm</th>
<th>Regression Slope for 7.5 mm&lt;sup&gt;1&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>AL&lt;sub&gt;1&lt;/sub&gt;</td>
<td>4&lt;sup&gt;3&lt;/sup&gt;</td>
<td>137 ± 43.9</td>
<td>2.3 ± 0.7</td>
<td>267 ± 51.8</td>
<td>2.5 ± 0.4</td>
</tr>
<tr>
<td>AL&lt;sub&gt;2&lt;/sub&gt;-S</td>
<td>4</td>
<td>171 ± 15.4</td>
<td>1.5 ± 0.2</td>
<td>347 ± 63.2</td>
<td>2.1 ± 0.5</td>
</tr>
<tr>
<td>AL&lt;sub&gt;2&lt;/sub&gt;-I</td>
<td>4&lt;sup&gt;3&lt;/sup&gt;</td>
<td>158 ± 21.0</td>
<td>1.5 ± 0.1</td>
<td>694 ± 188.2</td>
<td>1.2 ± 0.3</td>
</tr>
<tr>
<td>AL&lt;sub&gt;2&lt;/sub&gt;-D</td>
<td>4&lt;sup&gt;2&lt;/sup&gt;</td>
<td>80 ± 10.4</td>
<td>3.1 ± 0.5</td>
<td>176 ± 14.0</td>
<td>4.0 ± 0.4</td>
</tr>
</tbody>
</table>

<sup>1</sup>mean ± standard error of the mean in mm/100 cycles.  <sup>2</sup>Specimen excluded from 2.5 mm group.  <sup>3</sup>Specimen excluded from 7.5 mm group.

#### 2.5 mm posterior translation

The reconstruction configuration did not affect the number of cycles or the rate of posterior translation return, Table 5.2 and Figures 5.6, 5.7, 5.8, and 5.9. Since the ANOVA did not reveal a significant effect in either case a power analysis was performed to determine the validity of the results. For cycle number, an 80% power with $\alpha = 0.05$ required a difference of 79 cycles. For rate of return, a power of 80% with $\alpha = 0.05$ required a difference of 1.3 mm/100 cycles.

#### 7.5 mm posterior translation

The AL<sub>2</sub>-I reconstruction was the only two-bundle reconstruction that provided an improvement in the number of cycles to reach 7.5 mm of posterior translation in comparison to the AL<sub>1</sub> reconstruction ($P = 0.011$), Table 5.2 and Figures 5.6, 5.7, 5.8, and 5.9. As for the two-bundle reconstructions, the AL<sub>2</sub>-I reconstruction was significantly better at resisting the return of posterior translation than either the AL<sub>2</sub>-S or the AL<sub>2</sub>-D reconstruction ($P =0.022$ and $P = 0.002$, respectively), and the AL<sub>2</sub>-S reconstruction provided better resistance to the return of posterior translation than did the AL<sub>2</sub>-D reconstruction, but the difference was not significant. The power to determine the 171 cycle difference was 96.8%.
None of the two-bundle reconstructions significantly reduced the rate of posterior translation return at the 7.5 mm level in comparison to the AL₁ reconstruction. The AL₂-I reconstruction exhibited a reduction in rate of return, but the difference was not significant when compared to the one-bundle reconstruction. The power with $\alpha = 0.05$ to determine the 1.3 mm/100 cycles was approximately 76%. As for the two-bundle reconstructions, the AL₂-S and the AL₂-I reconstructions significantly reduced the rate of return in comparison to the AL₂-D reconstruction ($P = 0.006$ and $P = 0.01$, respectively), and the AL₂-S and the AL₂-I reconstructions were not significantly different.

**DISCUSSION**

The ability of the intact knee to recover and resist posterior creep during testing will allow for more meaningful future experimentation. We found that the knee kinematics were the same before cyclic testing and after a one-day recovery period following cycling. This finding will allow for the testing of more conditions in a single knee knowing that the initial conditions are the same, thus increasing the power in the study. We also found that cycling the intact knee did not produce large posterior translation increases. The largest increase in posterior translation was $1.0 \pm 0.1$ mm. The aged specimen used in this study exhibited a resistance to posterior creep so the results obtained would not be significantly different than the response of the younger patient population to cyclic fatigue testing.

The two-bundle reconstructions did not reproduce the creep resistance of the intact knee, but we did find that the AL₂-I reconstruction showed statistically significant increases in creep resistance as compared to the traditional anterolateral one-bundle reconstruction at the 7.5 level. The two-bundle reconstructions were unable to show statistical improvement at the 2.5 mm posterior translation level due to the large variability in the AL₁ reconstruction. The AL₂-I
reconstruction exhibited an increased cycled number to 7.5 of mm posterior translation as compared to the AL₁ reconstruction. The increase at the 7.5 mm interval was approximately 400 cycles. We have shown that the AL₂-I is better at creep resistance, but are the improvements found in this study clinically relevant?

In the literature, only two other studies have examined the cyclic fatigue of a PCL reconstruction. Bergfeld and colleagues[8] studied the increased posterior translation for both a tibial inlay one-bundle reconstruction and a tibial tunnel one-bundle reconstruction. They performed both reconstructions using a 10 mm patellar tendon graft. The reconstructions were tested by fixing the knees at 90° of flexion, and applying 150 N anterior-posterior force that cycled at a rate of 1 cycle per 5 seconds.[8] Bergfeld et. al.[8] found that after 72 cycles, approximately 2 mm of posterior translation had returned in both reconstructions. The amount of posterior translation increase found in our study and that of Bergfeld are similar, but the results for both studies pale in comparison to the performance of the intact knee.

Mehalik[87] studied a nonisometric one-bundle 11 mm Achilles tendon reconstruction. The knees were manually cycled from near full extension to 105° of flexion as a 50 N posterior force was applied.[87] Mehalik found an increase in posterior translation of approximately 1.7 mm at 90° of flexion after 1000 cycles.[87] These results are better than those found in our study and the Bergfeld et al study. The improvements could be attributed to a lower force, to a reduced range of motion and to the use of the Achilles tendon graft, where more fibers are available to resist the posterior creep, as compared to a patellar tendon graft.

The rate of posterior translation return for the two-bundle reconstructions did not provide as promising results as we found for the cycle number to 2.5 mm and 7.5 mm of posterior translation. The rate of return for all of the two-bundle reconstructions was not significantly
different than the one-bundle reconstruction for the data up to and including 2.5 mm of posterior translation. All of the rates were greater than 1 mm/100 cycles, which in comparison to the intact knee at less than 1 mm/2000 cycles is poor. For the rates calculated for 7.5 mm of posterior translation and less, the AL2-I reconstruction had a better outcome in comparison to the AL1 reconstruction, but the rate of 1.2 mm/100 cycles is still not as good as the intact knee. One interesting point in this data is that the rate for the AL2-I reconstruction decreases from the 2.5 mm group to the 7.5 mm group (1.5 mm/100 cycles to 1.2 mm/100 cycles). This may indicate that maintaining 2 to 2.5 mm of posterior translation at the time of graft tensioning may increase the graft’s resistance to the return of posterior translation.

Another interesting finding from the two-bundle reconstruction data was the trend in bundle failure order. For the AL2-S reconstruction, the S bundle consistently failed first. This can be attributed to the large forces in the S bundle in high flexion at the beginning of cycling (approximately 275 N). For the AL2-D reconstruction, the AL2 bundle was the first bundle to fail because the AL2 bundle was the only bundle resisting the posterior translation in flexion. For the AL2-I reconstruction, an unexpected trend appeared in which the AL2 bundle failed first in all but one case in which the loading pattern for the I bundle resembled the loading pattern for a S bundle; even though the femoral tunnel was 9.4 mm from the cartilage edge. Due to the initial load sharing between the bundles, we believe that the bundles should fail in a similar fashion. A possible explanation for this trend is that the load sharing we produced, an equal magnitude between the groups, is not as important as producing a functional equivalence between the bundles. To produce the equivalent functionality, the level of tension in the I bundle would need to be higher to account for the increased medial-lateral deviation when compared to the AL2 bundle.
The results from the two-bundle reconstructions did not provide a construct that would eliminate the return of the abnormal posterior translation, but the results did provide insight into factors that may increase the longevity of the grafts. The AL2-S and the AL2-D reconstructions exhibited high bundle tension in the extremes of motion. Since the knee were cycled from full extension to 120° of flexion, we hypothesize that a reduction of flexion and possibly extension would allow longer graft survival. The reduction in flexion would provide a more dramatic increase in graft viability because the reduction in extension would only influence the AL2-D reconstruction, but we found that the AL2 bundle, which is unloaded in extension, failed first.

A second factor that could increase the longevity of the graft is reducing joint load. The significance of this factor can be seen when comparing our study to the one by Mehalik. The testing methods of Mehalik are similar to those used in this study with two exceptions. First, the posterior force applied was only 50 N compared to our 100 N force, and Mehalik used an 11 mm Achilles tendon graft. The increased number of fibers in Mehalik’s graft would provide better graft longevity, but it could not account for the over 10 fold increase in cycle number to achieve approximately 2 mm of posterior translation.

Residual posterior translation maintained during reconstruction would protect the graft before the remodeling process is complete. From this study we found that the rate of return in the AL2-I reconstructions decreased beyond 2.5 mm of posterior translation. Several studies have examined problems that result from the residual translation and the subsequent loss of physiological tension. First, will the lower tension in the graft significantly affect the mechanical properties of the graft? Bush-Joseph and colleagues found that the maximum load to failure was 50% less for an ACL reconstruction with approximately a 5 mm residual anterior translation, but Bush-Joseph and colleagues accomplished the increased laxity by moving the
tibial insertion of the ACL 5 mm posterior. Majima et al[80, 91] studied how removing all or part of the load (approximately 70 %) from a patellar tendon would affect the mechanical properties. Majima et al[80] found that a partially loaded patellar tendon (30% of control) regained approximately 73% of the tensile strength of the control at 12 weeks while the unloaded patellar tendon only regained approximately 21% of the tensile strength at 6 weeks. The hope is that the effects of the reduced tension will not manifest because we are maintaining an anatomical PCL attachment sites and a larger graft tension. The second question is is it possible for surgeons to produce the appropriate residual posterior translation on a consistent basis? We feel this may be difficult to do clinically, and the variability in the level of residual posterior translation may be detrimental to the reconstruction.

A limitation to this study is the size of the graft bundles. In this study we used two 5 mm wide patellar tendon graft bundles, and clinically, surgeons are using a minimum of 7 to 8 mm wide bundles. We used the 5mm wide bundle to minimize the size of the femoral tunnels needed. The femoral tunnel not only contained the graft but also the fixture that held the graft, measured bundle tension, and tensioned the graft. If a larger graft was used, the femoral tunnel would also have to be larger, which may have compromised the femoral condyle and the accuracy of graft placement. We feel that if larger graft bundles were used that the number of cycles to failure would increase and the rate of posterior translation return would decrease, but the behavior of the reconstructions would be the same if the same graft centers were used. To examine the effects of larger grafts, we plan on pursuing the use of in vivo force transducers to monitor bundle tension enabling us to increase the size of the graft bundles.

This study did not produce a two-bundle reconstruction that was superior to the one-bundle PCL reconstruction in resistance to posterior translation return, but this study did
elucidate several areas that may produce improvements to existing PCL reconstruction. The AL2-I reconstruction was the only two-bundle reconstruction that exhibited improved posterior translation resistance, but the 400 cycle improvement to 7.5 mm of posterior translation was minor when compared to the intact knee, which allowed only 1 mm of posterior translation to return after 2000 cycle. The rate of posterior translation return for all reconstructions to the 2.5 mm level was not significantly different. The AL2-I reconstruction’s rate of return was reduced beyond 2.5 mm of posterior translation. If a residual posterior laxity was employed in PCL reconstruction, the resistance to posterior translation return may improve because of the reduced bundle tension. In addition to the residual posterior laxity, a limited range of motion could provide a better outcome because the S bundle exhibited high forces in deep flexion. A reduced joint load may also improve the resistance to posterior translation. The significance of joint load was found when the present study was compared to the study of Mehalik, who used a 50 N posterior force and found less than 2 mm of posterior translation increase after 1000 cycles[87].
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CHAPTER 6

LOCATION OF FAILURE FOR POSTERIOR CRUCIATE LIGAMENT RECONSTRUCTIONS DURING CYCLIC FATIGUE TESTING

ABSTRACT

Anatomic one-bundle posterior cruciate ligament (PCL) reconstructions initially restore the posterior translation limit for the knee,[12, 48] but with time, abnormal posterior translation frequently returns.[65, 81, 85] Several investigators have theorized that the acute angle at the tibial tunnel exit caused the PCL reconstructions to fail.[7, 8] This study was designed to determine if PCL grafts demonstrated a preferential failure region during cyclic fatigue testing.

The one and two-bundle reconstructions were performed on 19 cadaveric knees (5 one-bundle and 14 two-bundle). The femoral attachment of the one-bundle (AL₁) graft and one bundle of all two-bundle (AL₂) grafts were located within the PCL’s anterolateral band. The second bundle of the two-bundle grafts was placed posterior to the AL₂ bundle in one of three locations that varied in their depth within the notch: shallow (S), intermediate (I), and deep (D). The tibial end of the one-bundle and two-bundle grafts was placed at the center of the PCL’s tibial attachment. The specimens were cycled from near full extension to 120° of flexion while a 100 N posterior force was applied. The knee cycling continued until the tension in each bundle was 50 N or less. A digital image of the graft was taken prior to fatigue testing and after graft failure to determine where and how the graft failed. The bundle failures were divided into one of three regions (tibial, midsubstance and femoral).
Of the 33 bundles, eight were excluded from analysis due to an abrasion or an avulsion failure or poor image quality. The femoral region was shown to be the preferential failure region.

The tibial tunnel exit was not the preferential failure region for this study. However, the tibial inlay procedure should still be used to reduce surgical time\cite{8} (graft passage and graft pretension) and to improve healing (poor graft integration\cite{89, 130} due to large bone loss and high internal graft pressures at tunnel exit and poor orientation of graft fibers\cite{8, 130}).

**INTRODUCTION**

The natural history of injury and repair of the posterior cruciate ligament (PCL) has been given little mention in the literature, but recently, more research has focused on the biomechanics and the repair of the PCL because clinical success of PCL repair is unlikely.\cite{40, 75, 106, 121, 127} In addition to poor clinical outcome, an untreated PCL injury predisposes the knee to the early onset of osteoarthritis. Therefore, the goal of repair is to slow or stop the onset of the osteoarthritis \cite{33, 71} with reconstruction exhibiting several advantageous characteristics. First, PCL reconstruction has the ability to restore normal knee kinematics,\cite{12, 48} and secondly, anterior cruciate ligament (ACL) reconstruction has produced normal or nearly normal knees.\cite{35, 71}

Based on the ACL reconstructive procedure, clinicians first reconstructed the PCL using an isometrically placed femoral tunnel. This reconstruction produced unacceptable results because the abnormal posterior translation was not eliminated beyond 45° of flexion\cite{48}. Biomechanical studies determined that an isometrically placed graft did not represent the native PCL\cite{48, 111} because only 5 to 15 % of the native PCL is isometric\cite{35, 125}. To accommodate
the behavior of the native PCL, the position of the femoral tunnel was moved into a nonisometric position near the anterolateral (AL) portion of the PCL. The AL portion of the PCL was chosen because these fibers become taut with flexion,[35, 90] which is the region where the PCL resists over 90% of the posterior subluxing force.[23] This AL reconstruction was found to restore the normal posterior motion limit,[12, 48] but over time the abnormal posterior translation returns.[65, 81, 85] The return of the posterior translation has been linked to improper graft placement,[48, 81] inadequate graft pretension,[48, 81, 85] graft elongation,[48, 81] and the acute angle formed at the exit of the tibial tunnel[7, 8, 89, 90].

The tibial tunnel approach for PCL reconstruction has been questioned in the last decade. The use of the tibial tunnel in ACL reconstruction is appropriate because the graft exits the tunnel along the centerline; however, the PCL graft wraps around the edge of the tunnel, which can cause graft abrasion[7, 89]. The tibial tunnel in the PCL reconstruction also requires that a large portion of tibial bone stock be removed because the tunnel traverses the full width of the tibia.[7] These concerns and others lead to the naming of the tibial tunnel exit as the “killer turn”.[7, 90] Investigators also felt that the tibial tunnel approach leads to poor graft placement,[8] makes graft passage difficult,[7] and shields the applied pretension from the intraarticular portion of the graft.[7, 8] The tunnel exit has also been theorized to hamper the incorporation of the graft,[89, 130] to inhibit proper graft fiber orientation,[8, 130] and to alter the cellular differentiation and viability.[130] With the concerns surrounding the tunnel approach, little literature has studied the failure mechanism and location in PCL reconstruction. The purpose of this study was to determine the location of failure for a PCL reconstruction during a cyclic fatigue test.
METHODS

Study Design

The failure location was investigated using both one and two-bundle patellar tendon (PT) grafts. The reconstructions were performed on 19 cadaveric specimen from 14 donors (4 male, 10 female). The mean age of the specimens was 70.4 years with an age range of 50 - 88 years. Prior to implanting the graft, a digital image was taken of each bundle to determine the initial length; no axial load was applied. The femoral attachment of the one-bundle (AL₁) graft and one bundle of all two-bundle (AL₂) grafts was at the site of the PCL’s anterolateral band, Figure 6.1. The second bundle of the two-bundle grafts was placed posterior to the AL₂ bundle in one of three locations that varied in their depth within the notch: shallow (S), intermediate (I), and deep (D), Figure 6.1. The tibial end of the one-bundle and two bundle grafts was placed at the center of the PCL’s tibial attachment. The reconstructions were pretensioned to restore the posterior translation to within ± 1 mm of the intact knee at 90° while a 100 N posterior force was applied to the proximal tibia. Bundle tension was measured at the femoral end of the graft, during bundle tensioning and subsequent knee motion limit testing, using strain gage load cells. Both the AL₂-S and AL₂-I reconstructions were tensioned to share load, and the AL₂-D reconstruction was tensioned so the AL₂ bundle carried 80% of the resistive force. After the reconstruction, the knee was cycled to failure while 100 N of posterior force was applied. The bundle tension was recorded at the 2ⁿᵗʰ cycle until cycle number 256, and then, the data was collected at every 128th cycle until failure. Failure occurred when the tension in each bundle was less than 50 N. The graft was then removed from the knee, and a second digital image was taken of the graft under
the same conditions as before. If the bundle failed as a result of an abrasion or an avulsion tear, the failure location for that bundle was discarded from the study.

Detailed Methods

The preparation of the specimen followed the protocol published in the Mannor et. al. study.[81]

_Graft Preparation and Documentation:_ On the day prior to testing, the PT was removed from the freezer and allowed to thaw to room temperature. Bone-patellar tendon-bone grafts were used because the bone ends facilitated gripping the graft and prevented slippage. The one-bundle graft was created by removing a 10-mm section from the central third of the PT including 20-mm long bone blocks at both ends. Two-bundle, Y-shaped, grafts were formed by cutting the one-bundle grafts into medial and lateral halves. The tibial bone was not divided leaving the two bundles attached at one end. The graft bone blocks were shaped to fit into the bone grips (one-bundle: two 10-mm inner diameter; two-bundle: two 5-mm inner diameter and one 10-mm inner diameter). The bone blocks were secured in the bone grips using polymethyl methacrylate (PMMA). The graft was then wrapped with medium saturated gauze and allowed to cure at room temperature for one hour, and then the graft was placed in the refrigerator (4°C) overnight. The medium was used to maintain pH, water content and inhibit collagenase activity. The composition of the medium was the same as reported in the Mannor et al. study.[81]

On the day of testing, the graft was removed from the refrigerator and allowed to warm to room temperature. Prior to implanting the graft into the knee, a digital image of each bundle was captured using a camera (PULNiX TM-7CN, Fuji Optical Co., Japan) and imaging software (NIH Image Version 1.62a, Scion Corporation, Frederick, Maryland). The image was taken as the graft rested on the stage and with no applied axial load. A second image was taken after the
reconstruction had failed using the same conditions as the first image. The location of the surface failure was determined by examining the second image. The location was categorized into tibial, midsubstance and femoral. The tibial region comprised the tissue within 12 mm of the tibial bone block, the midsubstance region comprised the region between 12 and 26 mm from the tibial bone block, and the femoral region was comprised of the tissue that was greater than 26 mm from the tibial bone block. The tibial block bone was chosen as the reference point because the tibial bone was set at the same depth in the tibial tunnel for each reconstruction.

**Reconstructions:** The graft ends were attached to the tibia and femur by cylindrical fixtures that were placed within holes drilled through the bone.[81] The fixture design and the method of attachment to bone prevented axial rotation of the graft bone ends while allowing for adjustment of graft tension. Graft bundle tension was measured at the femoral end by strain gage load cells (ELW-B1-200L, Entran Devices, Inc., Fairfield, NJ) with a sensitivity of 0.9 mV/lb.

The tibial fixture was placed within a 16 mm drill hole that started just lateral to the tibial tubercle and exited at the center of the PCL’s tibial attachment. Within the tibial fixture, the bone grip was oriented to place the collagenous material along the proximal edge of the tunnel. The femoral fixture for the one-bundle AL₁ grafts was placed within a 16 mm drill hole that started medial to the trochlear groove and exited in the AL band of the PCL. The center of the hole was chosen so that the shallow edge of the graft was 3 mm proximal to the articular cartilage margin. The femoral fixtures for the two-bundle grafts were placed within a 10 mm drill holes that started on the medial femoral condyle and exited in the substance of the PCL. The center for the AL₂ and the S holes was chosen so that the shallow edge of the graft was 2 mm proximal to the articular cartilage margin. The center for the I hole was chosen so that the shallow edge of the graft was 5 mm proximal to the articular cartilage margin, and the center for
the D hole was chosen so that the center of the hole was on or just shallow to the proximal edge of the native PCL. For all of the bundles, the graft fibers were located along the shallow edge of the bone grip with the transverse axis of the graft in the anterior-posterior direction in the femur.

**Graft Tensioning:** The grafts were tensioned to restore posterior translation to within $\pm 1$ mm of the intact knee at $90^\circ$ while a 100 N posterior force was applied to the proximal tibia. The graft was implanted in the knee, and each bundle was tensioned to 45 N. The knee was then cycled five times from near full extension to $120^\circ$ of flexion to stabilize graft behavior, and the knee was returned to $90^\circ$ of flexion. The bundles were then tensioned to return posterior translation to normal. After each subsequent half turn of the tensioning nut, Figure D.1, the knee was cycled once from near full extension to $120^\circ$ of flexion to ensure that the applied tension reached the intraarticular portion of the graft. For the two-bundle reconstructions, two different tension distributions were employed. The two-bundle AL$_2$-S and the AL$_2$-I reconstructions were tensioned to share the load equally at $90^\circ$. The two-bundle AL$_2$-D reconstruction was tensioned so the AL$_2$ bundle would carry approximately 80% of the load at $90^\circ$. This load distribution was used so that the maximum tension in each bundle (AL$_2$ in flexion and D in extension) would not be different from each other.[81]

**Cyclic Fatigue Testing:** During testing, the knees were cycled from near full extension to $120^\circ$ of flexion and back in steps of 0.09$^\circ$ at a rate of one cycle every five seconds with a 100 N applied posterior force. The flexion-extension motion was produced by a stepper motor (M112-FF-206, Warner Electric, Bristol, Connecticut) with a 1.8$^\circ$ step size attached to a planetary gear head (PL42-020-M001, Warner Electric, Bristol, Connecticut) with a gear ratio of 20:1.
To establish the endpoints of knee motion, the specimen was stepped to a position near 90° of flexion, a data point was collected, and a goniometer reading was taken to verify the flexion angle. If the flexion angle was not 90°, the knee was stepped to the appropriate flexion angle. Once a 90° flexion angle was obtained, the knee was extended to 1 to 2° of flexion, and a limit switch was placed adjacent to the leading edge of the trigger, Figure 6.2. The knee was then stepped through 123.75° of flexion, and a second limit switch was placed adjacent to the leading edge of the trigger.

**Testing:** The knee was reconstructed with either a one-bundle or two-bundle reconstruction. The knee was then cycled 30 times with a 50 N posterior force applied. When the cycling was complete, the knee was wrapped in medium saturated gauze and cycled to approximately 90° of flexion, and then a 100 N anterior force was applied to the knee for one hour. After the hour, the knee was returned to the zero set point, and the knee was cycled from near full extension to 120° of flexion until the graft failed. The bundle tension was recorded every 2nth cycle for the first 256 cycles, and then the data was collected every 128th cycle.

**Statistical Analysis**

The failure location for the reconstructions was classified into one of three regions regardless of reconstruction type. A chi-squared test was performed to determine if there was a preferential region for graft failure. The level of significance used was $P < 0.05$, and the analyses were performed using SPSS version 10.1 (SPSS Inc., Chicago, Illinois).
RESULTS

Nineteen specimens (5 one-bundle and 14 two-bundle) were cyclically tested to failure. From these nineteen specimens, 33 bundles were examined to determine failure location. Eight of the 33 bundles were eliminated from consideration due an abrasion or an avulsion tear or poor image quality. Of the remaining bundles, nineteen of the bundles failed in the femoral region, five failed in the midsubstance, and one of the bundles failed in the tibial region, Figure 6.3. The chi-squared test revealed that the femoral region of the graft was the preferential failure region ($P < 0.001$).

DISCUSSION

PCL reconstruction initially followed procedures similar to those of ACL reconstruction because ACL reconstruction produced outcomes that were successful.[35, 71] When this technique was used for the PCL, the success rate did not replicate the success found for ACL reconstruction.[106] One of the major flaws in the technique was the use of an isometrically placed femoral PCL tunnel. The majority of the PCL is nonisometric with only 5 to 15% of the ligament being isometric.[35, 125] A second flaw in the technique may be the use of a tunnel for tibial graft placement. For the ACL reconstruction, the graft exits the tunnel with minimal deviation for the centerline. However for the PCL, the graft makes an acute turn just after it exits the tibial tunnel. With time, this acute angle became known as the “killer turn”.\[7, 90]\n
The “killer turn” is believed to present problems during the reconstruction itself and to contribute to reconstruction failure. The technical difficulties resulting from the “killer turn” or the creation of the turn range from tunnel placement problems[8] to graft pretension problems.[7,
This method also requires that a large bone tunnel be drilled, which will lead to slow healing and may lead to incomplete graft incorporation (Personnel communication with Frank Noyes), and makes the graft passage difficult especially when using smaller length grafts. In addition to the surgical problems with the tunnel procedure, a new set of problems arises after the surgery. The acute turn is believed to cause poor fiber orientation, which prevents the graft from restoring the native structure and function of the PCL. The ridge at the exit of the tunnel may abrade the graft during knee motion. Graft healing may be hindered because of the high internal graft pressure and/or the large pressure gradients in the graft at the exit of the tunnel. The abnormal graft pressure has been linked to altered cellular differentiation and lowered cellular viability in the region. These factors alone and in combination maybe responsible for the recurrence of abnormal posterior translation in PCL reconstruction.

The potential detrimental effects of the tunnel PCL reconstruction have lead investigators to examine the tibial inlay technique. The introduction of this technique can be traced back to several studies in the last decade. The inlay technique eliminates the technical difficulties found using a tibial tunnel technique. The inlay technique is also believed to alleviate the problems that occur post-operatively. Most importantly the graft fiber orientation is believed to improve and the abrasive ridge is eliminated. The inlay technique may also lead to reduced graft pretension.

Biomechanical investigation comparing the inlay technique to the tunnel technique during cyclic testing has been limited to a study by Bergfeld et al. Bergfeld et al. found that the inlay procedure produced a knee that was not significantly different than the intact knee when the tibia was rotated internally or externally, and when the tibia was in a neutral rotation,
the posterior translation found using the tibial inlay technique was significantly less than that of the intact knee. Bergfeld et al.[8] also found that the posterior translation for the tunnel reconstruction was not significantly different than the intact knee except at 90° of flexion when the tibia was internally rotated. However, Bergfeld et. al.[8] found that when a cyclic load was applied to the proximal tibia that the posterior translation in the tunnel group was significantly greater than the posterior translation for both the intact knee and the inlay reconstructed knee, and the graft in the tunnel reconstruction appeared to be more degraded than the graft in the inlay reconstruction in the region of the tibial tunnel exit. The significance was based on absolute posterior translation, but when the change in posterior translation is examined, both reconstruction methods produced an increase of approximately 2 mm after 72 cycles. The difference found for absolute posterior translation was due to the initial overconstraint of the inlay group.

The methods used to measure graft degradation for the two groups also may not represent a true difference. The optical measurement of the graft size indicated that the tunnel group had thinned during the 72 cycles of repetitive loadings.[8] The thinned graft would indicate that the acute exit at the tibial tunnel abrades the graft, but other alternatives for the graft thinning exist such as a loss of water during the cycling. The ridge at the tibial tunnel exit could be pushing on the graft and forcing out water, not mechanically degrading the tissue. To truly test the mechanical integrity of the tissue, an uniaxial mechanical test needed to be performed. If the tibial tunnel degraded the graft, the grafts would have failed at lower loads, and the failures would have been located in the region adjacent to the “killer turn”.

The results of this study may not depict clinical PCL reconstruction failures because aged specimens were studied. With aged specimen, the belief is that the tissue quality has decreased,
and the failure mechanism has changed with age.[97] We examined the material properties of
the patellar tendons using uniaxial tests. We found that the maximum stress to failure was 55 ± 4.8 MPa, and we also found that the maximum stress to failure for the patellar tendon (PT) after 2000 cycles was 37 ± 4.8 MPa. Butler et al.[22] found that the maximum stress to failure in younger specimen was 68.5 ± 6.0 MPa, and the maximum stress to failure for the ACL, PCL, and LCL was 36.4 ± 2.5 MPa. This showed that an aged PT graft would be capable of reconstructing the PCL in young patients.

In this current study, we examined the failure location of both one and two-bundle PCL reconstructions in which a tibial tunnel was used. We found that graft failure was preferential to the femoral region. Therefore, we feel that the use of the tunnel reconstruction does not predispose the graft to be abraded and fail at the exit of the tibial tunnel. However, the other concerns involved with the tunnel reconstruction still exist. The use of the inlay technique should be further studied because of the potential reduction in technical difficulties,[7, 8] the increased graft incorporation,[89, 130] and the improved graft healing.[130]
Figure 6.1. Sagittal view of the femur at $90^\circ$ summarizing the center for each tunnel
Figure 6.2. A. Motor test stand, B. Close up of center of rotation and limiting switch, C. Motion limitation mechanism
Figure 6.3. Failure location A) Femoral, B) Midsubstance, C) Tibial
CHAPTER 7

PERSPECTIVES AND RECOMMENDATIONS

RESIDUAL POSTERIOR TRANSLATION

Several factors from the cyclic fatigue testing prompted the thought that maintaining a residual posterior translation at the time of surgery would improve the long-term success of the reconstruction. During cycling, the resistance to a 2.5-mm posterior translation increase was not significantly different for any reconstruction. In addition, the rate of posterior translation return for the AL2-I reconstruction beyond 2.5 mm of posterior translation decreased. If the graft was tensioned to maintain 2.5 mm of posterior translation, the long-term success of the reconstructions would improve because the graft tension would decrease, but would the residual laxity and the reduced tension in the graft harm the repair and/or other knee structures? Bush-Joseph and colleagues[21] found that an ACL reconstruction with 5 mm of residual translation produced a graft with only 50% of the load to failure as compared to control. However, this study was performed by moving the tibial ACL insertion 5 mm posterior so was the change in the ultimate strength of the ACL as result of the residual laxity or the new insertion site? In addition to the possible effects on the graft itself, a posterior tibial position has been shown to degrade the ACL due to a shorter physiological length and lower physiological tension.[96, 101] However, these studies were focused on complete PCL disruptions, which result in posterior translation increases of 10 mm or more, and not the minimal 2.5 mm posterior translation level proposed.

To study the effects of the residual posterior translation, many questions need to be answered. What is the relationship between posterior translation and graft tension at 90° of
flexion? Can a significant reduction in graft tension be achieved while maintaining a clinically acceptable posterior translation level? The relationship between bundle tension and posterior translation appears to be a nonlinear, inverse proportion so as posterior translation is removed, graft tension greatly increases. If a significant graft tension reduction is found, then the new pretensioning protocol will be implemented in vitro to determine if the long-term resistance to posterior translation is increased. If these experiments provide encouraging results, in vivo experimentation is needed to determine how the new protocol will behave during graft remodeling and if the residual posterior translation affects other collagenous knee structures. The effects of residual posterior translation will be elucidated using both mechanical testing and morphometry.

EQUAL MAGNITUDE OR EQUAL FUNCTION

The use of a two-bundle PCL reconstruction was advocated to reduce or eliminate the occurrence of graft elongation, which is believed to be the cause of failure in anatomic PCL reconstruction[48, 81, 85]. Graft elongation is theorized to be caused by a nonuniform graft load distribution. This study has shown that the reconstructions, which share load evenly based on bundle tension, produce a better resistance to posterior translation that the reciprocal load sharing construct. However, the improvement exhibited by the load sharing reconstruction did not approach the posterior translation resistance for the intact PCL.

Several possible explanations can be given for the premature failure of the load sharing reconstructions. For the AL₂-S reconstruction, a dramatic increase in the S bundle tension in high flexion is produced when an even load distribution is achieved at 90°. For the AL₂-I reconstruction, there is no dramatic increase in I bundle tension, and the load is shared throughout the range of motion except at the last flexion angle tested. However, the AL₂ bundle
failed first in all but one case in which the I bundle behaved in a similar manner to a S bundle. A possible explanation is that the AL bundle is resisting a disproportionate amount of the posterior subluxing force because the bundle placed posterior to the AL bundle has a greater medial-lateral deviation. Therefore less of the intraarticular force resists the posterior subluxing force. Based on the orientation of the one-bundle reconstruction, the AL2 bundle from the AL2-I reconstruction is resisting approximately 65% of the posterior subluxing force.

To determine if an equal functional distribution will provide better long-term resistance to posterior translation, a study using the AL2-I reconstruction would be conducted. A potential problem with reducing the discrepancy between the functional resistance for each bundle is that the I bundle could become overloaded and lead to premature reconstruction failure. Therefore, an additional study should be conducted to examine the junction between the equal functional distribution and the equal tension distribution. However, the clinical application does not appear to be feasible, but the results from these studies would provide insight into the behavior of the native PCL.

REDUCTION OF JOINT LOAD

In comparing the results from this study to those of others[8, 87], the magnitude of the externally applied load affects the reconstruction’s ability to resist posterior translation. In this study, the best reconstruction failed in less than a thousand cycles with the other reconstructions failing at less than five hundred cycles when a 100 N posterior force was applied. Bergfeld et al.[8] found a 2 mm increase in posterior translation after 72 cycles with a 150 N anterior-posterior force when the knee fixed at 90°, and Mehalik[87] found less than 2 mm of posterior translation increase after one thousand cycles of flexion-extension with a 50 N applied posterior force. The reduced joint load will also reduce the bundle tension possibly providing a similar
improvement theorized with the residual posterior translation, but the reduced joint load also has the same potential problems as with the residual posterior translation theory. If reducing the joint load produced better resistance to posterior translation and did not adversely affect the graft and the surrounding structures, clinical application would be feasible by slowing the return of weight bearing for the patient.

The determination of how a reduced joint load would affect the reconstructions would be based on in vitro and in vivo studies. First, in vitro studies would examine what level of joint load reduction would produce significant increases in the resistance to posterior translation. Then in vivo studies would be used to examine the integrity of the reconstruction as well as the effects of reduced tension on the graft material properties and the material properties of the other collagenous knee structures.

LIMITED RANGE OF MOTION

While examining the results from this study, several finding appeared to identify that the extremes of motion could lead to the premature failure of the reconstruction. For the two-bundle reconstructions, the second bundles produced peak tension in the extreme positions of motion (high flexion for the S bundle and the I bundle and near full extension for the D bundle). However, only the S bundle was shown to be adversely affected by the deep flexion produced in this study because the S bundle was found to fail first in all of the reconstructions. Therefore, a range of motion limitation should be focused on flexion, but a long-term range of motion limitation could cause that range of flexion to be permanently lost, which is not clinically acceptable. In the proposed case, a flexion contracture could be caused by the shortening of the quadriceps muscle and/or the thickening of the anterior capsular structures. If limiting range of
motion improved PCL reconstruction and did not produce a flexion contracture, clinical application would be feasible by restricting postoperative motion using a knee brace.

The effects of range of motion limitation could require several different trials to determine the benefits and side effects. In vitro experiments would determine the range of motion limitation needed to provide an increased posterior translation resistance. The motion limitation found would be incorporated into an in vivo study using quadrupeds, which would be followed for 3 to 6 months to determine the efficacy of the reconstruction and the ability of the knee to flex beyond the imposed motion limit. If a flexion contracture was produced, morphological analysis would be performed on the anterior capsular structures and the length of the quadriceps muscle would be found. To avoid the flexion contracture, unloading the knee in high flexion could produce the desired effect, but there is a possibility that unloading the knee in flexion could lead to structural damage of the graft and other collagenous knee structures. In vitro and in vivo experiments similar to the ones previously described in this paragraph would be used to determine when in the range of motion and to what extent to unload the knee and the detrimental effects on the collagenous knee structures.

FEMORAL ATTACHMENT AND LOCATION

While completing this study, a trend in the graft failure location appeared. To ensure graft failure was located within the graft substance and not an abrasion or an avulsion failure, images were taken both before and after the cyclic fatigue testing, and the images showed that a disproportionate number of graft bundles was failing near the femoral end. Of the failures that were not abrasions or avulsions, 19 of the 25 graft bundles failed in the femoral third of the graft. However, PCL grafts were believed to failure near the tibial end because of the acute angle produced at the exit of the osseous tunnel,[7, 8] which was named the “killer turn”. The tibial
inlay procedure was developed to eliminate the effects of the turn. The primary problem at the “killer turn” is that the graft rubs against the bony edge, but the graft also has the same problem at the femoral end. The prevalence of the femoral tunnel failures could be attributed to graft twisting as the knee cycles through the range of motion, which may increase the nonuniform load distribution.

The elimination of the potential problems at the femoral end is not possible without introducing adverse side effects. Two methods (placing the graft bone block flush with the tunnel exit and attaching the graft to the lateral wall of the medial femoral condyle) can eliminate the rubbing of the graft against the tunnel exit with knee motion. However if the location of the femoral tunnel is moved to the center of the notch and placed parallel to the anterior-posterior axis in the femur, all of the potential problems at the femoral end could be eliminated, but this tunnel placement would alter the relationship between the ACL and the PCL. This change would produce an extension contracture because the ACL and the PCL would contact earlier in the range of motion, and as both ligaments become taut, further extension would be inhibited.

This study has raised many questions that have the potential to improve the existing treatment for PCL injury, but I do not believe that with existing methodologies a treatment for PCL injury will be found that parallel the success found for ACL reconstruction. Current PCL reconstructive techniques use grafts taken from well organized structures that have parallel running fibers and possess an even load distribution. These grafts are then implanted to replace the PCL, which fans out at the femoral and tibial attachment sites, is not as well organized and resists loads by recruiting different fibers at different flexion angles. In addition, PCL reconstructions use tunnels at the femoral end and sometimes at the tibial end, which bends the naturally straight graft, attributing to the nonuniform load distribution. Even though the grafts
undergo a remodeling phase after implantation to better address the new mechanical loading environment, I feel that the disparity between graft function and the native PCL is so great that the reconstruction is compromised from the beginning. However, PCL reconstruction research needs to continue to improve upon the existing techniques and to explore new graft sources because the inability to slow or stop the onset of degenerative changes is unacceptable and leads to a life of discomfort and disability.
CHAPTER 8

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APPENDIX A

DEFINITIONS

Anatomical Position – A person standing erect with their arms at their side and their feet together. The palms of their hands are facing anteriorly so the thumbs point laterally. The feet are positioned together with the toes pointing anterior, Figure A.1.[54]

Frontal Plane -- The vertical plane that divides the body along the anterior-posterior direction, Figure A.1.

Transverse Plane – The horizontal plane that divides the body along the superior-inferior direction, Figure A.1.

Sagittal Plane – The vertical plane that divides the body along the right-left direction, Figure A.1.

Anterior -- A direction towards the front of the body or the front surface of the body, Figure A.1.

Posterior -- A direction towards the back of the body or the back surface of the body, Figure A.1.

Lateral – A direction away from the center of the body,
**Medial** – A direction towards the center of the body, Figure A.1.

**Proximal** – A direction towards the origin of a body limb, Figures A.1 and A.2.

**Distal** – A direction away from the origin of a body limb, Figures A.1 and A.2.

**Shallow** – A direction within the femoral notch that is towards the distal articular cartilage margin. In terms of the anatomical nomenclature, shallow is distal and slightly anterior, Figure A.2.

**Deep** -- A direction within the femoral notch that is away from the distal articular cartilage margin. In terms of the anatomical nomenclature, deep is proximal and slightly posterior, Figure A.2.

**High** – A direction within the femoral notch that is towards the roof of the notch. In terms of anatomical nomenclature, high is anterior and slightly proximal, Figure A.2.

**Low** – A direction within the femoral notch that is away from the roof of the notch. In terms of anatomical nomenclature, low is posterior and slightly distal, Figure A.2.

**Isolated PCL Injury** – An injury in which only the PCL is injured.

**Combined PCL Injury** – An injury in which the PCL and one or more structures are injured.

**Reconstruction** – “The attempt to restore stability to the knee through ligamentous replacement, augmentation, or redirection.”[39]

**Isometric** – “A pair of points on opposite sides of the joint that remain equidistant as the knee goes through a normal range of motion”[12]. The definition is relaxed in the laboratory setting to be a length change of less than 2 mm over the entire range of knee motion.[12, 35, 112]

**Subjective results** – Qualitative results reported by either the patient or the physician

**Objective results** – Quantitative results reported by physician or physical therapist
APPENDIX B

DETERMINATION OF FORCE OUTPUT FROM PNEUMATIC ACTUATORS

ABSTRACT

The accurate application of external loads in cadaveric studies is critical to the quality and validity of the results. This study was performed to validate the theoretically calculated pressures required to produce 50 and 100 N of applied load. The theoretical calculation found that the air pressures of 13.6 psi and 27.2 psi were needed to obtain the 50 and 100 N applied loads. The validation of these values was conducted using the forcing measurement system from our posterior cruciate ligament studies. For this study, the air pressure was increased from 0 psi to 30 psi in 1 psi increments, and the output from the strain gage load cell was recorded using a large scale integration system. The study revealed a linear relationship between the applied load and the air pressure (between 4 and 30 psi), and the air pressure needed to produce 50 and 100 N of applied load was 15.3 psi and 27.8 psi. The results from this study confirm that the theoretical calculations were within reason given the potential error in the system (±4.0 N). This study also revealed a frictional element within the pneumatic actuators based on an air pressure change without a corresponding load change, and a two times increase in applied load did not result from a 2 times increase in air pressure (only a 1.8 times increase).

INTRODUCTION

The application of external loads in cadaveric studies has varied greatly. The external loads have been applied using manual means,[84] a mechanical testing system,[8] a robotic
system[65] and pneumatic actuators, [81] which are currently employed in our laboratory. To
determine the external loads applied by our pneumatic system, theoretical calculations were
made based on the surface area over which the load was applied and the air pressure in the
system. The validity of the theoretically calculated external loads came into question as initial
data from a posterior cruciate ligament (PCL) bundle tension study were being evaluated against
data from the literature. Our recorded bundle tensions were consistently higher than those
present in the literature at 90° (chosen because PCL resisted approximately 95% of load at
90°[23]). Our bundle tension was twice as large as the tension data from other studies.[65, 81,
85] The discrepancy in the recorded tension levels called into question our methods of applying
the external load and recording the intraarticular tension. The external load was examined first
because the values were based on theoretical calculations. The purpose of this study was to
determine if the theoretical calculations were valid.

METHODS

Study Design

The validation of the theoretical calculation was performed using the force measurement
system from the PCL bundle tension studies, Figure B.1. The load cell was calibrated to produce
a relationship of 1 V for 31.2 N of force. The pneumatic actuators and the force measurement
system were positioned so that the line of action for the pneumatic actuators was coincident with
the axial loading axis of the strain gage load cell, Figure B.1. The air pressure supplied to the
pneumatic actuators began with 0 psi and increased by 1 psi until 30 psi. From the theoretical
calculation, the air pressure needed for 50 and 100 N of applied load was 13.6 psi and 27.2 psi.
Detailed Methods

Load Cell Calibration: The force measurement system, except the anti-rotation collar, used in the PCL bundle tension study was placed in a wooden block so that the end of the femoral guide tube was exposed, Figure B.1. The system was oriented so the load-sensing axis of the strain gage load cell (ELW-B1-200L, Entran Devices, Inc., Fairfield, NJ) was vertical. An 11.2 lb load was attached to the thread rod using a nylon rope secured in a bone grip with polymethyl methacrylate, and the load remained overnight to stabilize the load cell output. On the day of testing, the load cell was calibrated so a 1 volt output correlated to 31.2 N of force. The load cell was unloaded, and the zero point was set. The 11.2 lb load was applied, and the gain on the signal amplifier (2310 Strain Gage Conditioning Amplifier, Measurements Group, Inc., Raleigh, NC) was adjusted until the multimeter output read 1.6 volts. The load was removed, then reapplied, and if the multimeter output was not 1.6 volts, the gain on the signal amplifier was readjusted. If the multimeter output was 1.6 volts, the loading unloading cycle was repeated, and if the output was 1.6 volts, the 11.2 lb load was removed, and the system was reset. The 11.2 lb load was then reapplied, and if the multimeter output was 1.6 volts the load cell was calibrated.

System mounting: The force measurement system was mounted in a similar manner as before with the orientation of the loading axis now in the horizontal plane, Figure B.1. A second tensioning nut was placed on the end on the threaded rod so the larger diameter surface was nearest the pneumatic actuators (SDR-12-4, Clippard Instruments Laboratory, Cincinnati, OH). A small block of wood was placed adjacent to the tensioning nut, and on the opposite side of the wooden block, a 73-mm polyvinyl chloride hemicylinder was attached using two screws. The hemicylinder was used to simulate how force was applied in the PCL bundle tension study. The
pneumatic actuators were placed adjacent to the hemicylinder with a linear bearing separating the structures. The line of action for the pneumatic actuators was aligned to be coincident with the loading axis of the load cell.

**Voltage Measurement:** The output from the strain gage load cells was recorded using a large scale integration (LSI) system. The output from the load cell was sent to the LSI system as an auxiliary channel. This channel was designed to provide a large impedance to reduce noise, and a multiplication factor of 10 was set for the channel to increase the accuracy of the measurements.

**Air Pressure:** The air pressure, which were controlled by a regulator (R00-02-000, Wilkerson Corporation, Englewood, CO), started at 0 psi and ended with 30 psi using 1 psi steps.

**Theoretical calculation:** The surface area at the end of the pneumatic actuators is 0.827 in$^2$ so the air pressure required to produce a 50 and 100 N force was 13.6 N and 27.2 N.
The air pressures of 1, 2 and 3 psi did not produce an applied force. The relationship between the applied force and the air pressure of 4 to 30 psi was linear with a correlation coefficient of 0.99, Table B.1. The equation for the line was

\[
F_{\text{applied}} = 4.02 \frac{N}{\text{psi}} P_{\text{air}} - 11.7N \quad (1)
\]

Therefore the air pressure needed to provide 50 and 100 N of applied force was 15.3 psi and 27.8 psi respectively.
CONCLUSIONS

The experimental relationship found between the applied force and the air pressure reveals that the theoretical calculated pressures do not produce the prescribed applied force. The 13.6 psi and the 27.2 psi pressure produce 43.0 N and 97.6 N of applied load, respectively. For the higher load level, the theoretical calculation was within the accuracy of the system (± 4.0 N). The diminishing difference between the theoretical calculations and the experimental values as the air pressure increased may indicate the presence of a frictional element in the pneumatic actuators. The friction in the pneumatic actuators may also be responsible for the absence of an applied force change as the air pressure increased from 0 to 4 psi. However, the results from this experiment proved that the application of the external loads in our PCL bundle tension studies was not responsible for the discrepancies between our study and those in the literature.
Figure B.1. Experimental set-up to measure applied load
APPENDIX C

EFFECTS OF OFF AXIS LOADING AND RECESS DEPTH ON FORCE OUTPUT FROM AN AXIAL STRAIN GAGE LOAD CELL

ABSTRACT

Measuring forces in collagenous structures in in vitro and in vivo studies has presented problems. This study was performed to determine a correction factor for an axial strain gage load cell to accommodate changes in the exit angle and the recess depth. The experiments were conducted using the force measurement system from our posterior cruciate ligament studies. For the experiment, the exit angle was set at 0, 30, 60 and 75°, and the recess depth was set at 0, 2, 4, 6, 8, 10, and 12 mm. The output from the strain gage load cell was recorded using a large scale integration system. The variation in the correction factor was minimal for all cases studied except for the 0 mm recess depth case with either an exit angle of 60 or 75°. A theoretical calculation was performed to determine the recess depth that would provide a recorded force that was 90% of the actual force, but no level of recess depth in this study produced this relationship other than when the exit angle was 0°. The asymptotic behavior of recorded versus actual force may signify the presence of a friction in the system, but the use of the correction factor will compensate for the frictional losses.

INTRODUCTION

Accurately representing the forces produced during in vitro and in vivo studies has proven to be a challenge for researchers; therefore many methods have been devised to overcome
the potential pitfalls of load measurement. An older method for measuring force restricted motion to only one plane and used displacement control to load the joint while the force was measured.[23] The same test was performed for two different conditions (for example: a normal knee and an posterior cruciate ligament (PCL) deficient knee), and the change in the force was attributed to the missing structure. Recent studies have advanced this method by allowing a full range of motion and measuring all of the resulting forces and motions, but the principle of superposition was still used to determine the forces.[26, 41] Other research groups have employed methods to only measure force in the structure of interest. An in vivo force transducer (IFT), previously proposed by this laboratory,[52] is implanted within the structure. As the structure is loaded, the IFT changes shape and force is recorded based on the shape change. However, the implanted IFT can lead to changes in the properties of the structure, and the IFT will only measure the local force not the overall force.[52] The use of strain gage load cells that sense force in one dimension has presented a new set of problems.[81, 87] While the load cells measure the overall force in the structure, the accuracy of the measurement depends on the angle between the line of action (LA) of the structure and the loading axis of the load cell (exit angle) as well as on the depth of the load application point within the force measurement system (recess depth). The belief is that the force measured by the load cell will decrease as the exit angle increase and as the recess depth decreases. The purpose of this study was to determine a correction factor for the strain gage load cell based on exit angle and recess depth.

METHODS

Study Design

The determination of the correction factor was performed using the force measurement system from the PCL bundle tension studies, Figure C.1. The force measuring system was
positioned so that the axial loading axis of the strain gage load cell was vertical. The load cell was calibrated to produce a relationship of 1 V for 31.2 N of force. The exit angle was set at 0, 30, 60 and 75°, and the recess depth was set at 0, 2, 4, 6, 8, 10, and 12 mm. A theoretical calculation was performed to determine the recess depth (9.3 mm) in which the load cell would record 90% of the force.

**Detailed Methods**

**Load Cell Calibration:** The force measurement system used in the PCL bundle tension study was placed in a wooden block so that the end of the femoral guide tube was exposed. The system was oriented so the measurement axis of the strain gage load cell (ELW-B1-200L, Entran Devices, Inc., Fairfield, NJ) was vertical, Figure C.1. An 11.2 lb load was attached to the thread rod using a nylon rope secured in a bone grip with polymethyl methacrylate. The load remained overnight to stabilize the load cell output. On the day of testing, the load cell was calibrated so a 1 volt output correlated to 31.2 N of force. The load cell was unloaded, and the zero point was set. The 11.2 lb load was applied, and the gain on the signal amplifier (2310 Strain Gage Conditioning Amplifier, Measurements Group, Inc., Raleigh, NC) was adjusted until the multimeter output read 1.6 volts. The load was removed, then reapplied, and if the multimeter output was not 1.6 volts, the gain on the signal amplifier was readjusted. If the multimeter output was 1.6 volts, the loading unloading cycle was repeated, and if the output was 1.6 volts, the 11.2 lb load was removed, and the system was reset. The 11.2 lb load was then reapplied, and if the multimeter output was 1.6 volts, the load cell was calibrated.

**Exit Angle:** The exit angles examined in this study (0, 30, 60 and 75°) were taken as a representative set of exit angles found for the femoral tunnels used in our PCL reconstruction study. The range of exit angles was calculated from the direction vectors for several surface
fibers of the native PCL and the direction vectors for the centerline of the femoral tunnels. The
direction vectors for the native PCL were determined by digitizing corresponding points on the
femoral footprint and the tibial footprint and cycling the knee from 0 to 120° of flexion. The
direction vectors for the femoral tunnels were calculation from photographs. The exit angles
were altered by changing the position of a frictionless pulley within a stainless steel frame.

**Recess Depth:** A theoretical calculation was performed to determine the depth in which
the load cell measured 90% of the actual force. The calculation was based on the geometry of
the femoral guide tube (the application point of the force is 4.5 mm from the edge of the guide
tube). A recess of 9.3 mm was found to produce the 90% relationship so the depths (0 mm to 12
mm by 2 mm increments) were chosen to verify the theoretical calculation and quantify the
behavior of the force measurement system. The 0 mm depth meant that the ends of the femoral
guide tube and the bone grips were flush, and a 2 mm depth meant that the bone grip had been
drawn into the femoral guide tube so that the ends were 2 mm apart. The depth increases were
produce by tightening the tensioning nut that rests on top of the load cell.
RESULTS

Table C.1: Correction factor for force output from strain gage load cell

<table>
<thead>
<tr>
<th>Exit Angle</th>
<th>0</th>
<th>30</th>
<th>60</th>
<th>75</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depth (mm)</td>
<td>0</td>
<td>2</td>
<td>4</td>
<td>6</td>
</tr>
<tr>
<td>0</td>
<td>1.01</td>
<td>1.02</td>
<td>1.04</td>
<td>1.01</td>
</tr>
<tr>
<td>2</td>
<td>1.30</td>
<td>1.23</td>
<td>1.23</td>
<td>1.21</td>
</tr>
<tr>
<td>4</td>
<td>2.21</td>
<td>1.58</td>
<td>1.42</td>
<td>1.31</td>
</tr>
<tr>
<td>6</td>
<td>5.33</td>
<td>1.68</td>
<td>1.60</td>
<td>1.35</td>
</tr>
<tr>
<td>8</td>
<td>1.01</td>
<td>1.04</td>
<td>1.04</td>
<td>1.04</td>
</tr>
<tr>
<td>10</td>
<td>1.30</td>
<td>1.23</td>
<td>1.34</td>
<td>1.35</td>
</tr>
<tr>
<td>12</td>
<td>1.21</td>
<td>1.11</td>
<td>1.33</td>
<td>1.35</td>
</tr>
</tbody>
</table>

The correction factor increased with increasing exit angle, but the increase was minimal except for the 60 and 75° exit angles with 0 mm of recess depth, Table C.1 and Figure C.2. The variation in correction factor due to recess depth was minimal (range: 29 to 66%, mean ± SD: 43 ± 14%) except for the 0 depth case in which the correction factor increased over 400%, Table C.1 and Figure C.3.

CONCLUSIONS

As the exit angle increase, the correction factor also increased, which was to be expected. For the exit angles of 60 and 75°, the correction factor decreased greatly when the recess depth was 2 mm or more. The change in correction factor due to recess depth was virtually nonexistent for depth of 6 mm or more. The greatest change in the correction factor was found in the 0 mm depth case, which was also an expected result, but the correction factor drastically decreased from 0 to 2 mm of recess depth for the 60 and 75° exit angles. The theoretical 9.3 mm of recess depth in which the measured force was 90% of the actual force was not verified in this study. The measured force for a recess depth of 6 mm or more averaged 83% of the actual force with none of the depths producing better than an 86% relationship. The inability to produce a 90%
relationship may be attributed to friction in the system because the data collected for the $0^\circ$ exit angle produced load cell outputs within 4% of the actual values.
Figure C.1. Force measurement system
Figure C.2 Change in the correction factor for the seven different recess depths
Figure C.3. Change in the correction factor for the four different flexion angles
Figure D.1. Complete femoral fixture for 5-mm wide bundles. Linear ball spline (Femoral: LT 6UU+150L; Tibial: LT 6UU+200L, THK CO., LTD., Elk Grove Village, IL), Load cell (ELW-B1-200L, Entran Devices, Inc., Fairfield, NJ)
Figure D.2. Femoral spacer
Figure D.3. Spline nut housing for femoral and tibial ends
Figure D.4. Femoral guide tube for 5-mm graft wide bundles

THE INNER SURFACE OF THE TUBE IS TO BE COATED WITH TEFLOM TO REDUCE THE COEFFICIENT OF FRICTION.
Figure D.5. Antirotation collar at femoral end for 5-mm wide bundles
**Figure D.6.** Femoral threaded rod
Figure D.7. Graft bone grip for 5-mm wide bundles
Figure D.8. Tibial spacer
Figure D.9. Tibial guide tube and femoral guide tube for 10-mm wide bundles
Figure D.10. Tibial threaded rod
Figure D.11. Bone grip for 10-mm wide bundles
APPENDIX E

MOTOR COMPONENTS AND DOCUMENTATION

COMPONENTS

Computer
- Parallel/Serial communications port
- DOS capability

Indexer (SLO-SYN Micro Series Model SPI-700 Programmable Indexer, Warner Electric, Bristol, CT)

Driver (SLO-SYN SS2000D6 Packaged Drive, Warner Electric, Bristol, CT)

Motor (M112-FF-206, Warner Electric, Bristol, CT)

Planetary Gear Head (PL42-020-M001, Warner Electric, Bristol, CT)

Motion Limiting Switches (V3L-1108-D8, MICRO SWITCH, Freeport, IL)

CONNECTIVITY

<table>
<thead>
<tr>
<th>Computer</th>
<th>Indexer</th>
</tr>
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<tbody>
<tr>
<td>25 pin connector</td>
<td>9 pin connector</td>
</tr>
<tr>
<td>Pin 2: Receive</td>
<td>Pin 2: Chain Out</td>
</tr>
<tr>
<td>Pin 3: Transmit</td>
<td>Pin 3: Receive</td>
</tr>
<tr>
<td>Pin 7: Reference Voltage</td>
<td>Pin 4: Reference Voltage</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Indexer</th>
<th>Driver</th>
</tr>
</thead>
<tbody>
<tr>
<td>10-pin fixed screw clamp terminal</td>
<td>8-pin screw clamp terminal</td>
</tr>
<tr>
<td>Pin 1: Opto</td>
<td>Pin 1: Opto</td>
</tr>
<tr>
<td>Pin 2: Pulse</td>
<td>Pin 2: Pulse</td>
</tr>
<tr>
<td>Pin 3: Direction</td>
<td>Pin 3: Dir</td>
</tr>
<tr>
<td>Pin 4: AWO</td>
<td>Pin 4: AWO</td>
</tr>
<tr>
<td>Pin 5: Reduce</td>
<td>Pin 5: RDCE</td>
</tr>
<tr>
<td>Pin 6: Boost</td>
<td>Pin 6: Boost</td>
</tr>
<tr>
<td>Pin 7: Reset</td>
<td>Pin 7: Reset</td>
</tr>
</tbody>
</table>

161
Driver | Motor
---|---
5-pin screw clamp terminal | 5-pin screw clamp terminal
Pin 1: Motor Phase A | Pin 1
Pin 2: Motor Phase A | Pin 3
Pin 3: Motor Phase B | Pin 4
Pin 4: Motor Phase B | Pin 5
Pin 5: Shield | Motor Casing
GND to Stand Base

Motion Limiting Switches | Indexer
---|---
3 terminal connector | 20-pin fixed screw clamp terminal
Clockwise | 
NO3 | Pin 2: Clockwise limit
COM1 | Pin 1: Common 1 (Reference Voltage)
Counter Clockwise | 
NO3 | Pin 3: Counter Clockwise Limit
COM1 | Pin 1: Common 1 (Reference Voltage)

**PROGRAMS**

Left.ms1 and Right.ms1 are the programs used to produce the cyclic fatigue tests. The following program is left.ms1.

N001 Call subroutine at line 50: Subroutine Repeat 2:
N002 Program stop:
N003 Call subroutine at line 50: Subroutine Repeat 3:
N004 Program stop:
N005 Call subroutine at line 50: Subroutine Repeat 7:
N006 Program stop:
N007 Call subroutine at line 50: Subroutine Repeat 15:
N008 Program stop:
N009 Call subroutine at line 50: Subroutine Repeat 31:
N010 Program stop:
N011 Call subroutine at line 50: Subroutine Repeat 63:
N012 Program stop:
N013 Call subroutine at line 50: Subroutine Repeat 127:
N014 Program stop:
N015 Call subroutine at line 50: Subroutine Repeat 127:
N016 Program stop:
N017 Call subroutine at line 50: Subroutine Repeat 127:
N018 Program stop:
N019 Call subroutine at line 50: Subroutine Repeat 127:
N020 Program stop:
N021 Call subroutine at line 50: Subroutine Repeat 127:
Licycle.ms1 and ricycle.ms1 are the programs used to produce the 50 cycles preconditioning for the intact knees. The following program is ricycle.ms1.

N001 Call subroutine at line 50: Subroutine Repeat 49:
N002 Program end:
N050 Boost current on:
N051 Move CW 1375 pulses: Speed 800 pulses/sec:
N052 Move CCW 1375 pulses: Speed 800 pulses/sec:
N053 Boost current off:
N054 Subroutine return:
N055
Lrcycle.ms1 and rrcycle.ms1 are the programs used to produce the 30 cycles preconditioning for the intact knees. The following program is licycle.ms1.

N001 Call subroutine at line 50: Subroutine Repeat 29:
N002 Program end:
N050 Boost current on:
N051 Move CCW 1375 pulses: Speed 800 pulses/sec:
N052 Move CW 1375 pulses: Speed 800 pulses/sec:
N053 Boost current off:
N054 Subroutine return:

AUTOCAD DRAWINGS FOR MOTOR SET-UP

Figure E.1. Base plate for motor and gear head
Figure E.2. Plate attached between the motor and gear head
Figure E.3. Plate attached to the front of the gear head
Figure E.4. Plate to hold limiting switches

<table>
<thead>
<tr>
<th>HOLE</th>
<th>RADIUS</th>
<th>ANGLE</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>45.4</td>
<td>+33.0</td>
</tr>
<tr>
<td>B</td>
<td>50.7</td>
<td>+61.9</td>
</tr>
<tr>
<td>C</td>
<td>37.1</td>
<td>+85.1</td>
</tr>
<tr>
<td>D</td>
<td>55.7</td>
<td>+104.9</td>
</tr>
</tbody>
</table>

- HOLES A-D ARE TO BE DRILLED THROUGH AND TAPPED M3X0.5, 8 PLACES
Figure E.5. Shaft to augment gear head shaft
Figure E.6. Collar that attaches gear head shaft to femoral grip base
Figure E.7. Pin to connect collar, Figure E.6, to shaft, Figure E.5[1, 11]