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A prospective study assessing patients after anterior cruciate ligament reconstruction with serial magnetic resonance imaging, genucom, kincom and subjective evaluation

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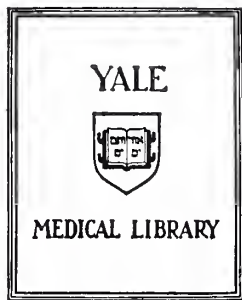
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A THESIS SUBMITTED TO YALE UNIVERSITY
SCHOOL OF MEDICINE IN PARTIAL FULFILLMENT
OF THE REQUIREMENTS FOR THE DEGREE OF
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ANN MARION SMITH
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A PROSPECTIVE STUDY ASSESSING PATIENTS AFTER ANTERIOR CRUCIATE LIGAMENT RECONSTRUCTION WITH SERIAL MAGNETIC RESONANCE IMAGING, GENUCOM, KINCOM, AND CLINICAL EVALUATIONS. Ann M. Smith, Christopher Pope, J.Kevin Lynch, and Peter Jokl. Departments of Orthopaedics and Diagnostic Radiology, Yale University, School of Medicine, New Haven, CT.

This prospective study was performed to follow changes in anterior cruciate ligament (ACL) grafts after reconstruction and to judge what role various instrument parameters might have in assessing the success of patellar tendon ACL reconstruction. Thirteen patients were followed after ACL reconstruction with serial testing that included Genucom (ligament laxity testing), KINCOM (muscle strength testing), magnetic resonance imaging (MRI) and clinical evaluations. Muscle strength testing was performed preoperatively and six and twelve months postoperatively. Clinical and ligament laxity evaluations were obtained preoperatively, and six, twelve and twenty-four months postoperatively. MRI evaluations were performed preoperatively, and 1.5, six, twelve and twenty-four months postoperatively. Data from these evaluations were analyzed with multiple regression analysis of variance (ANOVA) and paired T-tests.

There were a number of significant clinical findings in this study. Twenty-four months after ACL reconstruction, all patients had improved from their preoperative state as measured by ability to return to sports (p=0.0001). One patient developed significant ligamentous laxity twenty-four months after reconstruction. Changes in laxity did not correspond with changes in the clinical outcome. KINCOM testing at each of three different speeds, showed the involved quadriceps' strength increased over the follow-up period. A statistically

significant quadriceps deficit persisted even twelve months after reconstruction
($P<0.05$). No statistically significant relationship existed between the quadriceps
and hamstring strength and the patients' ability to return to athletic activities
($p>0.05$).

There were also a number of significant radiographic findings. All grafts
were identified on MR as intact at twenty-four months. Graft signal intensity was
most like normal tendon early after reconstructive surgery while little change in
the ACL graft occurred between six and twenty-four months. The clinical
outcome could not be predicted either on magnetic resonance signal or overall
appearance of the graft. Changes in the patellar tendon (the harvest site) and
menisci were detectable on MR.

Although parameters from MRI, Genucom, and KINCOM devices can be
used to detect changes after ACL reconstruction; these changes were not
predictive of clinical outcome. Further work is still needed to determine their
exact roles in management of patients following ACL reconstruction.

ANTERIOR CRUCIATE LIGAMENT RECONSTRUCTION

INTRODUCTION

Goals of Study

The purpose of this study was to examine the role of various instruments in following patients after anterior cruciate ligament (ACL) reconstruction. The growing interest in ACL reconstruction and rehabilitation has led to the development of new devices for monitoring patients. Despite numerous physicians' prescribing routine postoperative evaluations, the role of these assessments has not been established. This study was performed to determine if objective testing of ACL laxity, MR imaging, and muscle testing, would accurately predict clinical outcome.

Historical Background

In 170 AD Claudius Galen of Pergamum and Rome described ligaments as the "supporting structures of diathrodial joints, serving as stabilizers of these joints and limiting abnormal motion"(108). About 1700 years after this first description of ligaments, Stark described the rupture of the cruciate ligament (108)(111). In 1900, Battle discussed ACL repair. Although Battle is credited with the first report, Mayo Robson performed the first ACL repair and described his repair of anterior and posterior cruciate ligaments in 1903 (108)(101). Ten years later, Goetjes published a detailed study of ruptures of the cruciate ligaments (108). In 1917, Hey-Groves reported a reconstruction of the ACL using a strip of fascia lata. Hey-Groves' work more than 70 years ago is the basis of modern intra-articular reconstruction. The first extra-articular repair of the ACL was not reported until 1936 by Bosworth and Bosworth. Between 1917 and 1936, Alwyn Smith and Campbell made important contributions to the

understanding of the ACL by describing the ACL and mechanisms of injury (107). In 1936, Campbell first described the use of the patellar tendon as replacement for the ACL (13). Although these authors are credited for a great deal, it is O'Donoghue's presentation of ACL deficient athletes and details of his surgical technique that is credited as the impetus for the present era of research and treatment of the ACL deficient knee (95). These pioneers laid the foundation for modern day investigators to build an even greater understanding of the intricacies of the ACL. Only a few of the many investigators who have contributed to the present understanding of the anterior cruciate ligament have been mentioned. Despite the tremendous strides already made, questions and controversies about the ACL still persist.

Basic Concepts

Anatomy

Embryology

The cruciate ligaments appear in the human embryo by about 8 weeks of gestation from a condensation of mesenchyme. The posterior and anterior cruciate ligaments arise from the same tissue of origin as the menisci, although they remain extra-synovial at all times (28). The ACL begins to develop along with the femoral and tibial condyles between the 7th and 8th weeks of fetal life and is well defined by the 9th week (37)(54). Initially, the cruciates are highly cellular structures; however, by 15 weeks, they have become densely collagenous, and less cellular (54). By 20 weeks the cruciate ligaments and the menisci are fully formed (54)(Figures 1 and 2). By 27 weeks the greatest changes are increases in size rather than form. The ACL arises from the non-articular aspect of the tibia, passing

superiorly, laterally, and posteriorly to attach to the posterior portion of the intercondylar notch (10). Bony dysplasia of the intercondylar notch and hypoplasia of the tibial eminence are associated with congenital absence of the ACL (116). It is thought that the cruciate ligaments are of great significance in dictating the proper development of the articular surfaces, and thus joint motion and stability (50)(11).

Gross Anatomy

The ACL courses anteriorly, medially, and distally across the knee as it passes from the femur to the tibia. As it courses distally, the ligament turns on itself in a slight lateral spiral, which is a reflection of the orientation of its bony attachments (4). By examining 24 cadaver dissections, Girgis and his colleagues were able to study the detail of the anatomy of adult ACL's. Girgis et al. determined that "the ACL femoral attachment is semicircular in shape and averages 23 mm in length and is oriented in an oblique direction from the vertical, with its anterior edge almost straight and the posterior border convex, parallel to the articular margin of the condyle 4 mm off the articular surface. The tibial attachment begins 15 mm from the anterior border at the tibial articular surface between the tibial spines and averages 30 mm in length and gives off a slip to the anterior horn of the lateral meniscus and, in about 20% of specimens, also gives rise to a band which extends posteriorly to blend with the posterior horn of the lateral meniscus" (50) (Fig. 3). Girgis et al. noted the tibial attachment to be larger and stronger than the femoral attachment; however, Odensten and Gillquist's observations suggest the proximal and distal insertion sites are roughly the same size (50)(93) (Fig. 4). The average width of the ACL is about 11 mm, although the cross-sectional area varies along its length, being larger at its insertion sites than in the

mid-region. The average length of the ACL has varied among reports from 31 mm (93) to 38 mm (50). Odensten reported the approximate dimensions of the adult ACL: length 31 +/-3 mm, width 10 +/- 2 mm, and thickness 5 +/- 2 mm (93)(Figure 4).

Histology and Biochemistry

The number of anatomic divisions within the ACL is a rather controversial topic. Many authors describe two fascicles - a small anteromedial bundle and a large posterolateral bundle (50). Odensten and Gillquist disagree with the notion that any discrete ACL subdivisions exist on gross or microscopic inspection (93). Although investigators differ on the number of anatomical divisions, most agree there appears to be a functional arrangement of fascicles (Figure 5).

The ACL is made up of multiple fascicles, the basic unit of which is Type 1 collagen. Nonparallel, interlacing networks of these collagen fibrils (150-250 nm in diameter) are grouped into fibers (1-20 nm in diameter), which in turn compose a subfasciculi unit (100-250 nm in diameter). These subfascicular units are surrounded by a loose band of connective tissue known as the endotendon (4)(126). Three to twenty subfasciculi, bound together, form a fasciculus, which may range from 250 nm to several millimeters in diameter, and are surrounded by an epitendon. "These individual fascicles are oriented in a spiral fashion around the long axis of the ligament or pass directly from the femur to the tibial attachment. The entire continuum of fascicles, forming the ligament, is surrounded by the paratendon, a connective tissue covering similar to but much thicker than the epitendon" (4)(Figures 6 and 7).

The ACL attaches to the femur and tibia via the interdigitation of collagen fibers of the ligament with collagen fibers of the adjacent bone. The abrupt change from flexible ligamentous tissue to that of rigid bone is mediated by a transitional zone of fibrocartilage and mineralized fibrocartilage. A graduated change in stiffness and prevention of stress concentration at the attachment is a result of the alteration in structure from ligament to bone (4). "This zone may also impose a barrier to endosteal vessels entering the ligament at these attachment sites" (4).

Vascular Supply

In 1950, Gray and Gardner demonstrated in the embryo the vascular penetration of the ACL from the tibial and femoral attachment sites (54). Arnoczky has shown that the adult ACL appears to be supplied entirely by the vasculature of its synovial sheath, which forms an envelope over the ligaments (50)(4). The blood supply to the synovial sheath, and thus the ACL, is predominantly from the ligamentous branch of the middle genicular artery, with additional support from terminal branches of the inferior genicular arteries which reach the synovial sheath via the infrapatellar fat pad (4) (Fig. 8). The periligamentous vessels extend into the substance of the ACL. These branches anastomose with the endoligamentous vessels which course longitudinally within the ligament and lie parallel to the collagen bundles within the ligament (4)(Fig. 9, 10 and 11).

Nerve Supply

The ACL receives nerve fibers from branches of the tibial nerve. These fibers penetrate the capsule posteriorly and course along with the synovial and periligamentous vessels surrounding the ligament (4)(2) (Fig. 12). Kennedy et al. and Schultz et al. demonstrated neural elements on the

periphery and within the ACL. Although the majority of the ACL's neural elements appear to have a vasomotor function, investigators have demonstrated the presence of mechanoreceptors that look like Golgi tendon organs (104) or pacinian corpuscles (128) in the ligament. These mechanoreceptors possibly generate proprioceptive information which may protect the joint from damage and provide kinesthetic information (4)(104).

Biomechanical function

Various authors have described the ACL's biomechanical function. Most agree that one of its primary functions is as a check-rein resistance against abnormal anterior displacement of the tibia in relation to the femur in various degrees of knee flexion. Others describe its function by describing the actions of the two functional components, the anteromedial bundle (AMB) and the posterolateral bundle (PLB). Although the entire ligament functions as a unit, the AMB and PLB are taut and thus provide greater limitation of motion at different degrees of flexion and extension. The AMB is taut and resists the counteracting anterior luxation of the tibia at 90° of knee flexion. The posterolateral bundle (PLB) is taut and provides resistance against anterior displacement of the tibia in extension to 20° of flexion (47)(Fig. 5). Another important function of this ligament is the limitation of tibial rotation. The AMB and PLB work together to prevent internal rotation of the tibia when the knee is extended and slightly flexed (75). Clancy et al. emphasized that anterolateral instability is the more significant functional problem of an insufficient ACL. Other authors have emphasized the ACL's role as a "fine tuning" device that guides and controls knee motion, including rotation, especially rotation toward terminal extension, the so-called screw home phenomenon (26). Dye and

Canon have reported that the intact ACL acts as a block against hyperextension as it is forced against the intercondylar shelf, and may also be considered a secondary stabilizer to varus and valgus stresses (26)(biomechanical terms are defined in Appendix 11).

Feagin and Walton described the anterior cruciate ligament as "an essential stabilizer of the knee" and "critical to the function of the knee in a young athlete" (32). Butler, Grood and Noyes demonstrated the importance of the ACL as a primary stabilizer that seemed to become more important as loads approach the magnitude of stresses applied in functional activities. Butler and Grood determined the loads typically maintained by the ACL to be 169 N (38 lb) which increase to 400-500N (100lb) during strenuous activity (12). Their load analysis permits a better understanding of the ACL's clinical role in athletics.

Many factors work in conjunction with the ACL to provide stability of the knee. Ligamentous structures, osseous configuration, meniscal integrity, proper sequential muscle tension and load are all considered important in providing both functional control during ambulation and adequate support against stress. Knee position is crucial in determining the stress of particular loads on the ACL (50)(55)(59). Strain (elongation) in the ligament increases rapidly with extension past thirty degrees to fifty-five degrees; minimum strain is noted between thirty and thirty-five degrees of flexion (50)(59)(55).

Kapandj, Muller and others have described the basic kinematic principle of knee joint motion represented by the mechanism of the crossed four bar linkage. The ACL acts as one of the four bars of a system that produces a coupled rolling and gliding motion between the femur and tibia. The other three bars of the sytem in this model are the posterior cruciate

ligament, the femoral bone between the origin of the ACL and insertion of the PCL, and the tibial bone between the insertion of the ACL and the origin of the PCL (26). The two hinge points in the system must lie on a line at 40 degrees to the long axis of the femur, which corresponds to the 40 degrees formed by the long axis of the femur and the roof of the intercondylar notch. The lengths of the arms in the system correspond to relative lengths of the anterior and posterior cruciate ligaments (86)(69)(11). This system characterizes the obligatory motion of the joint surfaces, adhering to the rolling-gliding principle that predicts posterior shift of the contact point as the fusion occurs. This model allows evaluation of the consequences of an improperly inserted cruciate ligament (Fig. 13).

The ACL's function is determined not only by its position, but also by its properties. The ACL possesses a great deal of tensile strength and a degree of elasticity that allows it to function as a primary stabilizer in the knee. Dye and Canon noted that the nonparallel arrangements of multiple collagenous fibers, along with some associated elastin, seem to account for these properties (26). Noyes and his colleagues developed a hypothetical load-elongation curve that they believe characterizes the intact ACL-bone complex in young adults. "According to this concept, the ACL-bone complex is able to withstand approximately 1730 N or nearly 400 lb. of tensile force before complete rupture, with partial tearing of fibers occurring at lower levels of loading" (90). For most normal activities the ACL is loaded only to a range of about 454 N or 100 lb. Noyes and Grood (55) reported that the ACL can withstand up to 25 percent elongation without failure in young adults, and others have reported that there is a

decrease in the ability of the ligaments to withstand loads with increasing age (26).

Not only increasing age but also immobilization can decrease both the ultimate tensile strength and elasticity of the ACL. Immobilization seems to affect the ligament insertion sites more than the ligament itself by decreasing the tensile strength of the insertion sites. When the ligament is loaded beyond its maximum capacity, resultant tears may require reconstruction.

ACL Grafts

Sources for Autogenous ACL Grafts

In the past, most authors believed that the histology and biochemistry of ligaments were identical to those of tendons. Because tendons are commonly used as ligament grafts, a number of authors, including Amiel and his colleagues, investigated this theory. Using the rabbit model, they determined that relative to the patellar tendon and Achilles tendon, the cruciate ligaments tend to be highly cellular, more active metabolically, and contain less total collagen, a higher percentage of type 3 collagen, and collagen cross-linking (2). Using canine specimens, Yahia and Drouin demonstrated morphological differences and similarities between the patellar tendon and ACL. They showed that canine ACL and patellar tendon fascicles are characterized by a collagen waviness. They also found that the ACL's centrally located fascicles are either straight or undulated in a planar wave pattern while those located at the periphery are arranged in a helical wave. Unlike those in the ACL, in the patellar tendon all the peripheral and central fascicles possess an undulated helical wave pattern (126). Amiel and his colleagues also showed that the human ACL and the semitendinosus tendon are different in that collagen fiber bundles in the

ligament are oriented in a less parallel fashion than those of the tendon (1). They also believe that these findings suggest that ligaments may be better suited than tendons to adaptation based on activity, growth, and functional demands (1).

Despite the histologic, biochemical, and morphologic differences between tendons and ligaments, the rabbit model has shown that the ACL graft undergoes changes after reconstruction when autogenous patellar tendon is used. Through a process termed "ligamentization, the transferred tendon comes to resemble the normal ligament both histologically and biochemically, with ligamentous cell morphology, increased concentration of type 3 collagen, and more collagen cross-linking" (2). Amiel, Kleiner and their colleagues demonstrated that the cellular population of the rabbit autograft is derived entirely from a source other than the patellar tendon fibroblasts (2). They also showed that a critical period exists during the first three postoperative weeks. During this period, patellar tendon fibroblasts die and are replaced by cells from an extra-graft source. The migration of fibroblasts and restoration of the cellularity of the autograft occur without an autograft blood supply (2). This ligamentization may allow the graft to behave more like the native ACL. By analyzing human ACL graft biopsy specimens, Frank, Woo, and others found that in human ACL grafts, collagen fibrils less than 100 nm in diameter are the major contributors to the collagen fibril cross-sectional area, regardless of length of time in vivo (nine months to 6 years)(43). The patellar tendon grafts consist not only of fewer large diameter fibrils (100 nm) than the normal human patellar tendon but also more large diameter fibrils than the normal human ACL. The patients from whom the biopsies were taken all had progressive laxity of the ACL; however, the collagen fibril population did

not alter as much for the older grafts. These findings are consistent with the theory that ACL grafts stretch out over time.

Vascular Supply of ACL Grafts

It is important for orthopaedists to appreciate the differences in revascularization following reconstruction. When no bone tunnels are utilized, revascularization in the dog model begins at 6 weeks and is complete by 20 weeks following reconstruction (95). Reconstructive methods that utilize bone tunnels for graft placement appear to lead to earlier revascularization of the transferred tissue. This appears to be from the endosteal circulation of the bone tunnels with completion of the revascularization process by 8 weeks (15)(Fig. 15, 16 and 17). The synovium and fat pad may serve as potential sources for revascularization of repaired ligaments and reconstructive grafts. Vascular penetration of reconstructive grafts in the animal model also appears to be affected adversely by lack of bone tunnels and by application of excessive tension applied at the time of fixation (127). The optimal preload is not known, and may depend on the graft source (11).

Biomechanics of ACL Grafts

Noyes and his colleagues compared the mechanical properties of the ACL with those of various other tissues about the knee using animal specimens and failure tests (90). They found that 14 mm wide patellar tendon specimens are about 60% stronger than the ACL, but almost four times stiffer. The semitendinosus possesses 70% of the strength of the ligament, but is almost identical to the ACL in stiffness. The gracilis tendon's stiffness is also similar to the ACL, but possesses only 49% of the strength of the ACL. The fascia lata has only 36% of the ultimate strength of the ACL, and is significantly less stiff (90). One of the reasons the

semitendinosus and patellar tendon are the most common graft sources is their properties are those most similar to the ACL's.

Disruption of the ACL can result in altered kinematics and often subsequent degenerative changes, probably occurring not only because of its lack of structural integrity but also because of disruption of its proprioceptive function (26). The complexity of this ligament and associated normal kinematics of the knee present a challenge to orthopaedists attempting to reconstitute the function of ACL insufficient knees (26).

Clinical Concepts

Incidence and Mechanism of Injury

The ACL continues to be the most frequently injured ligament in the knee joint (80); however, Garrick claims that "we have virtually no idea of the frequency of ACL injury" since cases are lost (unreported) at many points in the chain of events between the occurrence and treatment of the injury (36). Johnson found that 1.2% of college freshmen were ACL deficient at routine entrance examination (66). There are five mechanisms of injury into which almost all of these commonly encountered injuries are classified:

1. Forced hyperextension of the knee
2. Hyperextension with rotation of the tibia on the femur
3. Forced flexion and external rotation of the tibia on the femur
4. Anteriorly directed force applied to posterior aspect of the proximal tibia with knee extended or flexed
5. Forced hyperflexion of the knee

The most common mechanism includes some component of knee hyperextension. This action may occur with or without rotation of the tibia. Feagin mentions that a large number of ACL lesions result from a deceleration motion with the foot planted and the knee in slight flexion (32). The injury may occur without a rotational component, but is most often seen with internal rotation of the tibia (32). This particular mechanism is often described as the cause of isolated ruptures of the ACL. Hyperextension injuries are also seen in contact sports such as football and hockey, the injury resulting from a posteriorly directed force applied to the anterior aspect of the femur - usually while the foot is planted. Gersoff and Clancy have described injuries involving hyperextension of the knee while the athlete was non-weightbearing (48). "An example of this injury is the basketball player, who coming down from a rebound, has the involved foot land on the top of someone's foot, producing hyperextension of that knee. This mechanism is also seen in gymnasts when they miss their dismount and hyperextend their knees" (48). Other authors have described ACL lesions as a result of a forceful contraction of the quadricep muscle group while the knee is in extension. This injury may be seen in skiers producing a tremendous quadricep contraction to prevent falling (48). The majority of hyperextension injuries do not involve direct contact from an external force.

A less-reported mechanism of ACL injury is knee flexion accompanied by external rotation of the tibia on the femur. This injury is sometimes seen in contact injuries involving a valgus load, which first tears the medial collateral ligament and often the posterior medial capsule and, if there is sufficient force the ACL (O'Donoghue's triad)(94). The clipping type force seen in football may disrupt the ACL with an anteriorly directed

force applied to the posterior aspect of the proximal tibia, while the knee is in flexion or extension (9). Hyperflexion is a much less common cause of injury described much less frequently in the literature. Fetto and Marshall defined the force of hyperflexion of the knee as that which may occur during sports that require jumping and landing (15).

Definition of ACL Injuries

ACL injuries are classified as acute or chronic. All injuries begin as acute tears which are managed either surgically or conservatively. Acute and chronic ACL injuries are clinically two different entities. All acute injuries can potentially become chronic ACL injuries and all chronic ACL injuries were once acute ACL injuries. It is predominantly the response to management after the initial insult to the knee which dictates which type of knee injury the knee will be classified. Acute ACL tears are often surgically reconstructed before stretching and tearing of secondary restraints begin which is usually within twelve months after the initial injury. These secondary restraints, which are often damaged in chronic ACL injuries, include the medial collateral ligament, lateral collateral ligament, the anterolateral femorotibial ligament, arcuate-popliteus corner including the lateral meniscus, and the semimembranosus corner including the medial meniscus (36). Acute ACL injuries become chronic knee injuries after enough time has passed to permit stretching and tearing of secondary restraints, particularly the menisci, and the initiating of arthritic processes (33). The progression to a chronic ACL injury can result from inadequate surgical intervention, unsuccessful conservative management, misdiagnosed initial injury, or failure to seek medical care. Not all ACL tears clinically become chronically ACL deficient knees; however, what determines which knees will respond favorably to which mode of

management is not known. Similarly, what determines which acute injuries develop into chronic ACL insufficiency is not understood. Several factors contribute to this ignorance. The most obvious reason that investigators do not completely understand the development of chonic ACL insufficiency is the lack of research prospectively studying the evolution of knee injuries over time. Since there is inadequate evidence supporting different modes of management for particular clinical presentations, the decision-making is predominantly influenced by the orthopaedist's experience.

Both acute loss and chronic insufficiency of the ACL function result in instability and giving way (41). Both entities lead to alterations in the fluid or gliding mechanics of the knee joint. Patients do not usually seek medical care until these alterations prove inconvenient or disabling. Patient's who are limited but do not feel restricted as a result of an ACL injury might not seek the advice of an orthopaedic surgeon.

Diagnosis

History

"For acute injuries the essential factors are diagnosis of the ACL injury and associated injuries, and choosing surgical or nonsurgical management" (20). In chronic cases, diagnosis of the ACL lesion is usually not difficult, or has already been made, and management depends on the presence of functional instability and a major meniscus tear (20).

Although acute and chronic knee injuries are often managed and present quite differently, a thorough history and skilled physical examination should be obtained for both. The first step in diagnosing an injury to the ACL is eliciting a complete and accurate history from the patient. It is important to inquire about the chronic knee's history since the

time of the initial insult. There are a few "classic" mechanisms, signs, and symptoms which may aid the examiner in identifying injuries to the ligament. The most common mechanisms of injury involve a rotational component while decelerating on an extended knee. An audible "pop" can often be heard in the case of an acute injury and if there is a significant tear of the ligament, the acute injury is usually accompanied by measurable swelling (10)(32). "In adult athletes, disruption of the ACL is the most common cause of an immediate effusion. One of the best predictors of an ACL tear is a history of a sudden, intense swelling" (88). A hemarthrosis that presents itself within the first 12 hours after injury is highly suggestive yet not pathognomonic for an ACL lesion. It is necessary to inquire about any previous ACL or other knee injuries (48). Identifying the activity at the time of the injury may make the diagnosis obvious since there are particular mechanisms of injury associated with ACL injuries.

Physical Examination/Clinical Tests

The second diagnostic step is a physical examination tailored to the mechanism of injury. In the acute setting muscle spasm and pain limit the physical examination. The examination should follow a logical progression that starts with the least painful test and ends with the most painful test (48)(33). Although the knee should be inspected for edema, ecchymosis and palpated for tenderness, the clinical stress tests usually provide the most information about a patient's ligaments. There are numerous tests for evaluating the integrity of this structure (Figure 18). The most frequently used tests to evaluate the ACL are the anterior drawer, Lachman and pivot shift tests described in the illustrations (Fig. 19, 20A, 20B and 21). Although rotational tests may be reliable in some cases, the anterior drawer test and the Lachman's test seem to be the most conclusive and reliable in

detecting ACL lesions (71). Until recently, the anterior drawer test was the most widely used test for evaluating the ACL (71). The Lachman test has recently proven to be the most accurate examination for anterior cruciate insufficiency in the acute setting, with sensitivity reported from 87% to 98% (24) (36). Accuracy of the Lachman test rises to 100% when anesthesia is used. Once the Lachman and the anterior drawer test have been performed the pivot shift test should be carried out (36). A positive result - a sudden anterior subluxation of the lateral tibial plateau as the knee is flexed 15 to 20 degrees - is very strong evidence of at least anterior cruciate damage. These tests should be performed on both knees since there is a great deal of anatomic variance in knees. Each test has its limitations, exacerbated in the acute setting in which muscle spasm and pain often preclude thoroughness.

Instrumented Tests

Many have attempted to use instrumented measurements of anterior laxity in the knee to quantitate the physical examination in order to obtain greater consistency among examiners. Instrumented clinical test devices measure displacement of the tibia relative to the femur as a force is applied to the tibia. Many authors feel instrumented devices will become an important tool for the orthopaedist, since visual estimation of motion and the clinical exam are often too subjective. Since 1969, when Quellet used x-ray overlays to measure tibial translation as a standardized force was applied by a weight on a pulley, a number of instrumented devices to provide data on knee laxity have been developed (38)(82). Currently, the Stryker, KT1000 (an earlier version of the KT2000), KT2000 and Genucom devices are most commonly used. Although all of these arthrometers can measure anterior displacements under anterior-posterior

(AP) forces, only the Genucom system measures the complete three-dimensional motion of the tibia with respect to the femur. In other words, the Genucom measures three-dimensional tibio-femoral motion in all six degrees of freedom including anterior-posterior, medial-lateral and superior-inferior, while the KT1000, KT2000 and Stryker measure motion only in the anterior-posterior direction (Figures 22 and 23). The Genucom's day-to-day variability for individual patients demonstrated in 1990, by Wroble and his colleagues, makes this seemingly versatile arthrometer less attractive as a diagnostic tool (125). Markoff and Amstutz argue that the stiffness and laxity as calculated by instrumented clinical knee testing devices could be used to detect the presence or absence of an intact anterior cruciate ligament in a noninvasive manner; thus, they could assist the physician in making diagnostic and management decisions (82).

A brief description of the Genucom knee analysis system is included in text; a more thorough description of the system and definitions of tests are included in Appendix 3. Faro developed the Genucom knee analysis system to provide the orthopaedic community with a means of obtaining objective quantitative information (61). The computerized system involves the localizing and digitizing of anatomical landmarks and then conducting a series of knee evaluation tests. The patients' data are stored on disks and printed for evaluation and diagnosis (61). The patient sits in the Genucom seat while the certified physical therapist operates the digitizer and electrogoniometer as depicted in Figure 23. The patient is put through a battery of tests similar to those performed by the orthopaedist in the examining room. The motion in the knee joint is measured by the system and translated by the computer into graphs and tables (Figures 24 and

24A). The principal diagnostic criterion is the comparable laxity of the knees.

<u>Imaging</u>

Evaluation of an ACL injury can also be assisted with diagnostic imaging. Roentograms of the acute anterior cruciate deficient knee may be normal except for the presence of an effusion (hemarthrosis)(97). In 2-4 percent of cases an avulsion of the tibial insertion may be identified as an osseous fragment superior and anterior to the tibial spines on either the lateral or tunnel views (11). Notch defects and lateral capsular sign (Segond's fracture) may be seen on radiographs of knees with an ACL injury but are not diagnostic.

While plain films of the knee can sometimes be helpful in the diagnosis, it is magnetic resonance imaging (MRI) which often plays a significant role in the diagnosis of ACL injury. This non-invasive mode of evaluating knee ligament injuries has recently become more popular. In 1986, Wojtys and his colleagues acknowledged its great potential to provide noninvasively detailed anatomical information about cruciate ligaments and menisci (121)

Investigators have utilized MRI to study the anterior cruciate ligament. In 1987, Jackson and his colleagues reported the accuracy defined as the percentage of patients correctly diagnosed with an ACL lesion using MR to be 96.6% (65). Jackson et al. argue that the results of their study demonstrated that "MRI is highly accurate in the diagnostic assessment of patients with suspected tears of the menisci and cruciate ligaments"; however, these authors did not differentiate between acute and chronic ACL tears (65). In 1989, Glashow and his colleagues reported a 95% accuracy rate for detection of complete tears of the anterior cruciate

ligament; however, once again the differentiation between acute and chronic was not made (51). While many authors support the use of MRI in the diagnosis of knee pathology, the need for more studies correlating MRI findings with arthroscopic findings and MRI with clinical assessments and knee stability tests is well recognized (100).

It is important to understand that the MRI appearance of an ACL depends on the age of the lesion and degree of disruption (83). Acute and chronic ACL tears possess different magnetic resonance imaging chracteristics. In an acute tear, the ACL is either clearly discontinuous or demonstrates a serpiginous or grossly concave anterior margin (83). In acute partial or complete tears, T1 weighted images may demonstrate a mass of intermediate signal most often found at the proximal end of the tendon, with or without an identifiable discontinuity of the tendon itself. The intermediate signal mass consists of fluid, hemorrhage and acute synovitis, which often increases in signal on T2-weighted images (83). Chronic ACL tears may manifest only a small remnant of tissue in the expected position of the anterior cruciate ligament. With chronic anterior cruciate deficiency, detection of meniscal pathology and articular surface damage is common with MRI scans (16)(11).

Magnetic resonance imaging currently plays an important role in the diagnosis of ACL injuries, and other related pathology, especially in the acute setting in which the clinical exam is limited by pain and muscle spasm. Diagnosis of the ACL injury should be based not only on MRI findings but also on the physical exam and history elicited from the patient.

Arthroscopy

Arthroscopy plays an important role in diagnosing ACL injuries despite the development of other modalities. Arthroscopy in the hands of a skilled orthopaedist is considered the standard to which other modalities are compared. Diagnosis of an ACL injury acute or chronic as well as diagnosis of a meniscal injury can normally be made by arthroscopy. The major disadvantage of arthroscopy is that it is invasive which makes the non-invasive MRI more attractive especially in the setting in which the patient opposes surgical intervention. In addition, some meniscal injuries such as anterior horn and posterior horn tears of the inferior surface may be missed on routine arthroscopy.

Management

Once the diagnosis of an ACL injury has been made, decisions regarding the management of ACL deficient knees need to be based on the expected course of the ACL deficient knee (11). It appears that most patients with acute ACL injuries who do not undergo surgery, do return to sporting activities, but the level and intensity to which they return is often diminished. The percentage of patients with ACL injuries who return to sporting activities is unknown, since asymptomatic patients do not typically seek medical care. Feagin emphasizes the integrated approach to each injury, with consideration of the patient's age, personality and goals (34).

The patient with a chronically ACL deficient knee usually falls into one of two categories: (1) a patient who was not correctly diagnosed with an ACL injury or (2) a patient correctly diagnosed with an ACL injury but failed conservative treatment. Patients who remain stable after undergoing conservative therapy do not typically seek the continuing care of the

orthopaedist. The patients who are not satisfied with conservative therapy often return to the orthopaedist for additional advice and therapy.

Many of the same devices used to diagnose chronic and acute knee injuries can also be used to follow the patient after the initial injury: physical exam with manual tests, patient's progress with evaluation forms, instrumented testing with KT2000, Stryker, or Genucom and the KINCOM. The KINCOM is a muscle testing device, which can be used to monitor the strength of the quadriceps and hamstrings. Treatment of the acute or chronic lesion in the ACL may be the most controversial area in sports medicine at the present time (34). Once an accurate diagnosis of the condition has been established, there are a number of treatment plans which may be implemented. The surgeon and patient may prefer surgical intervention or the patient and surgeon may decide to attempt nonsurgical management. Nonsurgical management should but often does not include strict rehabilitation with monitoring of progress. It is important to monitor all patients with ACL tears whether they opt for surgical or conservative management to assess the patient's response to therapy.

It is difficult to predict which patients will experience chronic anterior cruciate ligament deficiency, which typically presents with internal joint derangements, degenerative changes, and functional instability. Cabaud and Rodkey claim that up to sixty percent of patients with symptomatic chronic instability can avoid reconstructive surgical procedures by rehabilitation and activity modification (41). Although not all orthopaedists agree, Cabaud claims: "Despite the unpredictability of improvement with such a rehabilitation program, it is imperative that such a program be attempted before surgery is considered"(41).

"The deranged knee of the patient with chronic symptomatic anterior cruciate deficiency is exposed repeatedly to abnormal stresses. The risk of reinjury is high"(41). Mechanical alterations caused by loose bodies, torn menisci, or other problems may be treated arthroscopically. For the individual with symptomatic instability who desires or needs surgical correction, the age, sex, occupation, ability, and long-term expectations must be given thorough consideration before the operative procedure is undertaken (41).

Conservative Management

Every patient with ACL insufficiency should be followed by an orthopaedist and physical therapist. This monitoring allows the orthopaedist to advise the patient either to undergo surgical intervention or continue conservative management. Utilizing results from the tests as well as the patient's self-reports and the physical exam, orthopaedists could manage the ACL deficient knees in a more systematic fashion. Unfortunately, it is not usually practical or economical for every patient with an ACL injury to be monitored closely over time to assess the success of conservative management. Since ACL deficient knees are rarely followed with objective testing, little has been reported about how to use these parameters for managing ACL deficient knees. Optimally, the ligament and muscle testing (and possibly MRI) should be used to follow all ACL deficient knees to discern objectively which knees require surgical intervention.

Very little research has focused on following knees after an ACL injury with such devices, thus the literature fails to provide orthopaedists with guidelines for using such objective measurements. A number of investigators have attempted to study the natural history of ACL deficient

knees; however, they have measured the changes over time with diverse methods, which makes it difficult to compare studies. Since no standard for observing changes in the ACL has been implemented, physicians are forced to compare results of markedly different studies. In order to minimize the uncertainty plaguing the management of ACL deficient knees, research using instrumented devices and MRI to observe changes in the ACL deficient knee needs to be conducted.

The KT2000 and the Stryker, popular devices used to measure objectively ligament laxity, and the Genucom a less popular and reportedly less accurate device (125), are starting to take on a new important role in the management of knee injuries. In the hands of a qualified examiner, instrumented devices can provide impartial, objective measurements that quantitate the degree and type of instability (3). These machines have been most useful and accurate in measuring anterior-posterior displacements and forces. Instrumented measurement of anterior laxity can be used not only to follow a patient with a chronic knee injury and assist management decision making, but also to compare a knee's preoperative laxity to the post-operative laxity if the patient does decide to undergo surgery. Markoff and Amstutz concluded that "as sports medicine matures as a scientific discipline, improved instrumented test devices may ultimately provide a standardized means for reporting knee stability parameters" (82).

Since the knee's ability to function is influenced by a number of factors including muscular strength, many compare the major dynamic knee stabilizers of involved and non-involved legs while managing a patient with an ACL deficiency. The quadriceps femoris and hamstring muscles are the major dynamic knee stabilizers. In fact, it is these muscle groups which therapists stress in patient's being managed conservatively for an

ACL injury. An important factor in managing an ACL deficient patient is the muscular strength of hamstrings and quadriceps.

Each of these objective aids should be utilized to assist the orthopedist to discern which patients will develop chronic instability and benefit from reconstructive surgical procedures. Although not all orthopaedists agree as to when surgical intervention should be implemented, some authors advocate ACL reconstruction prior to the knee's becoming chronically ACL insufficient. Most orthopaedists agree surgery should be undertaken when there is objective evidence of deformation and destruction of the secondary restraints, particularly the menisci (33).

"There are indeed many different presentations and management philosophies for the symptomatic anterior cruciate deficient knee. Conservative management, although diversified, does play a major role in treatment for this difficult problem" (89). Managing patients with an ACL injury has been a challenge to orthopaedic specialists for decades (104). The goal of ACL injured patients is to prevent progressive deterioration of the knee joint and return individuals to their preinjury functional status (105). When this goal is not met by conservative management many patients opt for surgical intervention (105). It is also important to remember that "in the case of a younger, active individual, with a significant lesion of the ACL, the nonsurgical approach may lead to future complications" (1). If the patient returns to the physician with a history of recurrent episodes of "giving way," pain, locking and effusion despite compliance with a supervised rehabilitation program, the patient should be considered for ACL reconstruction.

Surgical Management

The management of patients with chronic ACL insufficiency is often difficult. Indications for minimal surgical intervention include mild to moderate laxity with mild to moderate demands on the knee, and clinical indications of an associated meniscus tear. Minimal surgical intervention includes evaluation under anesthesia (EUA) to assess more completely the types and degree of laxity as well as arthroscopic examination to determine the type of meniscus tear and to document the extent of articular damage. About 40% of meniscus repairs without ligamentous stabilization will tear again (21)(20). After repairing meniscal lesions, patients once again should be managed with supervised rehabilitation including periodic assessments with mechanical devices. "The indications for ACL reconstruction include functional instability, moderate or severe laxity in high-demand knees, severe laxity in knees with lower demand and failure of the nonsurgical management or minimal intervention surgery" (20).

"Most of the patients with ACL instability are candidates for surgical intervention. Many of these patients present because of persisting and perhaps increasing problems due to the cruciate instability, which leads to further destruction of the menisci or articular cartilages within the knee. Accordingly, one should seriously consider this group of patients as candidates for surgery" (36). The indications for ACL reconstruction are also dependent upon age, level of demand that will be placed upon the knee, degree of laxity of the ligament, and desire, commitment and motivation displayed by the patient to go through necessary steps involved with surgery (36).

Types of Reconstruction

The literature is replete with descriptions and outcomes of various types of reconstruction. The plethora of articles discussing different types of reconstruction with various outcomes reflects the controversy surrounding this topic. Jackson claims that the type of reconstruction depends upon the "individual surgeon's preference and the techniques with which he or she is most familiar" (36).

All of the reconstructions can be classified as intra-articular, extra-articular or a combination of these types. The intra-articular approach can be further divided into primary repair, substitution of autologous structures, and the use of prosthetic materials. Of these approaches orthopaedists have had the greatest success with the intra-articular approach with certain inert structures simulating the function of the ACL. The most common autologous structures utilized are the patellar tendon (74)(64)(91)(68)(44), the iliotibial band (44)(87)(91), the menisci (18)(118)(120), and the semitendinosus tendon (46)(110)(39). Autologous structures such as the patellar tendon and semitendinosus are more suitable to survive the stress placed on the repair in the early rehabilitation. Engrebretsen recently found patients with patellar tendon ACL reconstruction to have results superior to patients with primary repair and patients with Kennedy Ligament Augmentation Device (29). Proper revascularization and correct anatomical placement of the structures dictate the eventual outcome of the surgery (5). The general success of repairs involving the use of prosthetic ligaments has been unfavorable (52). More recently allograft ligaments have been used in ACL reconstruction and will most likely continue to take on an increasing role; however, it is too early

to assess the long-term outcome of patients after allograft ACL reconstruction.

Extra-articular repairs also involve the use of autogenous structures to provide stability to the knee joint. The most commonly used structures are the iliotibial band, biceps femoris, and the pes anserinus group (78)(27). Orthopaedists have had some success with extra-articular repair procedures (25). The most common problem is that the primary sutures used in this type of surgery often stretch out during rehabilitation and activities, thus leaving the patient with residual instability (70). Some orthopaedists augment intra-articular reconstruction with extra-articular stabilization. Roth et al. concluded that advancement of the biceps femoris tendon for extra-articular augmentation does not improve the efficacy of an intra-articular reconstruction of the ACL. Strum et al. also found no additional benefit from combined intra-articular and extra-articular stabilization when compared to intra-articular substitution alone (113)(105). Studies assessing the long term success of ACL reconstruction have just begun. The results of studies prospectively examining the success or failure of the various surgical repair procedures, will help determine the true efficacy of each procedure. The type of reconstruction should be made with respect to the patient's needs and the othopaedist's preferred method of reconstruction.

Patellar Tendon Reconstruction

Since it is still a matter of debate which type of reconstruction should be performed, the surgeons participating in this study chose intra-articular reconstruction using the middle one-third of patellar tendon of the involved knee (Figures 25, 26 and 27). It is this operation which will be discussed more thoroughly, although the possibility of performing other operations

is well recognized by the investigators participating in this study. The procedure used by the two surgeons participating in this study is that operation described best by Clancy in 1988. Most of the description of this procedure is paraphrased or taken verbatim from Clancy's procedure (17). The procedure and modifications of this method are also described in Appendix 1.

Rehabilitation

Proper rehabilitation is critical in determining successful outcomes of patients after ACL reconstruction. The purpose of rehabilitation is the same following nonsurgical and surgical treatment. The goals include prevention of progressive instability, postponement of the onset of degenerative changes, reinstatement of the preinjury level of performance, and protection from re-injury (71). If by any of the parameters measured by clinical exam or other devices, the patient's rehabilitation is not adequate, modifications in the patient's rehabilitation should be made accordingly. In order to know when adjustments in rehabilitation should be made, a thorough understanding of the expected outcome of the surgical procedure is necessary.

Success of Reconstruction

Clinical assessment

After a patient undergoes ACL reconstructon, it is the responsibility of the orthopaedic surgeon and the physical therapist to monitor the patient. By following a patient's progress with an evaluation form, it is easier to evaluate changes in the patient's clinical status. Optimally, preoperative measurements should be available to compare the patient's preoperative and postoperative parameters. Evaluation forms can be used to monitor the patient's clinical status and to evaluate the overall

success of the patient's reconstructive surgery and rehabilitation. The form should be used to record stability, function, symptoms, and physician's assessment. The form should also allow the physician to record systematically the patient's physical exam, including the battery of clinical tests used to diagnose the ACL tear. Feagin, Noyes, Grood, the Hospital for Special Surgery and many others have developed different forms for recording a patient's clinical status. Each form has advantages and disadvantages; unfortunately, orthopaedic surgeons have not yet accepted one particular evaluation form for following their patients with ACL injuries or ACL reconstruction. If a form were universally accepted, factors determining which patients are at greatest risk for developing ACL insufficiency and graft failure potentially could be established. Until the orthopaedic community has accepted one particular form, the burden of selecting an evaluation form is on the individual orthopaedist. Once the form or method for monitoring patients is chosen it should be completed or performed 2 weeks, 6 weeks, 12 weeks, 12 months and possibly 24 months after the reconstructive surgery (36).

Instrumented testing

Genucom

After ACL reconstruction patients should start supervised rehabilitation. In conjunction with this rehabilitation, the patient's progress should be monitored by the orthopaedic surgeon. The clinical exam and assessment of the patient's functional abilities provide a great deal of information to the orthopaedist about the patient's postoperative status. In addition to the clinical exam, instrumented devices can be used to aid the physician's management of his patients. These include Genucom or some

other ligament laxity measuring device and KINCOM or some other muscle testing device.

Since the goal of knee ligament reconstruction is to reestablish the normal knee laxity that was present in the patient's knee prior to the initial injury, comparison between the patient's non-injured knee and the involved knee before and after reconstruction is made. In order to compare knees one must recognize there is asymmetric laxity between knees even in an individual's normal knees (81)(115). Not only can clinical exams be used to make this comparison, but also instrumented measurements of knee laxity can be used to make this comparison and to monitor the patient's progress after surgery. If the device is used properly, controlling for variables such as knee flexion angle, knee rotation angle, muscle relaxation, and displacement force and direction (79), then long-term follow-up measurements can provide information on the evolution of the knee laxity after reconstructive surgery. As progress is made in the field of instrumented knee laxity testing, objective measurements will be used to monitor and manage patients after ACL reconstruction.

KINCOM

Measuring the strength of the dynamic knee stabilizers can also provide information about the patient's response to therapy. The KINCOM is used for strength testing and as an exercise device. The KINCOM is used in orthopaedics, physical therapy, and other similar fields, including research. It competes primarily with isokinetic devices such as the Cybex II (31). Kinetic communicator exercise system (KINCOM) is a hydraulically driven, microcomputer-controlled device for the testing, measuring, and rehabilitating of human joint function. The

KINCOM user performs a movement, or series of movements, against a resistance which the machine provides via a rotating lever arm system. The machine-controlled movement modes include isokinetic, semi-isotonic (lever arm speed is continually adjusted to maintain constant resistance), and passive joint movement. The unit can measure concentric, eccentric, or isometric contraction of the involved muscles (31). The device utilizes a strain gauge bridge for force measurement and a bar-encoded shaft for position and speed measurements (31)(see Appendix 10). Farrel and Richard reported the KINCOM produces valid and reliable measurement of the condition of the lever arm and the strain gauge systems (31).

KINCOM can be used as an integral part of a patient's rehabilitation and postoperative management. Using an instrumented device to measure muscular strength, Seto and others found a significant correlation between increased quadriceps and hamstring strength on the operated leg and return to functional activities (105). LoPresti and his colleagues showed that the quadriceps of the involved leg remains signficantly smaller than the quadriceps of the opposite leg one year postoperation (76). Lorentzon et al. published an interesting study examining the structure and function of the thigh musculature in patients who have chronic ACL insufficiency. They found a decrease in isokinetic performance of the quadriceps muscles in their patients, which could best be explained by a decreased activation of normally functioning muscle fibers of the quadriceps muscle caused by altered sensory feedback from the mechanoreceptors of the torn ACL (77). Giove et al. found that higher levels of sports participation were found in the patients whose hamstrings strength was equal to or greater than their quadriceps strength (49). KINCOM could be used to compare a patient's progress to the anticipated

results as defined by Lorentzon, Giove and others (77). The results from testing patients after ACL reconstruction with the KINCOM may contribute new information to the field of orthopaedics and rehabilitation, since little is known about the eccentric contractions of the quadriceps and hamstrings (60). Using the observations made by these investigators, the KINCOM can be used to monitor and manage patients after ACL reconstruction. The results from testing hamstrings and quadriceps strength on each patient could be used to tailor a patient's rehabilitation if the patient's strength is not progressing as expected.

Magnetic Resonance Imaging

Magnetic resonance imaging's role in the diagnosis of knee pathology has been well established (51)(83)(85)(100)(123). MR imaging of the knee has permitted greater accuracy of diagnosing ACL injuries; however, MR's role in management of reconstructed knees has not been established. The ACL graft has been described by Moeser as appearing different from a normal ACL and variable in visualization by MR (10). Howell et al. described the appearance of hamstring anterior cruciate ligament autografts during the first year of implantation. Unlike Moeser et al., Howell et al. were able to visualize and describe the graft at all time intervals after reconstruction (63). Howell noted a time-dependent increase in graft signal and was not able to predict the clinical outcome based on MR findings (63).

Previously the success of anterior cruciate ligament (ACL) reconstruction has been evaluated by clinical exam, functional ability and follow-up arthroscopy (3)(8)(18)(29)(58). One of the controversies concerning ACL reconstruction involves the expected outcome depending on the type of reconstruction. At the present time many orthopaedists use

the middle one-third of the patellar tendon for intra-articular reconstruction. There are few studies which have evaluated this procedure in a prospective fashion (29). To evaluate these patients muscle strength testing, subjective assessments and MRI evaluations were obtained at similar time intervals to evaluate the successfulness of ACL reconstruction and to allow comparison between different methods of assessing ACL reconstruction results.

MATERIALS AND METHODS

Study Design

Thirteen patients were entered into the study prior to ACL reconstruction, the first in September 1988, the last in June 1989. Evaluation of all patients was completed by July 1991. All were followed postoperatively for at least one year and most were followed for 24 months.

Criteria for inclusion were (1) willingness to participate for at least one year, (2) willingness to perform preoperative and postoperative tests, (3) willingness to undergo at least three postoperative knee MRI's at no cost (4) failure of conservative management of ACL insufficiency, (5) clinically confirmed ACL tear, (6) no prior ACL reconstruction to either knee, (7) no previous ACL injury to the non-involved knee.

Every patient contacted by the principle investigator was willing to participate in the study. At the first meeting the patient was questioned by the principle investigator (Appendix 2; 34), examined with the ligament testing and muscle testing devices by one of two registered physical therapists (T.P. and C.D.). Table 1 provides preoperative data.

All patients subsequently underwent ACL reconstruction using patellar tendon grafts as described by Clancy (17). At the time of surgery the arthroscopic exam revealed a torn medial meniscus in 8 cases and a torn lateral meniscus in 4 cases. None suffered any peri-operative complications, and all were prescribed analgesics for the postoperative pain. One patient developed a stitch abscess which was treated successfully with antibiotics. After reconstruction patients were followed not only in the typical manner by their orthopaedic surgeon but also by magnetic

resonance imaging, evaluation form completing, muscle testing and objective ligament testing. All followed the same rehabilitation protocol (Appendix 12). The schedule of events is outlined in table 2.

Patient Population

Table 1 provides a demographic summary of the study population. All but one of the thirteen knee injuries occurred while patients were participating in athletic activities. The remaining injury occurred during a work-related activity. The mean age was 21.5 years (S.D.=5.0; range=16-28 years). Six males and seven females participated in the study. The right knee was injured in six cases (3 females, 3 males), the left knee in seven (4 females, 3 males). The mean time from injury to operation was 462.6 days (S.D.=434.6; range=96-1153 days). All patients recalled the initial injury. Ten (77%) remembered experiencing subsequent similar knee injuries after the initial insult. Six (46%) recalled hearing a pop at the time of the initial injury. Twelve (92%) had MRI's documenting an ACL tear prior to surgery. The remaining patient had an arthrogram prior to undergoing ACL reconstruction. One (8%) patient had undergone knee surgery for a partial medial meniscectomy prior to entering the study. All ACL reconstructions were performed by two arthroscopic orthopaedic surgeons (K.L. and P.J.).

Subjective evaluation

Patients completed evaluation forms (Appendix 2; 34) pre-operatively, six, twelve and twenty-four months postoperatively. The responses were followed and analyzed over time using multiple regression analysis. Responses to these questionnaires were also compared to other

test results to determine if a correlation exists between either subjective evaluations and objective testing or subjective evaluations and MRI findings.

Surgical Technique

A bone-tendon-bone graft was harvested from the middle one-third of the patellar tendon as described by Clancy (17). Appendix 1 demonstrates how the graft was excised from the patellar tendon and used to reconstruct the anterior cruciate ligament.

Instrumented Laxity Testing

Before the study began, Faro (Genucom's manufacturer) certified the two physical therapists operating the three dimensional ligament testing device. This testing was performed by the same physical therapist preoperation and six and twelve months postoperation. During the study the physical therapists participated in an inter-user study to validate the device's reproducibility (121). The physical therapists operated the testing device as prescribed in Faro's Genucom manual (Appendix 3). The test battery included the AP drawer, AP drawer with internal rotation, AP drawer with external rotation, dual AP drawer, Lachman (AP drawer at 30 degrees), dual Lachman, varus stress at 10 degrees, valgus stress at 10 degrees, internal stress, external stress, medial pivot shift, and genu recurvatum/screw home test. These tests were performed to compare objectively the laxity of the involved knee to the non-involved knee before and after ACL reconstruction. The Genucom results were analyzed with repeated measure analysis of variance (ANOVA).

Two years after the study began, evidence demonstrated the KT2000 was more accurate than the Genucom (see introdution); thus, the KT2000 was used to assess ligamentous laxity twenty-four months after reconstruction. AP drawer at 20 degrees was performed using the KT2000 as prescribed in the manual. Twenty-four months postoperation ligamentous laxity testing was obtained using the KT2000 and physical exams. Additionally, manual pivot shift and Lachman exams were performed by the orthopaedist twenty-four months after reconstruction.

Ligamentous Laxity Analysis

Due to intertest variability (121)(125), absolute values were not compared over time. Instead, the difference in tibiofemoral displacement (measured in mm) between the non-involved and involved knees was calculated for each interval. This difference, using the non-involved leg as the control, was assessed to determine if a significant change in the patient's knee laxity occurred after ACL reconstruction. Knees were considered to be unstable if the relative displacement according to the instrumented Lachman was greater than five millimeters (112). A grading system (0-4) was employed to evaluate significant manual Lachman and pivot shift exams. The laxity data were analyzed with analysis of variance (ANOVA) and compared to MR and clinical findings.

KINCOM

The KINCOM testing was performed preoperatively and six and twelve months postoperatively. Testing included concentric and eccentric quadriceps and hamstrings strength testing at 60°/sec, 90°/sec and 120°/sec. Strength testing of involved and non-involved legs was

performed to evaluate absolute strength as well as quadriceps to hamstrings strength ratios preoperatively and postoperatively. Repeated measures analysis of variance and paired T-tests were used to evaluate changes in strength and differences between legs.

MRI

Imaging (see Appendix 5)

Following informed consent and safety screening (Appendix 4), magnetic resonance (MR) scans were obtained using a General Electric (Signa) superconducting whole body system operating at 1.5 Tesla linear extremity coil (transmit/receive) with 17 cm diameter bore. The imaging protocol consisted of spin echo (SE) imaging techniques in the axial, sagittal, and coronal planes. The knee was imaged in 0-15 degrees of external rotation in the extended position. Imaging parameters included axial, coronal, and sagittal T2 weighted images, as well as sagittal T1 weighted images. This set of 4 pulse sequences was obtained at 41.2 +/- 12 days (mean +/- 1 S.D.), 195 +/- 29 days, 371 +/- 25 days and 738 +/- 72 days following the surgical procedure. In addition, MR scans were obtained on average 159.4 days (S.D.=86.9; range=46-322) before surgery in 12/13 patients.

Evaluation of MR Scans

These scans were interpreted by an MR radiologist (CP) with attention directed to assessing the appearance of the ACL graft, menisci, hyaline cartilage, bone, and patellar tendon. A quantitative evaluation was made of the appearance of the ACL graft with respect to its signal intensity and dimensions. Qualitative and quantiative evaluations were made of the

patellar tendon. Appendix 6 is an example of the MR interpretation form. Signal intensity and appearance of the different areas of the graft on proton density weighted images were graded according to a comparative scale to assess temporal change from 1.5 to twenty-four months. To grade the overall appearance of the grafts, a scale from 0-4 was used (Appendix 7). A grade one graft appeared similar to normal tendon; whereas, a higher grade was assigned to grafts with an increased signal intensity relative to normal tendon. The MR findings were compared to instrumented and subjective evaluations.

A qualitative evaluation was made of the patellar tendon's appearance. The degree of patella/femoral cartilage overlap was measured to assess changes in the patellar tendon length. The form in Appendix 6 is an example of the MR interpretation reporting sheet. All measurements were converted to mm for statistical analysis including repeated measures analysis of variance (ANOVA). All MR scan evaluations were made without knowledge of the clinical or objective assessments.

Investigators' Responsiblities

Medical student:

1.) Scheduled patients for exams, including MRI, Genucom, and Kincom

2.) Gathered and analyzed all data excluding the detailed statistical analysis

3.) Recorded clinical questionnaire data

4.) Monitored patients on MR scanner

5.) Recorded all MR data/descriptions

6.) Interpreted all information into numerical data for
     statistical analyses

7.) Performed literature searches

Physical therapists

1.) Performed Genucom and KINCOM testing

Radiologist

1.) Interpreted MR images

Orthopaedists (in addition to responsibilities as primary physician)

1.) Performed the ACL reconstruction

2.) Performed clinical exams twenty-four months after
     reconstruction

Statistician

1.) Performed multiple regression analysis and t-tests

Photographer

1.) Photography of MRI's and other illustrations

Statistics

See Appendix 8 for description of statistical analysis.

RESULTS

Subjective Evaluations

Table 3 includes mean responses to the questionnaire (Figure 2). The following parameters improved with statistical significance in the postoperative period: pain, giving-way, ability to cut and turn, jump, and run (p<0.05). The following parameters did not change with statistical significance in the postoperative period: swelling, ascending/descending stairs, and walking (p>0.05). The patients in this study had very few problems with swelling, ascending/descending stairs and walking prior to undergoing reconstruction; therefore, these parameters could not improve significantly. The ability to perform sports improved significantly over time (p<0.05). Before ACL reconstruction and six months postoperation, most patients were significantly limited in ability to participate in sports as compared to the pre-injury status. By twenty-four months after surgery, all patients had progressively improved from the preoperative state as measured by ability to return to sports (p=0.0001). Twenty-four months after reconstruction, 7 (64%) patients were performing as well at the same sports, 3 (27%) were performing the same sports as prior to injury but at a slightly decreased level, and 1 (9%) was active, but performing different sports from prior to injury.

Clinical correlates between the parameters measured with this questionnaire and objective testing and MRI findings were evaluated. Patients' reports of swelling and the degree of effusion seen on MR had a high correlation (P=0.001). No other clinical symptoms and objective findings appeared to correlate over time.

Instrumented ligament testing

Ligament testing device data is presented to assess ligamentous laxity as measured by tibiofemoral displacement resulting from an applied force. The results are reported as the difference between noninvolved and involved mm displacement preoperatively, 1.5, six, twelve, and twenty-four months postoperatively.

Three of the Genucom tests demonstrated changes in the knee's laxity (table 5). The three tests which revealed a significant change over time were similar in that the average displacement (laxity) was greater in the involved leg than the non-involved leg preoperatively and the average displacement was less in the involved leg than the non-involved leg 6 months postoperatively. In other words, as measured by the Genucom the involved leg was even tighter than the non-involved leg six months postoperation (Appendix 9). This relationship remained about the same according to two tests, anterior-posterior drawer at 30 degrees and medial compartment subluxation test with anterior rotation. The dual anterior-posterior drawer at 30 degrees changed significantly between six and twelve months in that the involved and non-involved legs became more equal in laxity. The overall change in these relationships is best demonstrated in graphs in Appendix 9.

As quantified by these tests the involved knee was tighter than the non-involved knee six months postoperation. According to two of these tests, anterior-posterior drawer at 30 degrees (anterior) and the medial compartment subluxation (anterior rotation), little relative change occurred in the involved knee as compared to the non-involved knee after 6 months postoperation. According to the dual anterior-posterior drawer at 30

degrees (lateral-anterior) test the involved knee was tighter in the anterolateral direction than the non-involved knee at 6 months postoperation and almost equal in laxity in the anterolateral direction at twelve months postoperation.

The AP drawer at 30 degrees was used to determine if patients had significant ligamentous laxity at six and twelve months after reconstruction. A positive exam was defined as a knee which could be displaced more than 5 mm relative to the non-involved knee (table 4). According to the AP drawer at 30 degrees (Lachman) one patient had a positive exam ($\geq$5mm) at six months, but not twelve months. According to the AP drawer at thirty degrees, no patients had positive exams ($\geq$5mm) at twelve months. One (8%) patient at six months and none at twelve months were considered to have instability as determined by the instrumented laxity results.

Twenty-four months after reconstruction the KT2000 and physical exams identified one (10%) patient who developed ligamentous laxity (see table 6).

KINCOM

The KINCOM data was used to assess quadriceps strength, hamstring strength, and the ratio of these muscle groups strength before and after ACL reconstruction. The results are reported in tables 7-19. One patient missed the twelve month KINCOM testing.

Quadriceps The involved quadriceps' concentric force productions progressively increased at all speeds after ACL reconstruction. At the intermediate and fast speeds the concentric force produced by the quadriceps increased to a level of statistical significance (p<0.05). By

twelve months postoperation the involved quadriceps strength remained significantly less than the non-involved quadriceps ($p<0.05$) (Table 19). The change in the relationship between the non-involved and involved legs as measured as a ratio of the two did not change significantly over time ($p>0.05$).

Hamstrings Comparing the involved to the non-involved hamstrings, there were no statistically significant differences or changes as measured by concentric and eccentric force production ($p>0.05$) either preoperatively or postoperatively. The strength of the involved hamstrings decreased on average eccentrically and concentrically over time, but this change was not statistically significant. Twelve months post ACL reconstruction the hamstrings of the involved leg were almost equal in strength to the non-involved (Table 20).

Quadriceps/Hamstrings The quadriceps to hamstring strength ratio was calculated at each time interval. None of the ratios for the involved leg changed with statistical significance at any speed concentrically or eccentrically ($p>0.05$).

Magnetic Resonance Imaging

Magnetic resonance imaging data were compiled using the protocol outlined above. One patient missed the six month MRI appointment another the twelve month appointment. Four patients missed the twenty-four month MRI appointments. Results were recorded on the form in Appendix 6 and analyzed to assess changes over time.

ACL Grafts Table 22 demonstrates serial MRI analysis of thirteen patients' grafts following ACL reconstruction. The grafts were identified and noted to be anatomically intact. Each graft was described in terms of overall graft appearance and intrinsic signal intensity. The significant finding in these serial images is that the grafts showed the greatest signal change between 1.5 and six months postoperatively. Most of the grafts looked most like normal tendon at 1.5 months and then showed an increase in signal intensity compared to normal low signal tendon by six months. There was very little change in the grafts between six and twenty-four months. Appendix 13 demonstrates the changes in MR appearance. These images are representative examples of patients in this study. The greatest change is seen between 1.5 and six months; little change is apparent in the overall MR appearance of the grafts between six and twenty-four months.

Patellar tendon Table 21 includes the numeric data for the dimension assessment of the patellar tendons. The patellar tendon size increased significantly at all postoperative time points. The anteroposterior dimension of the inferior, middle, and superior patellar tendon increased significantly 1.5, 6 and 12 months after reconstruction. This dimension increased from a mean of 6 millimeters preoperatively to about 10 mm 1.5, 6 and 12 months after reconstruction. The degree of patella/femoral cartilage overlap, which is an assessment of patellar tendon length, increased from about 11 mm preoperation to 16 mm 1.5 and 6 months after reconstruction, but twelve months after surgery the degree of overlap, about 13 mm was not significantly different from the preoperative state. The diffuse tendon abnormalities persisted on MR images even one year post-reconstruction; however, these abnormalities decreased significantly during the first year after reconstruction. The diffuse tendon

abnormalities of the healing tendon have a similar appearance to tendinitis. In one patient a small stitch abscess was identified on the basis of a focal area of increased signal intensity involving the subcutaneous tissue and the patellar tendon.

Effusions The effusions were also analyzed and recorded in this form. The effusions decreased significantly over time. (P=0.0001)

Menisci   Table 24 summarizes serial MRI analysis of thirteen patients' meniscal tears before and after ACL reconstruction. In this study 26 menisci were analyzed preoperatively and postoperatively. Of these 26 menisci there were 9/13 medial meniscus tears and 5/13 lateral meniscus tears. No surgery was performed on one medial meniscal tear, since it was a residual tear from a previous partial medial meniscectomy and was considered stable. Three of the menisci were repaired. Twelve months after reconstruction, one (33%) of these repaired menisci showed a decrease in the signal abnormality in comparison to the initial postoperative MRI evaluation; one (33%) remained without change and one (33%) had increased defects. There were six meniscal tears not seen at surgery either arthroscopically or with preoperative MRI's but were seen by MR 1.5 months after ACL reconstruction. These tears were not treated surgically. Twelve months after ACL reconstruction, five of these tears showed evidence of decrease, one tear showed evidence of increase and three tears had resolved completely as assessed on the magnetic resonance images.

DISCUSSION

This prospective study evaluated patients before and after ACL reconstruction to assess patient's functional and clinical status subsequent to ACL reconstruction. The clinical symptoms evaluated pre-operatively and postoperatively reflected the improvement in the patients' knee function and were consistent with the expected results. The following parameters improved with statistical significance in the post-operative period: pain, giving-way, ability to turn and cut, jump and run. The parameters of swelling, ability to ascend and descend stairs, and walk did not improve significantly since these patients like most chronically ACL deficient patients had little or no difficulty with swelling or these activities prior to surgery. Since the ACL's role is to provide the knee with stability especially during demanding activities, these findings are those which were expected.

Since one of the primary goals of ACL reconstruction is to enable patients to return to the pre-injury activity level, the ability to return to sports was examined closely. Twenty-four months after surgery all patients had progressively improved from the pre-operative state as measured by ability to return to sports (p=0.0001). Twenty-four months after reconstruction, 7 (64%) patients were performing as well at the same sports, 3 (27%) were performing the same sports as prior to injury but at a slightly decreased level, and 1 (9%) was active, but performing sports different from prior to injury.

Overall, the patients improved as expected and some patients responded to intervention even better than had been anticipated. These results are similar to those reported in other studies such as Tibone and

Antich's two year ACL reconstruction study which reported 55% patients returned to their previous level of sports participation and 36% patients returned to limited participation and one patient (9%) was unable to return to athletic participation at all (57). In contrast, Shields et al. evaluated patients with ACL deficient knees after meniscectomy only. These authors reported fewer patients (80%) were able to return to 75% or greater pre-injury level sports activities in comparison to 91% patients in the present study. This difference may be a function of Shields' study follow-up range (2 to 9 years) and the necessity of ACL reconstruction rather than meniscectomy alone (106).

The patients in the present study experienced an increased ability to perform daily activities as their ability to return to sports improved. Seto et al. examined patients' sports participation after ACL reconstruction. Seto also found a positive correlation (p<0.001) between functional activity and sports participation. Functional activity was measured using a clinical data collection form quantifying symptoms associated with performing daily activities. This finding raises the possibility that the ability to function in daily activities may be a reliable predictor of successful return to sports activities. In the clinical setting orthopaedists often prefer the patient to be asymptomatic performing daily activities prior to their returning to sports. Seto's results and the present study's results indicate that using patients' ability to perform functional activities to monitor patients' progress is reasonable. The ability to participate in sports was also compared to objective findings, which is discussed in the subsequent sections.

A number of instrumented devices have been used to measure knee stability. The literature on the Genucom knee analysis system prior to commencing the study in 1988 was sparse (96)(60), since the machine was

not commercially available until 1986. A search of the Genucom literature in 1988 revealed articles by Oliver and Coughlin and Schmitt, Wroble, Grood, and Noyes agreeing that this computer-based instrument could potentially provide a significant contribution to objective measurement of knee joint stability. However, more studies were required to establish its role in orthopaedics and rehabilitation management. In 1989, literature criticizing the Genucom for its lack of reproducibility became available. In 1989, Highenboten and Jackson compared the reproducibility of the anterior and posterior knee laxity values for the Genucom, KT1000 arthrometer and the Stryker knee laxity tester. They found that the Genucom produces anterior laxity values greater than either the KT1000 or the Stryker. The Genucom was also found to produce larger standard deviations for knee laxity measurements, denoting a higher degree of inter-individual variability. However, intraclass correlations indicated that all three devices demonstrated excellent reproducibility for anterior and posterior laxity values across the three trials within a session. In 1989, Anderson and Lipscomb also compared the KT1000, the Stryker knee laxity tester and the Genucom. These authors found that the Genucom was the most versatile of the devices, but unlike Highenboten's findings, laxity recorded in patients with a torn ACL was significantly lower than the other devices. Anderson et al. concluded that Genucom generated measurements can be used to determine the success of an operation in restoring stability (3). These authors argued that the major advantage of the Genucom is that it is versatile and can simultaneously document flexion-extension, anterior-posterior displacement, varus/valgus displacement, and rotation. In 1989, Wroble, Grood, Noyes and Schmitt reported again on the reproducibility of the Genucom knee analysis system (125). These authors commented on

the 1987 data and reported additional findings. Their results indicate that measurements vary from day-to-day; therefore, care must be taken in interpreting the meaning of a single measurement or even of repeated measurements made within a single seating. Wroble et al. found significant differences between their two examiners, but there were no significant intraexaminer test-to-test (within seating) effects (125). In 1990, Steiner et al. compared the Genucom device with the Acufex, Medmetric, and Stryker devices. These authors found that the Genucom generated the least reproducible measurements and tended to register the greatest differences in displacement between the right and left knees (40). In 1990, Marc Warman reported in his Yale Medical School thesis the Genucom has significant ($p<0.05$) day-to-day variability (121). As a result of others' experience, the KT2000 rather than the Genucom was used to measure ligamentous laxity twenty-four months after ACL reconstruction.

To avoid interexaminer variabilty, one of two certified physical therapists was assigned to each patient; therefore, all Genucom testing was performed by the same physical therapist at all intervals for a particular patient. To minimize interseating variability, laxity was calculated for each test as the difference between the involved knee's displacement and the noninvolved knee's displacement. Despite the reported flaws in the Genucom system, three tests did detect significant changes in the knee's laxity after ACL reconstruction. The changes in the involved knee's laxity detected by the anterior-posterior drawer at 30 degrees (anterior), dual anterior-posterior drawer at 30 degrees (lateral-anterior) and the medial compartment subluxation (anterior rotation) tests were those which were expected. Other tests, such as the anterior-posterior drawer test at 90 degrees, also theoretically examine the ACL; however, they did not detect

any significant changes over time. As quantified by these tests, the involved knee was tighter than the non-involved knee by six months post-operation. According to two of these tests, anterior-posterior drawer at 30 degrees (anterior) and the medial compartment subluxation (anterior rotation), little relative change occurred in the involved knee as compared to the non-involved knee six months postoperation. According to the dual anterior-posterior drawer at 30 degrees (lateral-anterior) test the involved knee was tighter in the anterolateral direction than the non-involved knee six months postoperation and almost equal in laxity in the anterolateral direction twelve months postoperation.

The lack of significant findings in the other tests does not suggest that no change occurred in the involved knee rather that the Genucom may have intrinsic limitations. The three tests which detected significant changes in the knee's laxity are described below to allow a better understanding of what each test evaluates. Medial compartment subluxation test with measurement of anterior translation of the tibial plateau with rotation above and beyond that due to the straight Lachman test is evaluated by the application first of an anterior force followed by an external rotation and posterior force followed by an internal rotation while looking at the medial tibial plateau. This test theoretically evaluates the ACL, medial meniscus, medial collateral ligaments, PCL, medial one-third of the lateral capsule, iliotibial band and the lateral meniscus. The anterior-posterior drawer test at 30 degrees of flexion is an application of pure (anterior) force (at least 21 pounds) in the vicinity of the proximal tibia and then evaluating the resulting sliding of the tibia with respect to the femur. The examination is performed at 30 degrees to minimize the agonistic effect of the hamstring. This test has been used in the past

primarily for cruciate ligament injury but is also thought to evaluate the medial meniscus and the lateral capsule. The dual anterior-posterior drawer test is used to evaluate the difference in anterior and posterior displacement of the centers of the medial and lateral tibial plateaus. In the past, researchers suggested that excessive displacement of a particular tibial plateau is indicative of rotatory instability.

Interestingly, the Genucom detected the decreased laxity of the involved leg. In fact, the Genucom data imply that the surgery overtightened the injured knee, since the involved knee was tighter than the noninvolved knee. These significant changes detected by the Genucom require further investigation. It is reasonable that no significant change was detected in anterior displacement by the anterior drawer at 90 degrees of knee flexion but was detected at 30 degrees of knee flexion. The anterior cruciate ligament is the primary restraint at both 90 and 30 degrees of knee flexion; however, the secondary stabilizers are thought to play a greater role in restraining anterior displacement at 90 degrees than 30 degrees. These secondary stabilizers include the anteromedial meniscus, medial and lateral collateral ligaments, medial and lateral capsules and the iliotibial band. Noyes' findings are similar to the present study's (92). Noyes' prospective study used the KT1000 anterior drawer to measure laxity of knees 4 weeks and 3 months after ACL reconstruction (92). These authors also reported an increased tightness in the involved leg relative to noninvolved leg after reconstruction. They also described a normalization of this tightness relative to the noninvolved leg over time (92).

Twenty-four months after reconstruction, patients were tested with the KT2000. Relative displacement greater than 5 mm was considered

significant. One (10%) patient had a positive exam 24 months after reconstruction. Interestingly, this same patient had a positive instrumented Lachman exam six months but not twelve months after reconstruction. Two years following reconstruction, the same patient had positive clinical Lachman and pivot shift exams. There was a 100% correlation between the orthopaedists' clinical exams and the objective testing at twenty-four months after reconstruction. This patient's negative instrumented Lachman exam twelve months after reconstruction was most likely a false negative resulting from the intrinsic flaws of the Genucom or muscle tightening by the patient. This patient with $\geq$ 5mm relative displacement was symptomatic with knee pain and swelling. This same patient developed episodes of giving way and difficulty with running. Twenty-four months after reconstruction, this patient with ligamentous laxity had not returned to sports at the same level as prior to injury; in contrast to the majority (64%) of patients in this study who did return to sports at the same level as prior to injury. However, the three other patients in this study who also did not return to sports at the same level as prior to injury did not have significant ligamentous laxity.

Johnson's retrospective study reported that most ACL reconstructed knees were about 2.1 to 4.0 mm more displaceable than the noninvolved knee (67). In the majority of patients, primary laxity more than 1 mm greater than the noninvolved leg could be discerned in the reconstructed knee (anterior drawer test, 65.9%; Lachman test, 72.7%). Johnson's group followed-up patients 5 to 10 years after reconstruction which may not be comparable to laxity two years after ACL reconstruction since the ligament may stretch out over time (39). Ferkel et al followed 100 patients after reconstruction using a torn meniscus. The majority of patients had

trace or 1+ Lachman tests postoperatively (39). It is possible that many patients have an increased tightness in the involved knee relative to the noninvolved knee for a few months after ACL reconstruction; however, most studies do not evaluate the laxity of knees before one year after reconstruction.

Engebretsen et al. recently published a prospective study assessing 50 patients after ACL reconstruction using a patellar tendon graft (29). At one year after reconstruction 67% had less than 3 mm displacement greater than the noninvolved leg and 33% had 3-5 mm displacement greater than noninvolved leg. Some of these patients with <3mm displacement may also have had a "tighter" reconstructed knee than the non-operated knee one year after reconstruction; however, this information was not available in the publication (73). Engebretsen's KT1000 results are similar to the present study's Genucom results in that the involved knee was significantly less lax at one year after reconstruction than prior to reconstruction. Engrebretsen's two year results are similar to the present study's KT2000 results in that the majority of reconstructed knees could be displaced less than three millimeters relative to the involved knee.

As seen by previous authors (105)(52)(29) and of some interest, is that these changes in the objective assessment of knee stability did not correlate with the patients' ability to return to sports. No comparison of these results with any other Genucom analysis of ACL reconstructed knees could be done since no known study has examined ACL reconstruction results using the Genucom. Although no correlation with functional ability could be identified, no patient's graft ruptured. The role of such tests might be to detect failed grafts or failing grafts, for example loosening in the femoral tunnel. In order to maximize objective measurements to

identify failing grafts, expected degree of difference between involved and noninvolved legs following ACL reconstruction needs to be generated. A study including more patients with longer follow-up may help discern ligamentous laxity testing's relationship with clinical outcome.

Others have reported that the clinical Lachman test in the hands of an experienced orthopaedist may provide more accurate knee laxity data than objective laxity testing (3). Anderson's study also suggests that the clinical examination by an experienced examiner is the most accurate way to determine cruciate ligament integrity (3). In the present study a high degree of correlation between the clinical Lachman and the KT2000 tests was found. No comparison between the Genucom and clinical Lachman could be made since these tests were not performed at similar intervals.

Harter and others have found that the KT1000 and the clinical examination do not correlate with the patient's perception of knee stability and function (58). One example of the KT1000 measurements correlating with a patient's clinical state was reported in Noyes' prospective study (92). In Noyes' prospective study the authors used KT1000 measurements to evaluate effects of early knee motion after ACL reconstruction, the authors identified one patient in which the laxity of the involved knee compared to the noninvolved knee was 5 mm. The patient refused to comply with the advice not to kick from the knee during vigorous swimming activities. At the time of KT1000 testing the patient was warned that the kicking may prevent the ligament from healing normally. Subsequently, the patient resumed the protective postoperative regimen with no additional increase in laxity (92). Similar to the present study, Johnson's group found an association between one patient's objective laxity and clinical status (67).

In the present study, objective evaluation of the relative strength of the quadriceps and hamstrings was performed utilizing the Kinetics Communicator (KINCOM). In 1984, Farrell and Richards evaluated the accuracy and reproducibilty of the KINCOM; these authors concluded that KINCOM produces valid and reliable measurements (31). Similar results were later reported by Harding (57). Previous studies have used the Cybex II unit, an older and more well known isokinetic device, to evaluate quadriceps and hamstring strength. The KINCOM is capable of interpreting eccentric force production, the Cybex II unit is not capable of analyzing this type of data. Concentric work best describes exercise involving the shortening of actively contracting muscles. For example, lifting an object results in the development of tension in the active muscles and a subsequent decrease in muscle length. In contrast, the process of lengthening actively contracting muscles is termed eccentric contraction. In eccentric work, the tension generated by the muscles is overcome and the muscles are forcibly stretched resulting in lengthening of the muscle unit. As a result, relatively few fibers are recruited and relatively large forces are produced by eccentric contractions. Previous research has demonstrated that greater tension per muscle fiber is generated under eccentric contraction conditions when compared to concentric conditions. Research has not assessed patients following ACL reconstruction; therefore, the role of eccentric force measurements in evaluating the success of ACL reconstruction is not known.

All patients were tested at three different speeds 60 °/second (slow), 90°/second (intermediate) and 120 °/second (fast). Eccentric and concentric force productions were measured at each of these speeds pre-operatively and postoperatively. The involved quadriceps concentric force

productions increased at all speeds after ACL reconstruction. At the intermediate and fast speeds the concentric force produced by the quadriceps increased to a level of statistical significance ($p<0.05$). The involved and noninvolved quadriceps eccentric force productions increased during the first twelve months after ACL reconstruction; however, these increases were not statistically significant ($P>0.05$). The eccentric force production of the involved quadriceps was consistently greater than the concentric force production at all speeds. The non-involved quadriceps also produced a greater force eccentrically than concentrically. These findings are consistent with those expected.

Since one of the goals of rehabilitation after ACL reconstruction is to make the involved quadriceps as strong as the noninvolved quadriceps by twelve months after ACL reconstruction, the quadriceps of both legs were compared to one another using the paired T-test. At the slow and intermediate speeds, the concentric force production of the involved leg was significantly less than that of the non-involved leg. At all three speeds the eccentric force produced by the involved leg was significantly less than that of the non-involved leg. One year after reconstruction the involved quadriceps strength was significantly less then the noninvolved quadriceps ($P<0.05$).

This study's results provide support to the generally accepted opinion that quadriceps do not regain pre-injury strength after an ACL tear (36). Twelve months post ACL reconstruction the patients in this study had not completely attained equal strength in their quadriceps. The quadriceps' strength of the involved legs did increase and approach the strength of the quadriceps of the non-involved legs. However, a statistically significant

difference between involved and non-involved quadriceps persisted even 12 months post ACL reconstruction.

Lorentzon's findings are similar to the present study's preoperative findings (77). Lorentzon et al. studied eighteen patients who had untreated chronic ACL deficiency by evaluating muscle size, morphology, and isokinetic performance of the quadriceps muscle (77). These patients represent a normal population of patients who have an untreated ACL tear requiring surgical reconstruction. Compared to the non-injured leg, each of the measured parameters of isokinetic performance of the quadriceps muscle were significantly reduced at all velocities of angular motion (p<0.01). Lorentzon et al. concluded that the decrease of isokinetic performance of the quadriceps muscle can best be explained by decreased activation of normally functioning muscle fibers of the quadriceps muscle caused by altered sensory feedback from the mechanoreceptors of the torn ACL (77). LoPresti et al. quantified quadriceps size and function approximately twelve months postsurgery (76). Isokinetic torques for the quadriceps were reduced by 11-15% in the operative leg twelve months postsurgery (p<0.05). LoPresti's findings are consistent with those in the present study and data reported by Arviddson and Eriksson in other studies (8)(30). Harter et al. found a significant quadriceps deficit in the involved leg persisted 24 to 101 months after surgery (73). Seto et al. found a significant quadriceps strength deficit in their subjects' involved legs (105). Noyes' prospective study, using Cybex isokinetic testing preoperatively and one year postoperatively, described a quadriceps deficit at both these testing intervals similar to that which was found in the present study. The quadriceps strength was expected to increase, since this is one of the goals of rehabilitation. The quadriceps strength of the involved leg always

remained less than that of non-involved quadriceps, which is consistent with the expected not desired results. There are numerous factors to consider in understanding why the muscle strength of the quadriceps does not attain strength equality: preoperative muscle atrophy, postoperative immobilization, persistent instability, decreased muscle recruitment (neuromuscular), adequate rehabilitation program and patient compliance. None of these factors can be adequately analyzed in the present study to assess its particular role in influencing quadriceps strength.

It has been postulated that this decreased quadriceps strength could be a function of graft type, specifically patellar tendon graft; however, this theory could not be assessed in the present study. In studies examining quadriceps strength after other types of ACL reconstruction, such as prosthetic ligament reconstruction, patients also had a quadriceps deficit twelve months after ACL reconstruction (123).

Changes in hamstrings strength were also assessed. The involved and non-involved hamstrings did not reveal significant differences or changes as measured by concentric and eccentric force production. Preoperatively, the involved and non-involved hamstring strengths measured by concentric and eccentric force production were not significantly different. This similarity between the involved and non-involved hamstring strength persisted twelve months after ACL reconstruction. Eccentric force production of the hamstrings was slightly greater or almost equal to concentric force production at all speeds for the same leg. Of interest is that the hamstrings of the involved leg were slightly stronger than the non-involved leg at most speeds eccentrically and concentrically. The increased strength of hamstrings of legs with chronic ACL deficiency relative to non-involved legs has been reported in the literature (77). Lorentzon et al.

found a small spontaneous hypertrophy of the hamstrings, which are well-known agonists of the ACL (77). It has been proposed by Solomonow and Giove that increased hamstring strength is beneficial to patients who have a ruptured ACL (109)(49). This increased hamstring strength is thought to be a reflection of the hamstring's agonistic relationship with the ACL. After ACL reconstruction the hamstrings of both legs became more equal. This increased similarity in strength may reflect the ACL's decreased dependence on the hamstring's agonistic effect.

The quadriceps to hamstring ratio was calculated for each test at each time interval. None of the quadriceps to hamstring ratios for the involved leg changed significantly at any speed concentrically or eccentrically (P>0.05). In this present study no statistically significant relationship exists between the quadriceps and hamstring strength and the patients' ability to return to sports; however, both improved over time. A positive correlation between leg muscle strength and ability to return to sports exists, but the relationship is not statistically significant. Harter failed to find a significant correlation between patient's perception of knee function and quadriceps or hamstrings strength. Seto found a significant correlation (P<0.05) between increased quadriceps and hamstring strength on the operated leg and return to functional activities. Overall, the eccentric force productions were expected to demonstrate greater force productions than concentric force productions. Other than producing greater forces than concentric testing, eccentric force productions provided little additional information about patients after ACL reconstruction.

Atrophy is one of the great problems in rehabilitation after a knee ligament injury or surgery. The attitude towards postoperative rehabilitation after ACL surgery has changed from a reliance on a long

immobilization period with isometric muscle training to a more active postoperative program with early mobilization and more dynamic treatment. The rehabilitation protocol described in Appendix 12 was standardized for all patients in the study. The effect of lack of compliance was not measured in the present study. Ten (77%) patients complied with the rehabilitation protocol at the Sports Medicine Center; three (23%) complied with a modified protocol either at home or with a trainer at school. All patients were able to perform the appropriate exercises at six and twelve months postoperation. Although the number of patients is too small to evaluate the effects of rehabilitation location and compliance on the patient's outcome, there was no apparent difference in these two groups with respect to muscle testing. The observation that rehabilitation at home or with a trainer at school may be sufficient for certain motivated individuals is consistent with the findings reported in Phil Stull's Yale Medical School thesis (114). In 1988, Stull reported that a randomly selected group of individuals rehabilitated themselves as quickly and as thoroughly as a randomly selected group training with direct supervision and more sophisticated techniques (114). Stull's study population included patients who had undergone meniscectomies, which is an operation requiring less rehabilitation in terms of mobilization, muscle strengthening, and range of motion than rehabilitation after reconstruction. The present study and Stull's study support the theory that a certain subgroup of motivated patients benefit from home exercise programs. The role of the physical therapist for this subgroup would be to monitor patients at well-defined intervals. Since all the patients in the current study improved clinically and the quadriceps and hamstrings strength became more similar to the noninvolved leg, no conclusions with respect to muscle strength's

predictability and poor clinical outcomes could be made. In the present study, the patients who improved clinically also experienced normalization of quadriceps and hamstring strength using the non-involved leg as the control.

Prior to commencing this study, there were no available studies prospectively assessing patients after patellar tendon ACL reconstruction using MRI. It was hypothesized that MRI might be used to assess objectively a patient's clinical status, diagnose any clinically significant problems and perhaps predict graft failure. The grafts were identified and noted to be anatomically intact. Each graft was described in terms of its overall graft appearance and intrinsic signal intensity. Most of the grafts looked most like normal tendon at 1.5 months and then showed an increase in signal intensity compared to normal low signal tendon by six months. The increased signal can be interpreted as an indication of an increase in the water content of the graft or a decrease in the percentage of tightly bound water component. Very little change occurred in the grafts' appearance between six, twelve and twenty-four months postoperation in the majority of grafts.

The authors suspected that the MR appearance of the graft may correlate with changes in the patient's clinical status and objective findings. No correlation between MR findings and these objective findings was demonstrated two years after reconstruction. Patients all improved dramatically in their ability to return to sports, turn and cut, and run in the twenty-four months post-ACL reconstruction. The signal intensity of the grafts, however, increased rather than decreased, thus appearing less like normal tendon over time. The patients' subjective evaluations and ability to return to sports did not correlate with the changes in graft appearance.

The postoperative changes in the MR appearance of the graft did not appear to be related to changes in muscle strength as measured by the KINCOM or changes in ligamentous laxity as measured by objective testing devices.

There are multiple potential explanations for the changes in the MR appearance. It is possible the changes in MR appearance are related to activity level. The activities of the patients during this postoperative period were examined. Between 1.5 and 6.0 months after ACL reconstruction, patients followed a rehabilitation protocol which instructed patients to increase their range of motion. The patients were advised to increase their range of motion from 0-90 degrees at 1.5 months to full range of motion (about 130 degrees) by six months. This progression may have resulted in an increase in tension in the ACL. When the ACL increases in tension micro and macroscopic damage to the ligament results as reflected by the load-elongation curve described by Noyes (90). Possibly, the changes in the ACL graft appearance can be partially atttributed to damage resulting from an increased range of motion which caused elongation during this post-operative period. This elongation may cause inflammatory changes reflected in the increased signal between 1.5 and 6.0 months post-ACL reconstruction. The full range of motion (ROM) remains constant between six and twenty-four months thus the elongation and tension may remain constant. The constant ROM may partially explain the lack of significant change in the graft's appearance by MR between six and twenty-four months. The actual role of the increased tension and increased range of motion in affecting the graft's appearance on MR requires further investigation.

Howell et al. recently reported findings similar to those in the present study (63). Howell et al. assessed serial MRI's of hamstring ACL autografts during the first year of implantation. These authors also found an increased signal of the ACL graft; however, the increased signal intensity appeared to be regionalized and confined to the distal two-thirds of the intra-articular portion of the graft (63). Howell's findings are similar to this study's in that the increases in MR signal were time-dependent, well established by six months, and unchanged at 1 year. Howell also found that the clinical outcome could not be predicted based on the MR signal of the grafts (63).

Rak found that MR imaging is an excellent noninvasive means of evaluating ACL bone-tendon-bone reconstructions (99). Rak studied patients after reconstruction and correlated the findings with clinical examinations. Rak found a 92% correlation with clinical examination, and a 100% correlation with arthroscopic findings (99). These findings are consistent with the present study's in that the grafts could be identified; however, no significant correlation between MR findings and clinical examinations could be demonstrated in this study. This discrepancy could be due to differences in study population size and patients' clinical outcome. Unlike in this study, there were graft failures in Rak's study.

Kleiner et al. described increasing uniform metabolic and histologic appearances of rabbit patellar tendon grafts with increasing age (72). The lack of change between six and twelve months in the grafts in Kleiner's study is consistent with this study's MR findings (72). Unlike this study's magnetic resonance images, Kleiner's newly formed ligaments had the macroscopic appearance of the normal ACL. (72). Van Rens studied newly formed ligaments using canine iliotibial band grafts at twelve weeks, 16

weeks, six months, twelve months, eighteen months and thirty-six months postoperatively. Van Rens et al. found there are no major differences in findings after sixteen weeks just as no significant MR appearance changes were found after six months in this study (119).

Van Rens et al. also demonstrated microscopic changes in the neoligament. Compared to the normal ACL, the graft's collagen fibers have a more coarse undulant appearance, are more or less in a parallel arrangement and there is more cellular and hypertrophic synovial lining intra-articularly. Vans Rens found little macroscopic difference between normal ACL's and ACL substitutes (119). The changes in collagen fiber apppearance and arrangement may be partially responsible for the MR appearance of the grafts.

Graft revascularization studies on ACL reconstruction also imply that fewer changes in the MR images would be expected after six months. Arnoczky found that the intrinsic vascular response of the canine patellar tendon graft seemed to subside and the vasculature of the infrapatellar pad and posterior soft tissues of the joint appeared normal at twenty-six weeks (89). Histologically, the cellular response as well as the vascularity of the graft appeared less proliferative. Arnoczky's study ended at twenty-six weeks; therefore, twenty-four month comparisons are not possible. Arnoczky's revascularization findings may partially explain the lack of significant change in the appearance of the grafts after six months.

Another possible explanation for the appearance of this study's grafts could be impingement. Impingement may cause inflammation which results in edema. The effect of impingement could not be evaluated since this diagnosis is made on conventional radiographs which were not available in all cases. Howell et al. found that intercondylar roof

impingement is associated with an increased signal intensity in the distal two-thirds of the ACL graft (63). The actual role of impingement requires further investigation.

Moeser et al. reported that the ACL graft was poorly visualized by MR (85). These authors hypothesized that the neoligament was poorly visualized because greater amounts of low signal fibrous tissue were adjacent to the low signal repair which would result in absence of MR contrast (85). They predicted that MRI would be of limited value in assessing grafts after ACL reconstruction. Unlike those in the present study, the MR evaluations in Moeser's study were performed at varied time intervals with various MRI techniques rather than at defined intervals with high resolution technique. Unlike Moeser's study, the neoligament was easily visualized at 1.5, six, twelve and twenty-four months postoperatively. The disparity in the results between Moeser's study and the present study may be partially explained by differences in technique.

J.F. Meyers et al. arthroscopically evaluated ACL reconstructed human knees (84). These authors found poor-quality ligamentous tissue, adhesions, scarring, and significant cartilage lesions. Meyers et al. also found that postoperative findings and appearance of the graft did not correlate with patients' symptoms and function (84). Meyer's findings are consistent with the MR and clinical findings of this study in that the grafts did not appear like normal tendon but the patients were clinically without symptoms and were performing activities at a level close to pre-injury status.

The present study shows conclusively that the ACL graft can be easily identified and the intrinsic signal characteristics assessed. The neoligament was easily visualized and assessed 1.5, six, twelve and twenty-

four months postoperatively. In the present study the intrinsic signal intensity and overall graft appearance did not correlate with other assessed functional clinical parameters and objective measures of stability. Further investigation is required to determine the factors influencing the MR appearance of the grafts. The sample size in this study is small; therefore, detection of relationships between MR image findings and other parameters may require a larger study population.

Since there is little data available regarding the changes in vascularity, macroscopic, microscopic, and MR appearance, and metabolism of human ACL grafts, a number of theories need to be considered to explain the changes in MR appearance of the grafts. The appearance of the graft by MR may be a reflection of the patient's activity level and exercise intensity as well as the graft's revascularization pattern and neoligament formation. Other possible causes of the increase in MR signal intensity over time include impingement and inflammation. These theories require further investigation to define their roles in determining the MR appearance of the grafts after ACL reconstruction. It is conceivable that all of these factors influence the MR appearance of the graft.

Repeating studies similar to Arnoczky's, van Rens' and Kleiner's which also include MR evaluations may provide explanations for the MR appearance. Clinical studies with longer follow-up, radiographs, clinical exams, MRI's and subjective evaluations are necessary to assess the value of MRI in predicting clinical outcome after two years. The MR appearance of grafts one year after reconstruction may help predict the clinical outcome at two years after reconstruction. MRI is an excellent tool to assess an intact ACL graft.

MRI evaluations demonstrated persistent abnormalities at the harvest site even twelve months after reconstruction. These diffuse abnormalities at the harvest site should not be misinterpreted as an infected or partially torn tendon. One needs to consider the patient's clinical status when interpreting the MR scans. The appearance of the tendon was not predictive of changes in muscle strength or the patients' clinical status.

The anterior-posterior dimension did increase significantly after surgery. This increase may reflect the amount of collagen and ground substance laid down. Overall, the magnetic resonance images of the patellar tendons reflected persistent thickening of the donor sites, changes which correlate with traditionally accepted phases of ligament healing. Future studies will be needed to corroborate these findings.

MR imaging of the patellar tendon allowed the diagnosis of a stitch abscess at the harvest site in one patient whose clinical state was unknown to the radiologist. A focal region of abnormality in the subcutaneous tissue and patellar tendon was detected which correlated exactly with a stitch abscess at the donor site. Complications such as abscesses may be identified, but caution needs to be exercised in view of the similarity to normally healing tendon. The focal nature of the abnormality is an important criterion in making this diagnosis.

The quadriceps deficit decreased yet persisted even twelve months after reconstruction. The tendon abnormalities diminished but persisted even twelve months after harvesting the graft. Improvement in the patients' clinical status was reflected in the normalizing MRI appearance of the tendon and KINCOM's assessment of quadriceps strength; however, no obvious correlation was found between these parameters. Since all patients improved sigificantly postoperatively, conclusions regarding the predictive

value of MRI are limited. No comments can be made about patellar tendon rupture and muscle strength since no patient in this study suffered patellar tendon rupture or unsuccessful rehabilitation.

The changes in the patellar tendon on MR images did not appear to have any significant correlation with changes in muscle strength of the involved legs as measured by KINCOM. KINCOM demonstrated persistent quadriceps' deficits and normalization of hamstring strength at twelve months after reconstruction. No association with changes in muscle strength and patellar tendon appearance was appreciated.

Following reconstruction the patellar tendon underwent changes which were detectable by MRI. The harvest site demonstrated persistent abnormalities even twelve months after ACL reconstruction. The healing patellar tendon appeared similar to tendinosis and tendinitis. Abnormalities at the harvest site should not be misinterpreted as an infected or partially torn tendon when the involvement is diffuse. The postoperative changes in the patellar tendon did not appear to be related to changes in muscle strength as measured by the KINCOM.

Others diagnoses such as meniscal tears can also be made by MRI. As expected, the patients in this study had a high incidence of meniscal tears. Investigators have reported meniscal pathology associated with ACL tears for decades. Warren and Marshall reported a 98% incidence of meniscal injury in chronic ACL disruption (122). Woods and Chapman reported 87.7% incidence of meniscal lesions in chronic ACL deficient knees (124) Jackson, Jennings et al. reported tears of the medial meniscus to be approximately three times more common than tears of the lateral meniscus (64) Warren and many others have reported that medial meniscus injury rate (86.9%) is much higher than the lateral meniscus

injury rate (28.9%) (122) The meniscal findings in this study are consistent with those found in other studies.

In this study twenty-six menisci were analyzed pre-operatively and post-operatively. Of these twenty-six menisci there were 9/13 medial meniscus tears and 5/13 lateral meniscus tears. No surgery was performed on one medial meniscal tear, since it was a residual tear from a previous partial medial meniscectomy and was considered stable. Three (33%) of the menisci were repaired. By twelve months after reconstruction one (33%) of these repaired menisci showed a decrease in the signal abnormality in comparison to the initial postoperative MRI evaluation; one (33%) remained without change and one (33%) had an increased defect. There were six meniscal tears not seen at surgery either arthroscopically or with pre-operative MRI's but were seen by MR at 1.5 months after ACL reconstruction. These tears were not surgically repaired. By twelve months after ACL reconstruction, five (84%) of these tears showed evidence of decreased defect (two tears had resolved completely), none (0%) showed no change, and one (16.7%) showed evidence of an increased defect as assessed on the magnetic resonance images. The changes in the repaired menisci are consistent with findings reported by Pope, who followed patients with MR scans after meniscal repair and found 60% demonstrated decreased defects, 20% demonstrated an increased defect and 20% demonstrated no change twelve to eighteen months after repair (45).

This study provides evidence supporting the hypothesis that some stable meniscal tears may undergo spontaneous repair. DeHaven reported seventeen cases of meniscal lesions which had healed despite no attempts to achieve healing (22). "The majority of meniscus lesions that can appropriately be left alone are incidental findings in knees with more

extensive pathology, such as ACL tears"(22). Clinical studies have supported this concept of meniscal healing and repair in the vascular zone, showing excellent results (refer to Figure 29 and 30). Arnoczky, Warren and Spivak found that meniscal lesions which connect with the peripheral vascular network demonstrate a capacity to heal through proliferation of vascular tissue (6)(7).

It appears that MR can play a very significant role in evaluating and assessing a patient's postoperative status. Although no test will ever replace a patient's actual functional status, it is hoped that MRI will be used in the future as a more objective evaluator and predictor of outcome. If MR continues to be used as a postoperative monitoring device, it is hoped that many postoperative complications can be minimized by earlier intervention, such as altering rehabilitation. As more is learned about the appearance of reconstructed knees on MR examinations, the more useful MRI will become in managing the postoperative patient. Information that was only indirectly available from physical exams or more directly but invasively obtained by arthroscopy may be more easily acquired by MR images. The small population size and duration of follow-up limit this study's conclusions. Despite these limitations, it is the first known study to describe the MR appearance of the patellar tendon ACL autografts as well as serial clinical assessments during the first two years after reconstruction.

CONCLUSION

Before this study was begun, it was hypothesized that objective, instrumented tests of ACL laxity, MR imaging, and clinical evaluations, would accurately predict ACL reconstruction's success and outcome. It was discovered that the least expensive of these, the manual clinical examination, may be the best means of evaluating patients after reconstruction. Unquestionably, MRI and instrumented testing, such as KINCOM and KT2000 are of significant diagnostic and prognostic value. They are, nevertheless, both expensive and limited in availability. More to the point, their ability to predict outcome remains so restricted that they should only be used in conjunction with a clinical exam performed by an orthopaedist or an experienced physical therapist.

It is apparent, then, that our study does not support the routine use of MRI and instrumented testing to monitor patients after ACL reconstruction. MRI is useful in the postoperative setting, when a patient is symptomatic, because the MRI can help diagnose infection, ACL tears, meniscal tears and other intra-articular pathology. It is equally obvious, however, that our study's limited size and duration together with the absence of graft failure limits the weight of our conclusions. For these reasons, it is recommended that further research involving longer follow-up and larger populations be undertaken.

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Table 1

Patient	Duration Since Initial Injury (Days) at Time of Injury	Heard pop at Time of Injury	Knee/Sex	# of Subsequent Knee Injuries 1=none, 2=1-5, 3=>5	Previous Knee Surgery	Other Procedures at Time of Surgery	Activity at Time of Injury /Mech. of Injury
1	336	no	L/F	2	none	partial lateral meniscectomy	Skiing/Unknown
2	100	yes	R/F	2	none	no surgery	Soccer/Unknown
3	155	no	L/F	2	none	no surgery	Basketball/Various
4	376	no	R/M	2	none	medial meniscus repair	Racquetball/Ext. rotation of knee while foot was planted
5	198	yes	L/M	2	none	no surgery	Basketball/Extension
6	1153	yes	L/M	3	none	partial medial meniscectomy	Basketball/External rotation on planted foot
7	742	no	R/F	3	none	lateral meniscectomy	Gymnastics/Valgus
8	136	no	L/F	1	none	partial medial meniscectomy	Soccer/Collision
9	1463	no	L/F	2	none	partial lateral meniscectomy	Soccer/External rotation
10	391	no	R/F	2	none	partial medial meniscectomy	Soccer/Valgus & tibial ext. rotation & foot planted
11	759	yes	R/M	3	partial medial meniscectomy 11/88	none	Valgus & hyperextension occupational - fell at work
12	774	yes	L/M	2	none	partial medial meniscectomy lateral meniscectomy	Skiing/hyperextended twisted internal rotation
13	96	yes	L/M	1	none	medial meniscus repair lateral/meniscus repair	Basketball/Jumped - feet forward landed with knee laterally rotated
Summary (Patients 1-13) mean=482 days S.D.=434.6 days Range=96 - 1463 days		Summary (Patients 1-13) 6=no 7=yes	Summary (Patients 1-13) 7 female 6 male 7 right knees (4 female, 3 male) 6 left knees (3 female, 3 males)	Summary (Patients 1-13) 2 = no subs. injuries 8 = 1-5 subs. injur. 3 = greater than 5 subsequent injuries	Summary (Patients 1-13) none	Summary (Patients 1-13) 3 medial meniscus repair 5 partial medial meniscectomies 1 residual medial tear - left alone 2 lateral meniscus repair 3 partial lateral meniscectomies	Summary (Patients 1-13) 12 sports related 1 occupational

Table 1 includes the patients' demographic data.

TABLE 2

ACL Reconstruction Study Schedule
of Events

	Pre-operation	Post-operation
	Physician discussed ACL reconstruction with patient; patient chose reconstruction surgery.	1.5 months MRI Patient followed by Surgeon
	A.S. contacted patient to discuss study.	6.0 months Kincom Testing Genucom Testing Evaluation form (Feagin's) MRI
	Pre-op appointment included the following: Discussion of study with patient Completion of consent form Completion of evaluation form	12.0 months Kincom Testing Genucom Testing Evaluation form (Feagin's) MRI
	Genucom and Kincom Testing by C.D. or T.P.	24 months KT2000 testing Evaluation form MRI Clinical examination

TABLE 3

SUBJECTIVE EVALUATIONS	PRE-OP T1 MEAN (S.D.)	6 MONTHS T2 MEAN (S.D.)	12 MONTHS T3 MEAN (S.D.)	24 MONTHS T4 MEAN (S.D.)	P VALUE	Significant Change
PROBLEMS WITH GIVING WAY	2.45(0.5)	3.8 (0.6)	3.55 (1.05)	3.65 (0.8)	0.001	T1<T2 T1<T3 T1<T4
PROBLEMS WITH STAIRS	3.8 (0.4)	3.5 (0.9)	3.8 (0.6)	3.9 (0.3)	0.50	NONE
PROBLEMS WITH KNEE PAIN	2.55 (0.69)	3.36 (0.81)	3.36(0.92)	3.46 (0.69)	0.02	T1<T2 T1<T3 T1<T4
PROBLEMS WITH SWELLING	3.27 (0.65)	3.64 (0.51)	3.64 (0.81)	3.91 (0.30)	0.07	NONE
PROBLEMS WITH JUMPING	2.0 (1.2)	1.8 (1.0)	3.5 (0.9)	3.9 (0.3)	0.0001	T1<T3 T2<T3 T1<T4 T2<T4
PROBLEMS WITH TURN/CUT	1.36 (0.51)	1.46 (0.82)	3.18 (1.25)	3.73 (0.47)	0.0001	T1<T3 T2<T3 T1<T4 T2<T4
PROBLEMS WITH ABILITY TO RUN	2.82 (1.2)	3.18 (1.17)	3.55 (0.93)	3.91 (0.30)	0.02	T1<T3 T1<T4 T2<T4
PROBLEMS WITH ABILITY TO WALK	4.0 (0.0)	3.9 (0.3)	4.0 (0.0)	4.0 (0.0)	0.41	NONE

ABILITY TO RETURN SPORTS	1.91 (0.83)	2.09 (1.3)	3.73 (0.79)	4.55 (0.69)	0.001	T1<T3 T2<T3 T1<T4 T2<T4 T3<T4

Table 3 demonstrates the responses to the subjective evalutation form shown in figure 1. To evaluate statistically significant changes over time a P value of less than 0.05 was used. T1= preoperation response; T2= 6 month response; T3= 12 month response; T4= 24 month response 4= no problem with activity; 3= mild difficulty with activity; 2= moderate problem with activity; 1= severe problem with activity.

TABLE 4

Genucom Results

Test/Grade	Pre-op	6 months post-op	12 months post-op
30° AP (< 5 mm)	8	12	13
30° AP (≥ 5 mm)	5	1	0

Table 4 includes the results from the Genucom testing. The difference in displacement of the involved knee relative to the displacement of the noninvolved knee was used to evaluate the laxity of the involved knee. The noninvolved knee was used as the control.

TABLE 5

80

Pre-operative and Post Operative Mean Laxity Difference (Non-involved - Involved) (In mm)

Laxity Test	T_1 Pre-Op Mean Difference (S.D.)	T_2 6.0 Months Mean Difference (S.D.)	T_3 12.0 Months Mean Difference (S.D.)	P Value	Statistically Significant Difference
90° AP Drawer					
Anterior	0.86 (2.8)	0.86 (4.13)	-0.87 (2.67)	0.48	
Posterior	-1.36 ((1.74)	0.43 (2.1)	-0.33 (1.91)	0.18	
90° AP Drawer with Internal Rotation					
Anterior	1.27 (3.64)	0.21 (3.07) ·	-0.87 (4.17)	0.56	
Posterior	-0.82 (1.83)	0.64 (3.18)	0 (1.77)	0.22	
90° AP Drawer with External Rotation					
Anterior	-0.45 (2.66)	-0.14 (2.03)	-0.67 (2.29)	0.84	
Posterior	-0.45 (1.86)	0.14 (2.8)	0.13 (1.51)	0.84	
30° AP Drawer					
Anterior	3 (4.42)	-1.21 (3.17)	-0.2 (3.65)	0.03	$T_1 > T_2$
Posterior	-1.14 (2.54)	-1.36 (1.45)	-1.4 (1.68)	0.68	
Dual AP at 30°					
Medial Anter.	3.43 (4.18)	-0.57 (4.15)	0.6 (4.97)	0.14	
Medial Post.	-0.07 (3.38)	-0.5 (1.87)	-0.6 (2.13)	0.74	
Lateral Anter.	2.71 (3.87)	-1.64 (3.97)	0.8 (4.06)	0.02	$T_2 < T_3$, $T_1 > T_2$
Lateral Post.	-0.14 (3.86)	-1.67 (2.26)	-1.47 (2.72)	0.30	
Varus/Valgus					
Varus	0.07 (1.38)	-1.36 (2.13)	-0.8 (1.26)	0.16	
Valgus	-0.71 (2.37)	-0.93 (1.64)	-0.47 (0.99)	0.62	
Internal/External Rotation					
Internal	-0.71 (3.77)	-0.14 (4.33)	-0.27 (3.86)	0.80	
External	-2.23 (6.33)	1.07 (4.34)	-2.13 (4.36)	0.11	
Genu-Flexion	0.36 (7.1)	5.92 (8.37)	4.29 (8.54)	0.26	
Gerurecurvatum Screw Home	-0.43 (3.65)	-1.36 (2.92)	-1.73 (3.53)	0.52	
Medial Compartment Subluxation					
Anterior	1.21 (3.14)	-3 (4.44)	-0.73 (4.79)	0.035	$T_1 > T_2$
Posterior	1.21 (2.81)	-0.29 (4.55)	-0.87 (2.2)	0.17	
Lateral Compartment Subluxation					
Anterior	1.29 (4.92)	-1.29 (3.24)	0.27 (4.3)	0.37	
Posterior	-0.71 (.31)	-1.43 (4.24)	-1.93 (2.4)	0.75	

TABLE 6

PATIENT	KT2000 INVOLVED KNEE DISPLACEMENT (MM)	KT2000 NONINVOLVED KNEE DISPLACEMENT (MM)	KT2000 DIFFERENCE IN LAXITY (INVOLVED KNEE-NONINVOLVED KNEE (MM))	LACHMAN	PIVOT SHIFT
1	1.5	4	-2.5	0/4	0/4
2	12	6	+6.0	2/4	2/4
3	8.5	9.0	-0.5	0/4	0/4
4	7.5	7	+0.5	0/4	0/4
5	2.5	1.0	+1.5	1/4	0/4
6					
7					
8	5.5	8.0	-2.5	0/4	0/4
9	8.0	9.5	-1.5	1/4	1/4
10	10.5	7	+3.5		
11	6.0	8.5	-2.5	0/4	0/4
12				0/4	0/4
13	0.5	0.5	0.0	0/4	0/4

Table 6 demonstrates results from KT200 and manual ligamentous laxity testing twenty-four months after reconstruction.

TABLE 7

**Quadriceps of Involved Leg
Concentric Force Production
in Newtons as Measured
By Kincom
Mean (S. D.)**

Speed °/Sec.	T1 Pre-operation	T2 6 Months Post-operation	T3 12 Months Post-operation	P Value	Statistically Significant Change
60	411.07 (148.56)	435.36 (155.05)	465.69 (164.77)	0.084	-
90	379.43 (132.00)	429.71 (153.42)	453.08 (145.58)	0.006	$T_1 < T_2$ $T_1 < T_3$
120	366.86 (108.55)	405.14 (153.03)	435.15 (156.84)	0.029	$T_1 < T_3$

TABLE 8

**Quadriceps of Involved Leg
Eccentric Force Production
in Newtons as Measured
By Kincom
Mean (S. D.)**

Speed °/Sec.	T1 Pre-operation	T2 6 Months Post-operation	T3 12 Months Post-operation	P Value	Statistically Significant Change
60	471.71 (184.78)	497.71 (178.55)	528.62 (201.13)	0.399	-
90	495.64 (191.52)	522.00 (183.79)	577.15 (192.99)	0.308	-
120	521.50 (173.13)	530.21 (178.22)	563.23 (199.28)	0.637	-

TABLE 9

Quadriceps of Involved Leg
Eccentric Force Production
in Newtons as Measured
By Kincom
Mean (S. D.)

Speed °/Sec.	T1 Pre-operation	T2 6 Months Post-operation	T3 12 Months Post-operation	P Value	Statistically Significant Change
60	574.79 (201.60)	592.50 (198.31)	632.31 (195.25)	0.061	-
90	567.00 (163.58)	603.00 (197.57)	623.00 (188.78)	0.071	-
120	578.71 (185.85)	606.71 (201.65)	618.29 (191.30)	0.164	-

TABLE 10

Quadriceps of Non-Involved Leg
Concentric Force Production
in Newtons as Measured
By Kincom
Mean (S. D.)

Speed °/Sec.	T1 Pre-operation	T2 6 Months Post-operation	T3 12 Months Post-operation	P Value	Statistically Significant Change
60	484.79 (154.76)	494.29 (168.33)	524.08 (201.60)	0.202	-
90	448.00 (134.34)	475.43 (168.07)	511.85 (161.89)	0.053	$T_1 < T_3$
120	437.57 (143.01)	463.00 (168.03)	471.31 (159.91)	0.116	-

TABLE 11

Hamstrings Involved Leg Concentric Force Production in Newtons as measured by Kincom. Mean (S. D.)

Speed °/Sec.	T1 Pre-operation	T2 6 Months Post-operation	T3 12 Months Post-operation	P Value	Statistically Significant Change
60	177.36 (73.48)	160.86 (54.90)	168.00 (70.63)	0.679	-
90	174.29 (65.34)	153.14 (50.91)	157.85 (64.95)	0.547	-
120	158.57 (61.05)	148.36 (48.04)	149.31 (59.53)	0.503	-

TABLE 12

Hamstrings of Non-involved Leg Concentric Force Production in Newtons as Measured By Kincom Mean (S. D.)

Speed °/Sec.	T1 Pre-operation	T2 6 Months Post-operation	T3 12 Months Post-operation	P Value	Statistically Significant Change
60	174.64 (75.16)	170.93 (59.16)	174.46 (61.03)	0.736	-
90	162.21 (63.08)	159.07 (54.37)	164.23 (57.79)	0.518	-
120	155.00 (59.64)	147.00 (49.98)	150.62 (56.02)	0.792	-

TABLE 13

Hamstrings of Involved Leg
Eccentric Force Production
in Newtons as Measured
By Kincom
Mean (S. D.)

Speed °/Sec.	T1 Pre-operation	T2 6 Months Post-operation	T3 12 Months Post-operation	P Value	Statistically Significant Change
60	218.00 (92.66)	180.43 (62.05)	200.23 (86.45)	0.281	-
90	223.50 (84.64)	180.93 (60.68)	197.62 (77.50)	0.098	-
120	218.79 (82.64)	181.71 (60.84)	204.69 (81.49)	0.137	-

TABLE 14

Quadriceps to Hamstrings of Involved Leg
Ratios Over time
Based on Kincom Measurements
Mean (S.D.)

Test	T1 Pre-oop	T2 6.0 Months Post-op	T3 12.0 months Post-op	P Value	Statistically Significant Change
Concentric at 60°/Sec	2.8 (1.4)	2.9 (0.7)	3.0 (0.8)	0.906	-
Concentric at 90°/Sec	2.5 (1.3)	3.0 (0.8)	3.1 (0.8)	0.378	-
Concentric at 120°/Sec	2.7 (1.4)	3.2 (1.0)	3.1 (0.8)	0.849	-
Eccentric at 60°/Sec	2.6 (1.3)	2.9 (0.6)	2.8 (0.7)	0.763	-
Eccentric at 90°/Sec	2.6 (1.4)	3.1 (0.6)	3.0 (0.7)	0.628	-
Eccentric at 120°/Sec	2.6 (0.9)	3.1 (0.8)	2.9 (0.7)	0.562	-

TABLE 15

Quadriceps To Hamstrings of Non-involved
Leg Ratio Over Time
Based on Kincom Measurements
Mean (S.D.)

Test	T1 Pre-op	T2 6.0 months Post-op	T3 12.0 months Post-op	P Value	Statistically Significant Change
Concentric at 60°/Sec	2.9 (0.9)	3.0 (0.9)	3.1 (0.7)	0.684	-
Concentric at 90°/Sec	2.9 (0.8)	3.0 (1.0)	3.3 (0.9)	0.324	-
Concentric at 120°/Sec	3.1 (1.1)	3.2 (1.0)	3.3 (1.0)	0.710	-
Eccentric at 60°//Sec	2.7 (0.6)	3.2 (1.1)	3.3 (0.7)	0.078	-
Eccentric at 90°/Sec	2.8 (0.7)	3.1 (0.9)	3.2 (0.6)	0.031	$T_1 < T_2, T_3$
Eccentric at 120°//Sec	2.9 (0.9)	2.9 (0.7)	3.1 (0.7)	0.620	-

TABLE 16

**Correlations of Involved Legs' Quadricep Strength
With Ability To Return To Sports
R^2 (P Value)**

	T1 Pre-operation	T2 6 Months Post-operation	T3 12 Months Post-operation
Concentric at 60°/Sec	0.001 (0.94)	0.01 (0.74)	0.001 (0.93)
Concentric at 90°/Sec	0.007 (0.78)	0.039 (0.49)	0.001 (0.92)
Concentric at 120°/Sec	0.004 (0.82)	0.07 (0.36)	0.001 (0.91)
Eccentric at 60°/Sec	0.005 (0.80)	0.006 (0.79)	0.006 (0.80)
Eccentric at 90°/Sec	0.001 (0.90)	0.0004 (0.98)	0.002 (0.87)
Eccentric at 120°/Sec	0.008 (0.76)	0.001 (0.92)	0.001 (0.93)

TABLE 17

**Correlations of Involved Legs' Hamstrings
Strength With Ability To Return To Sports
R^2 (P Value)**

	T1 Pre-operation	T2 6 Months Post-operation	T3 12 Months Post-operation
Concentric at 60°/Sec	0.096 (0.28)	0.005 (0.82)	0.038 (0.51)
Concentric at 90°/Sec	0.009 (0.75)	0.008 (0.98)	0.51 (0.44)
Concentric at 120°/Sec	0.052 (0.43)	0.001 (0.92)	0.033 (0.53)
Eccentric at 60°/Sec	0.046 (0.46)	0.002 (0.88)	0.012 (0.70)
Eccentric at 90°/Sec	0.002 (0.87)	0.0004 (0.94)	0.037 (0.51)
Eccentric at 120°/Sec	0.006 (0.80)	0.00007 (0.98)	0.021 (0.62)

TABLE 18

	Correlations of Involved Legs' Quadriceps (Q) To Hamstrings (H) Ratio With Ability To Return To Sports R² (P Value)		
Q/H Concentric at 60°/Sec	0.064 (0.38)	0.025 (0.59)	0.13 (0.20)
Q/H Concentric at 90°/Sec	0.005 (0.81)	0.077 (0.34)	0.076 (0.34)
Q/H Concentric at 120°/Sec	0/052 (0.43)	0.079 (0.33)	0.034 (0.53)
Q/H Eccentric at 60°/Sec	0.061 (0.40)	0.084 (0.31)	0.072 (0.35)
Q/H Eccentric at 90°/Sec	0.014 (0.69)	0.036 (0.51)	0.115 (0.24)
Q/H Eccentric at 120°/Sec	0.037	0.028 (0.57)	0.024 (0.59)

TABLE 19

TEST	INVOLVED LEGS' FORCE PRODUCTION IN NEWTONS (MEAN (S.D.))	NON-INVOLVED LEGS' FORCE PRODUCTION IN NEWTONS (MEAN (S.D.))	P VALUE	STATISTICALLY SIGNIFICANT DIFFERENCE
CONCENTRIC 60 °/SEC	460 (160)	522 (165)	0.005	NON-INVOLVED > INVOLVED
CONCENTRIC 90 °/SEC	451 (140)	512 (156)	0.007	NON-INVOLVED > INVOLVED
CONCENTRIC 120 °/SEC	433 (151)	475 (154)	0.141	NONE
ECCENTRIC 60 °/SEC	520 (196)	629 (188)	0.001	NON-INVOLVED > INVOLVED
ECCENTRIC 90 °/SEC	551 (189)	627 (182)	0.013	NON-INVOLVED > INVOLVED
ECCENTRIC 120 °/SEC	557 (193)	620 (191)	0.007	NON-INVOLVED > INVOLVED

Table 19 includes the KINCOM testing results for the quadriceps 12 months after reconstruction.

TABLE 20

TEST	PREOPERATION			12 MONTHS POST-OPERATION		
	INVOLVED LEGS' FORCE PRODUCTION IN NEWTONS (MEAN (S.D.))	NON-INVOLVED LEGS' FORCE PRODUCTION IN NEWTONS (MEAN (S.D.))	P VALUE	INVOLVED LEGS' FORCE PRODUCTION IN NEWTONS (MEAN (S.D.))	NON-INVOLVED LEGS' FORCE PRODUCTION IN NEWTONS (MEAN (S.D.))	P VALUE
CONCENTRIC 60 °/SEC	177 (74)	175 (76)	0.77	178 (74)	175 (61)	0.44
CONCENTRIC 90 °/SEC	174 (65)	162 (63)	0.05	158 (65)	164 (56)	0.36
CONCENTRIC 120 °/SEC	159 (61)	155 (60)	0.48	149 (60)	151 (56)	0.89
ECCENTRIC 60 °/SEC	218 (93)	209 (91)	0.47	224 (85)	202 (80)	0.92
ECCENTRIC 90 °/SEC	224 (85)	218 (94)	0.64	198 (78)	200 (71)	0.68
ECCENTRIC 120 °/SEC	219 (83)	220 (82)	0.85	205 (82)	212 (80)	0.35

Table 20 demonstrates the KINCOM testing results. The hamstrings of the involved and non-involved legs are compared preoperatively and 12 months postoperatively.

TABLE 21

**MRI measurements
in MM Mean (S.D.)**

Location	Pre-op	1.5 Months	6.0 Months	12.0 Months		P Value	Statistically Significant Change
A1	6.0 (1.1)	9.6 (2.5)	10.0 (2.4)	10.0 (2.6)		0.0001	$T_1 < T_1,' T_2,' T_3$
A2	5.0 (0.8)	9.2 (2.8)	8.6 (2.4)	8.0 (2.1)		0.0001	$T_1 < T_1,' T_2,' T_3$
A3	5.9 (1.0)	8.8 (2.4)	10.0 (2.6)	8.9 (2.7)		0.0001	$T_1 < T_1,' T_2, T_3$
B	45.3 (7.2)	46.1 (9.0)	42.9 (7.8)	45.4 (7.9)		0.2172	-
C1	29.3 (3.7)	31.5 (4.7)	29.9 (4.7)	29.4 (4.1)		0.1443	-
C2	27.6 (3.8)	27.1 (3.6)	26.9 (4.3)	26.6 (4.1)		0.8246	-
C3	25.6 (3.7)	23.6 (4.0)	24.1 (4.4)	24.3 (4.1)		0.6316	-
D	11.6 (2.6)	16.3 (5.0)	15.2 (5.0)	13.4 (4.5)		0.0054	$T_1 < T_1', T_2, T_3<$

TABLE 22

ACL Graft Overall   **Scale = 0-4**

Patient	T_1 (1.5 Months)	T_2 6 Months	T_3 12 Months	T_4 24 Months
1	2.0	3.5	3.0	3.0
2	1.0	2.0	2.25	2.0
3	2.5	2.5	2.5	
4	1.0	1.5	1.0	2.5
5	1.5	3.0	3.0	3.5
6	1.5	3.0	2.5	
7	2.5	3.0	3.0	
8	1.0	1.0	1.0	1.0
9	2.0	4.0	3.0	3.0
10	1.5	2.0	2.0	2.0
11	1.5	1.5	3.0	
12	2,0	2.5	3.0	
13	2.5			2.5
Mean	1.73	2.46	2.44	2.44

TABLE 23

Effusions/MRI

	1.5 Months (T_1')	6.0 Months (T_2)	12.0 Months (T_3)	
MEAN	2.7 (0.7)	2.1 (0.7)	1.6 (0.5)	
MEDIAN	3.0	2.0	2.0	
RANGE	1 - 4	1 - 3	1 - 2	

Table 24

Magnetic Resonance Imaging Evaluation of Menisci

| Patient	Pre-op Medial	Pre-op Lateral	1.5 Months Medial	1.5 Months Lateral	6 Months Medial	6 Months Lateral	12 Months Medial	12 Months Lateral	Surgery
1	-	posterior horn tear	-	bucket handle posterior horn tear	decrease in tear	decrease in tear	-	-	Partial lateral meniscectomy
2	-	-	posterior horn tear	posterior horn tear	decrease in tear	decrease in tear	no tear	no tear	No surgery
3	-	-	posterior horn tear	posterior horn tear	decrease in tear	no change	decrease in tear	partial (single image)	No surgery
4	posterior horn tear	-	posterior horn tear	posterior horn tear	no change	no change	no change	no change	Medial meniscus repair
5	stage II tear	tear	partial	almost	increase in tear	-	increase in tear	-	No surgery
6	tear	-	meniscectomy	total meniscectomy	decrease in tear	increase in posterior horn tear	increase in tear	increase in tear Body & Post horn. no change	Partial lateral meniscectomy
7	bucket handle tear	partial single image	posterior horn and body tear	stage II partial tear	decrease in tear	no change	increase in tear	no change	Partial medial meniscectomy
8	no scan	no scan	stage II meniscectomy	stage II	no change	increase in tear	no change	no change	Partial medial meniscectomy
9	-	-	posterior horn tear	-	decrease in tear	decrease in posterior horn tear	increase in tear	-	No surgery
10	posterior horn and bucket handle tear	posterior horn/body tear	tear	tear	decrease in tear	decrease in tear	no tear	increase in tear	Partial medial meniscectomy
11	posterior horn and bucket handle prior meniscectomy tear	-	-	-	increase in tear	increase in tear	partial	-	Previous partial medial meniscectomy 2nd partial medial meniscectomy 9 mos. after reconstruction
12	degenerative	flap post. horn	degenerative tear	residual tear	no change	no change	no change	no change	Partial medial meniscectomy / Medial meniscus lateral meniscectomy
13	tear	flap tear	residual tear	partial meniscectomy	no scan	no scan	18 months no change	Degenerative Ant-horn tear (1 cm)	Partial medial meniscectomy / Medial meniscus lateral meniscus repair
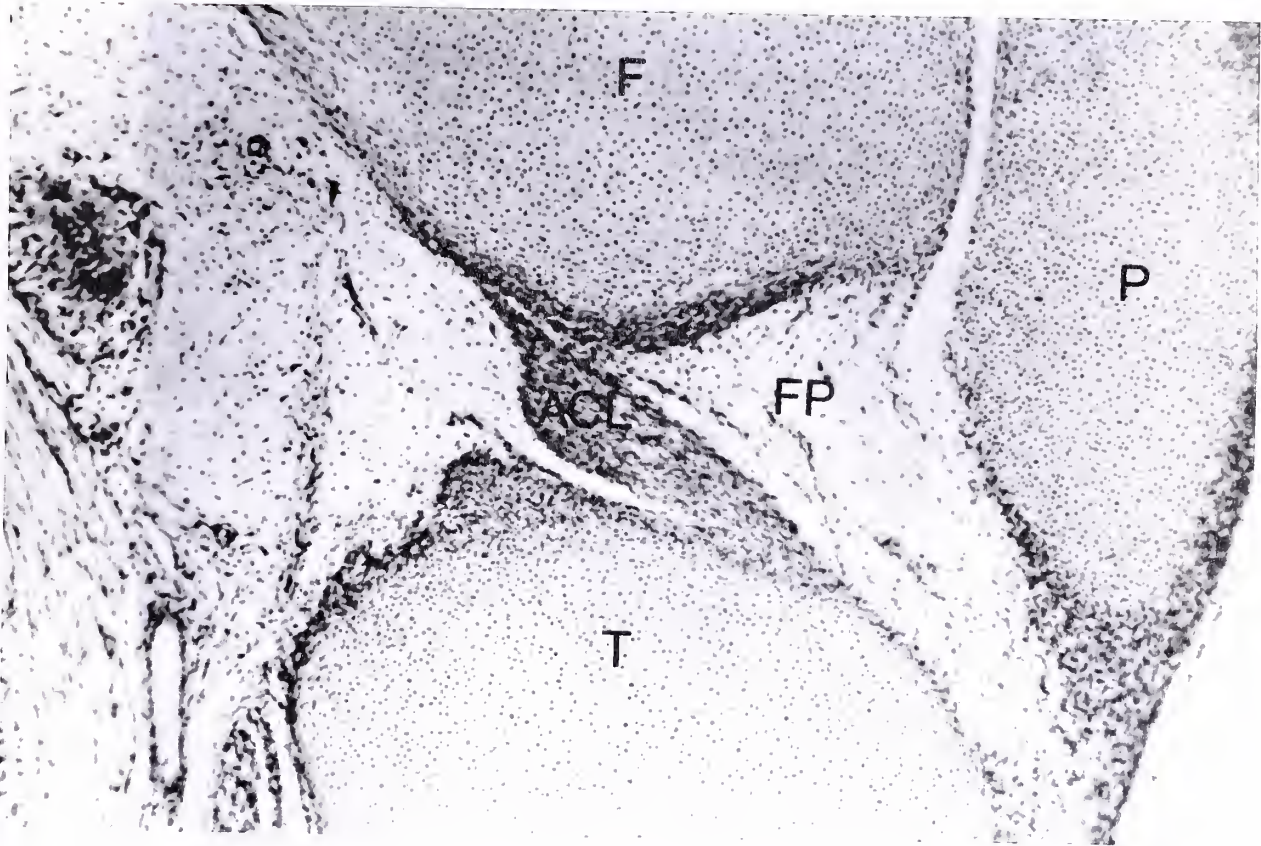
FIG. 1 Photomicrograph of a sagittal section of a human knee joint in approximately the 20th week of development. The anterior cruciate ligament (ACL), infrapatellar fat pad (FP), and patella (P) are clearly visible. F, femur; T, tibia. (H & E, original magnification X 40.) (Feagin: The Cruciate Ligaments p. 181.)

FIG. 2 Photograph of a human knee joint at approximately the 20th week of development. Note that the cruciate ligaments and menisci are fully formed at this time. (Feagin: The Cruciate Ligaments p. 181.)
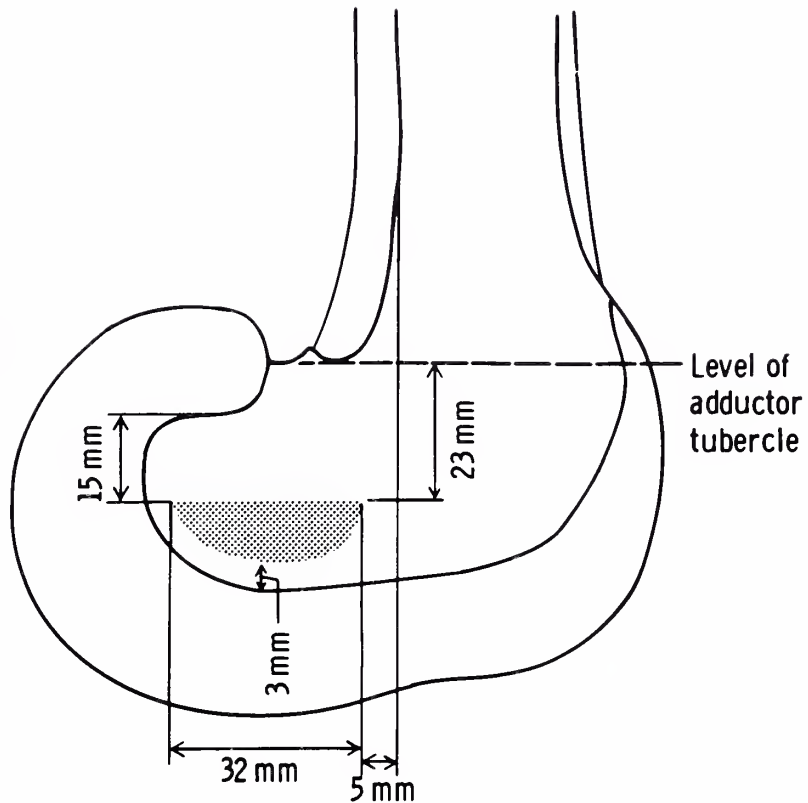
FIG. 3 Drawing of the lateral surface of the medial condyle of the femur showing the average measurements and bony relations of the femoral attachment of the PCL. (Girgis FG, Marshall JL, Monajem ARS: The cruciate ligaments of the knee joint: anatomical, functional, and experimental analysis. Clin. Orthop. 106L:216-231, 1975.)
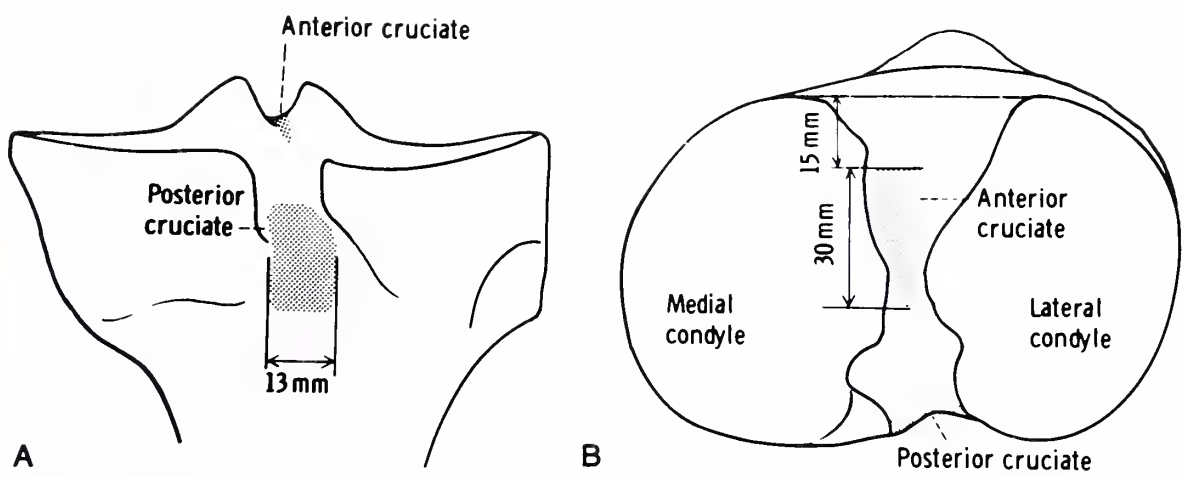
FIG. 4 Drawing of (A) the posterior surface of the tibia and (B) the upper surface of the tibial plateau to show average measurements and relations of the tibial attachments of the ACL and PCL. (Girgis FG, Marshall JL, Monajem ARS: The cruciate ligaments of the knee joint; anatomical, functional, and experimental analysis. Clin. Orthop. 106L:216-231, 1975.)
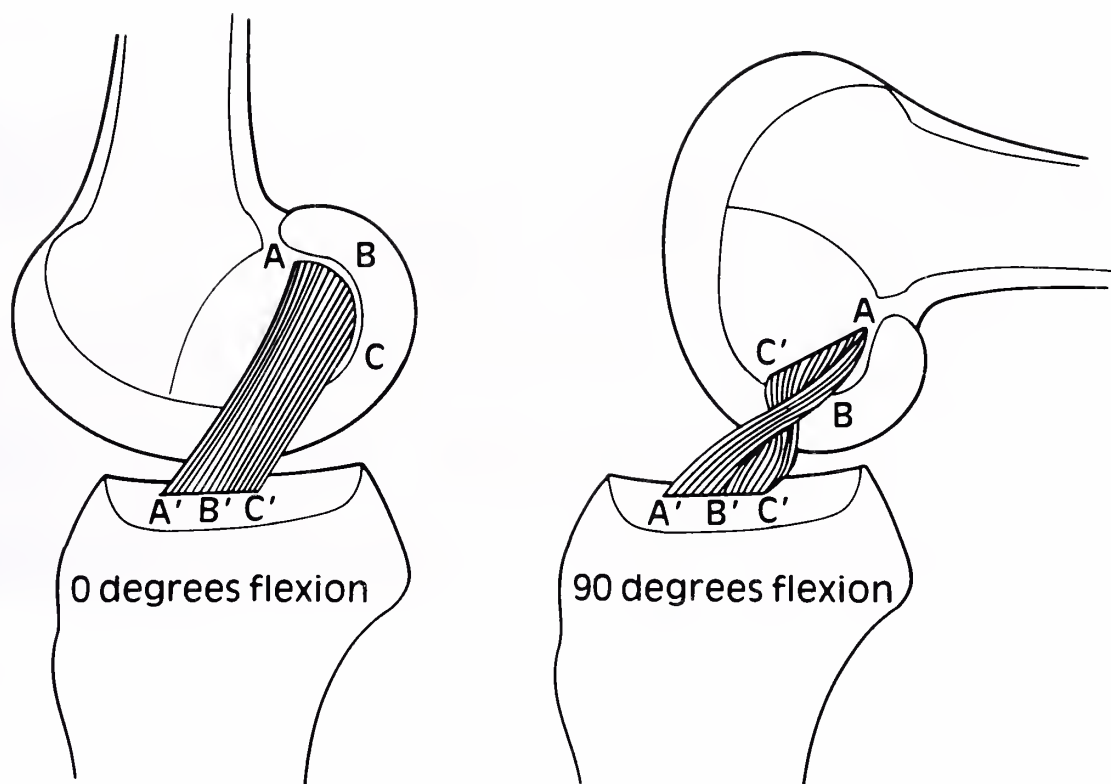
FIG. 5 Schematic drawing representing changes in the shape and tension of the ACL components in flexion and extension. In flexion, there is lengthening of the anteromedial band (A-A') and shortening of the posterolateral aspect of the ligament (C-C'). Also present, however, is an intermediate component (B-B') which represents the transition between the anteromedial band and posterolateral bulk, with fascicles in varying degrees of tension. (Girgis FG, Marshall JL, Monajem ARS: The cruciate ligaments of the knee joint: anatomical, functional, and experimental analysis. Clin. Orthop. 106L:216-231, 1975.)
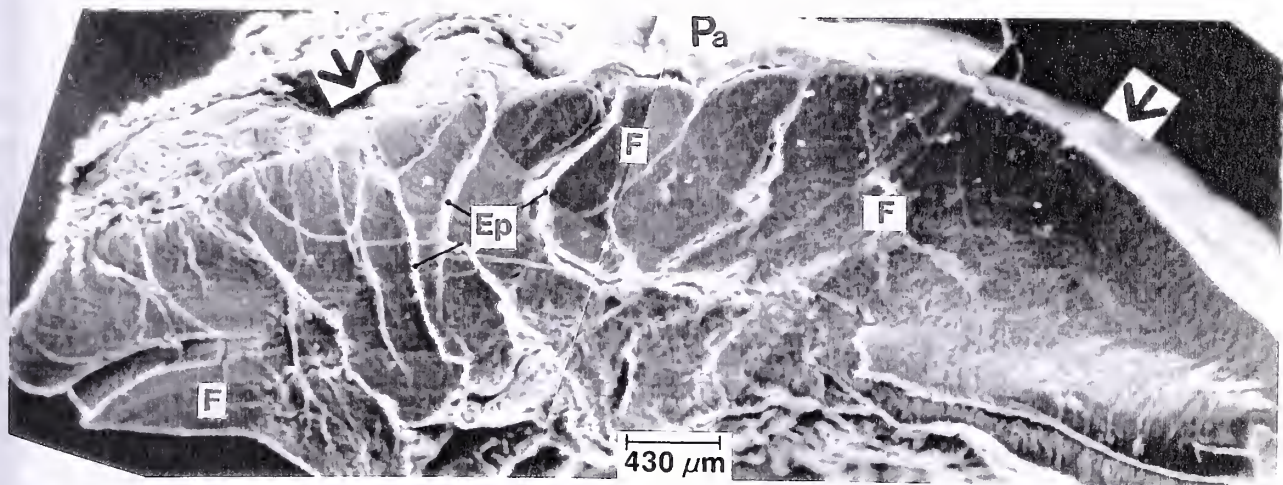
FIG. 6 SEM micrograph of tibial cross-section of a canine ACL showing individual fascicles delineated by epitenon (Ep) on the left and a single fascicle (F) on the right. The sheath seen on the anterior side of the ligament (arrow) is the paratenon (Pa) embedded in a loose connective tissue (original magnification, X29). (Yahia and Drouin G: Canine ACL and Patellar Tendon, J. Orthop. Res. 7(2), 1989.)
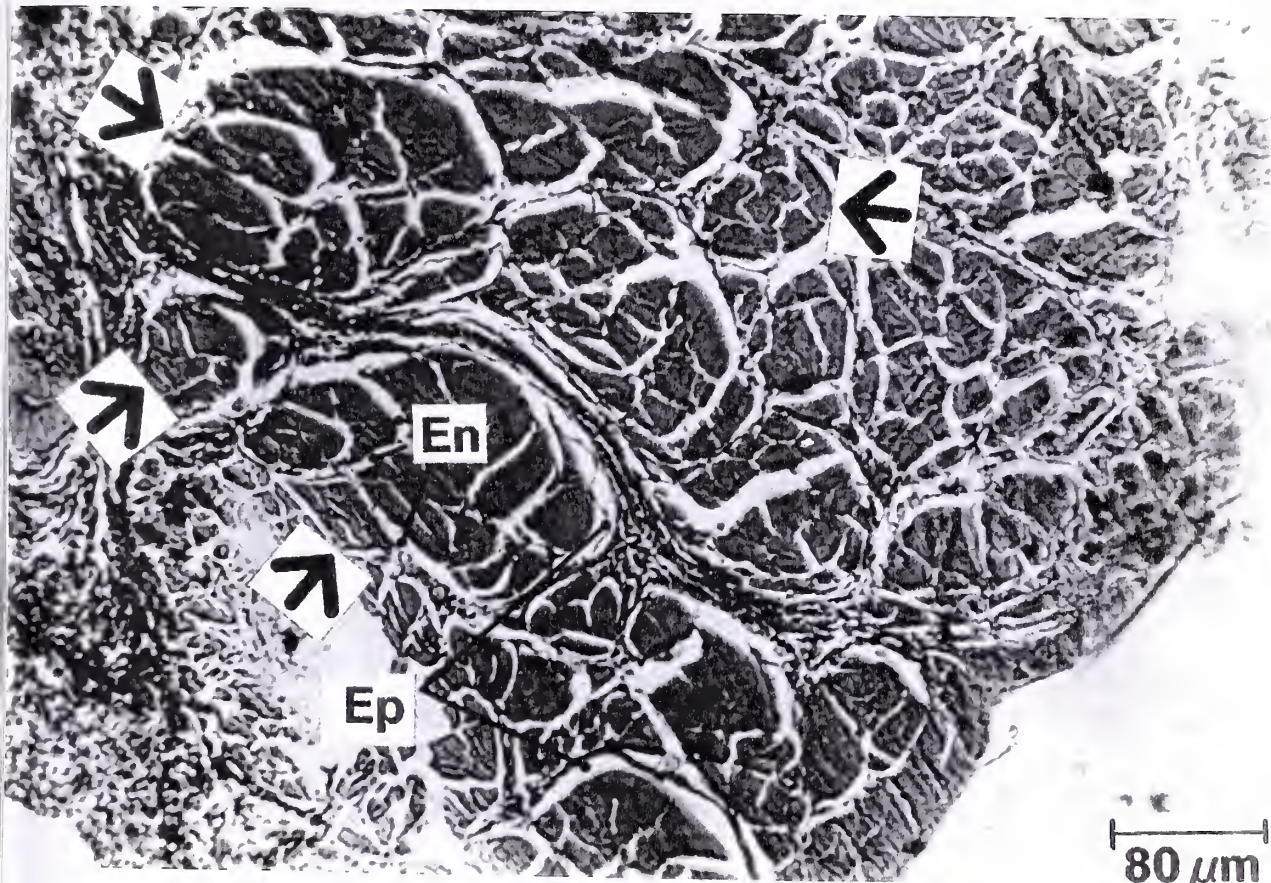
FIG. 7 LM micrograph of a canine ACL cut transversely at the tibial level. Arrows indicate fascicles of different sizes covered by epitenon (Ep) and with nearly elliptical outlines. Each fascicle is subdivided into subfascicle by the endotenon (En) (Hemalunpholoxin-safran, X140). (Yahia and Drouin G: Canine ACL and Patellar Tendon, J. Orthop. Res. 7(2), 1989.)
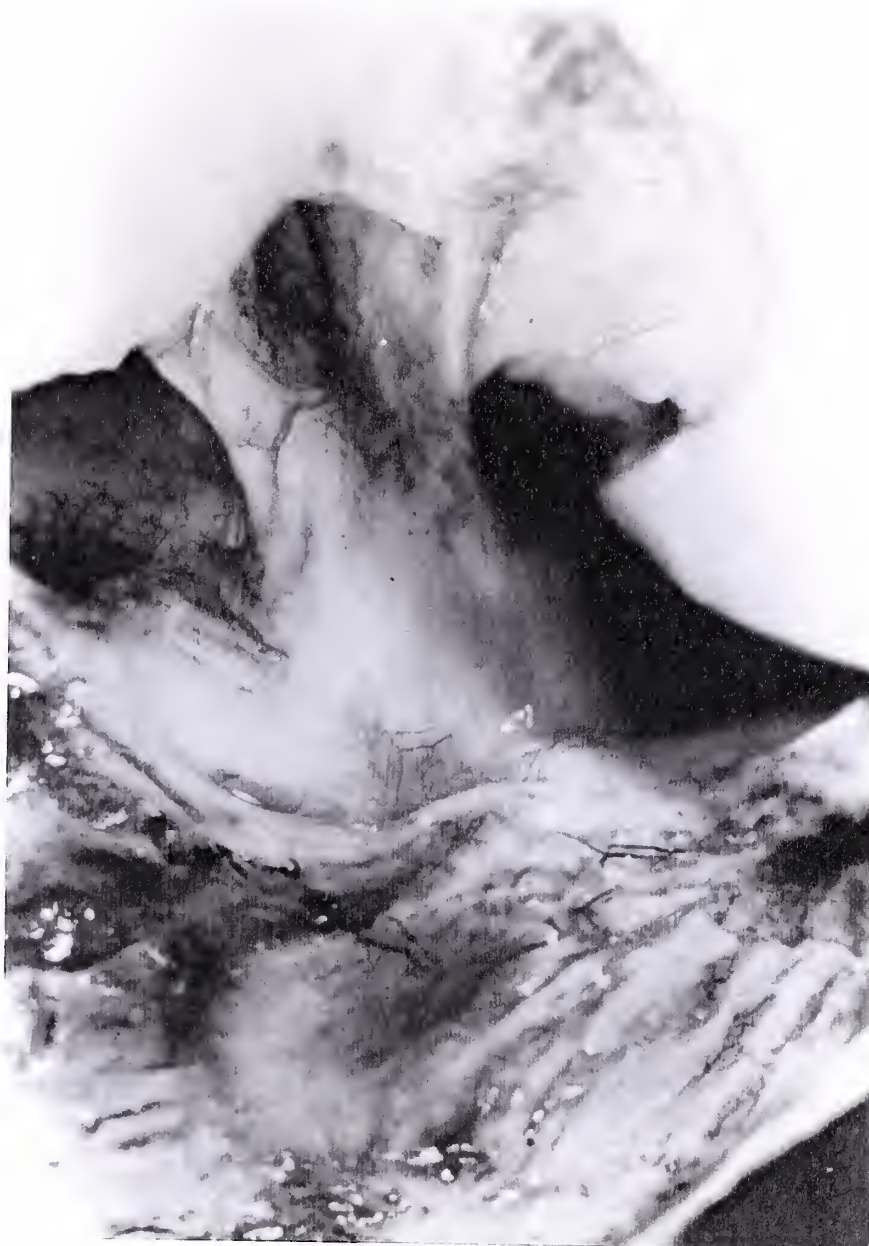
FIG. 8 Photograph of a human knee specimen injected with India ink demonstrating the synovial (periligamentous) vasculature on the surface of the ACL. (Note that the infrapatellar fat pad has been removed for better visualization). (Arnoczky AP, Blood supply to the anterior curciate ligament and supporting structures, Orthop. Clin. North Am 16:15-28, 1985.)
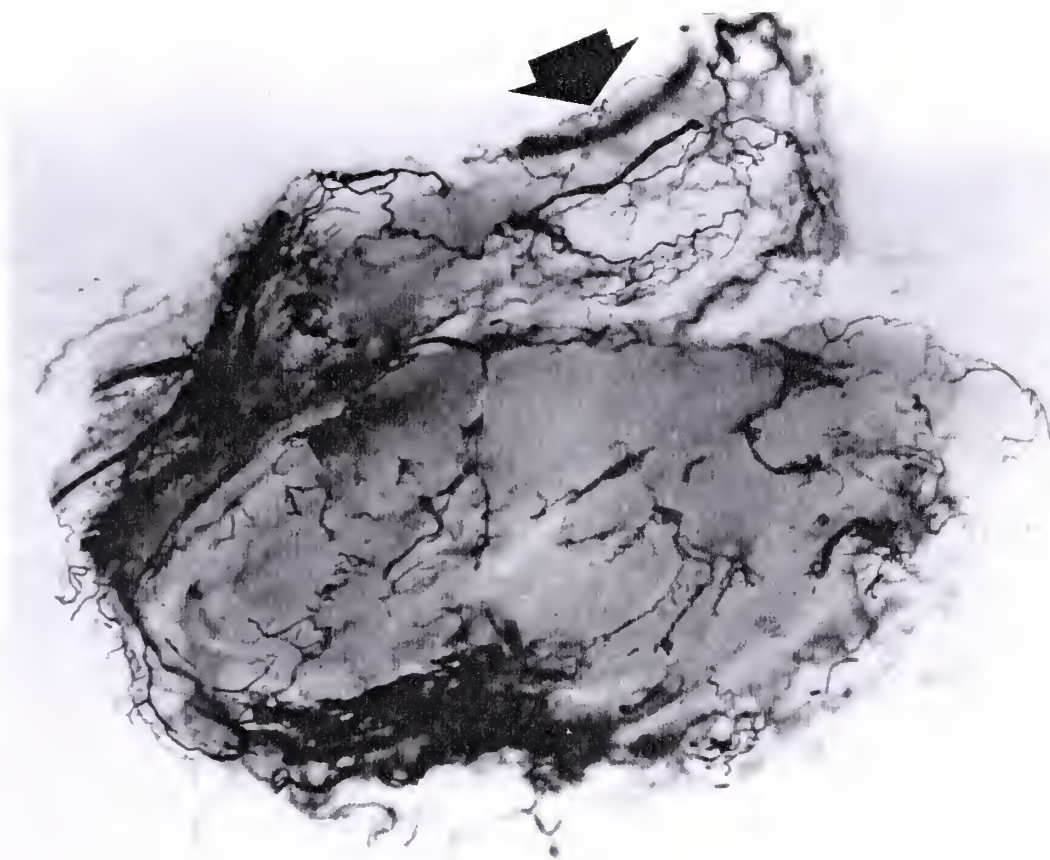
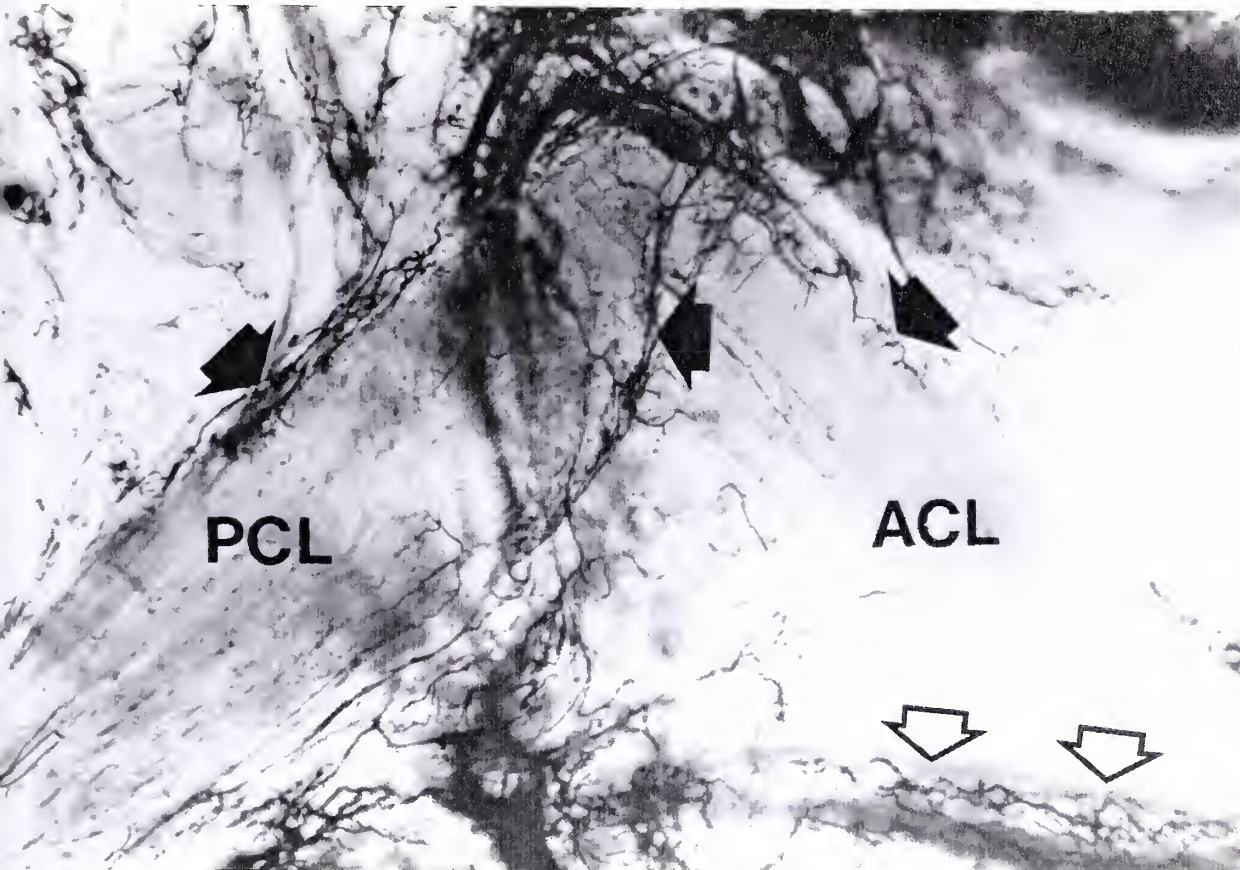FIG. 9 Cross-section of a human ACL (Spalteholz technique) demonstrating the periligamentous as well as endoligamentous vasculature. The fold of synovial membrane (arrow) can be seen supplying vessels to the synovial covering of the ligament. (Arnoczky SP, Anatomy of the anterior cruciate ligament, Clin. Orthop. 172:19-25, 1983.)
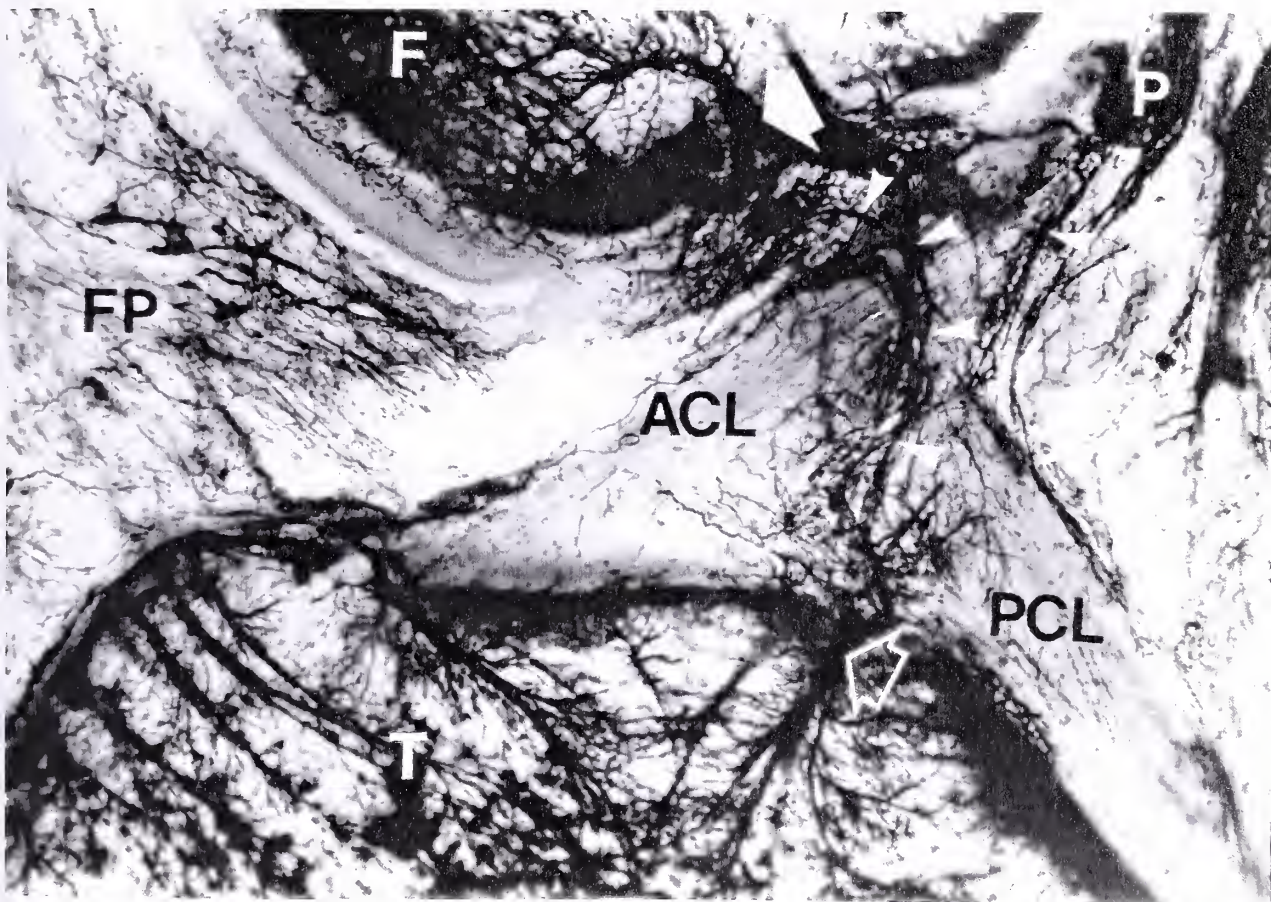
FIG. 10 Sagittal 5 mm thick section of a human knee (Spalteholz technique) showing the branches of the middle genicular artery that supply the distal femoral epiphysis (large white arrow), the proximal tibial epiphysis (large open arrow), and the cruciate ligaments (small white arrowheads). (F, femur; T, tibia; FP, fat pad; P, popliteal artery.) (Arnoczky AP, Blood supply to the anterior curciate ligament and supporting structures, Orthop. Clin. North Am 16:15-28, 1985.)

FIG. 11 Sagittal 5 mm thick section of a human knee joint (Spalteholz technique) showing the periligamentous vasculature of the ACL and PCL (closed arrows). Note the absence of vessels crossing the ligamentous-osseous attachment of the ACL (open arrows). (Arnoczky AP, Blood supply to the anterior curciate ligament and supporting structures, Orthop. Clin. North Am 16:15-28, 1985.)
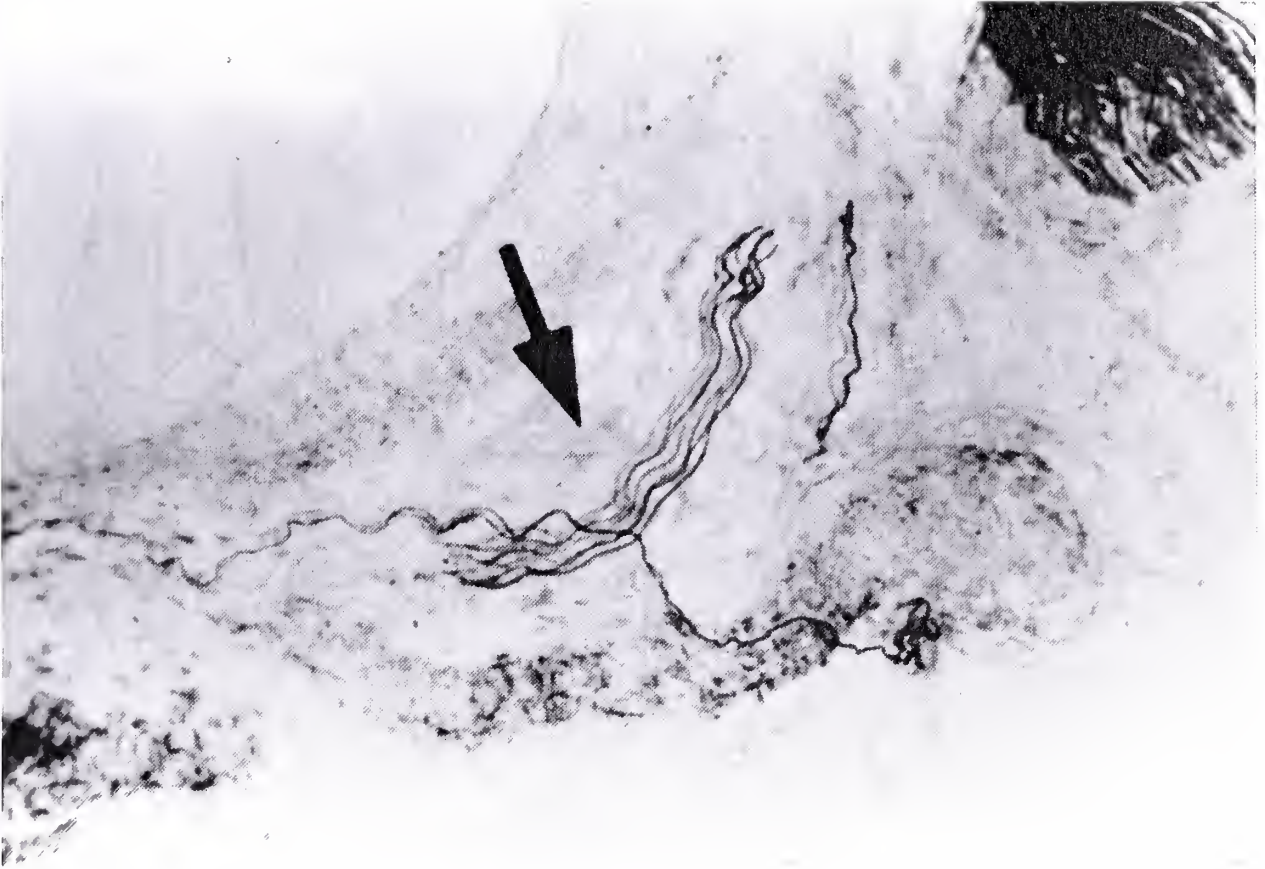
FIG. 12 Photomicrograph of an ACL showing nerve fibers in the synovium investing the ligament. (Kennedy JC, Alexander IJ, Hayes KC, Nerve supply of the human knee and its functional importance, Am. J. Sports Med. 10:329-335, 1982.)
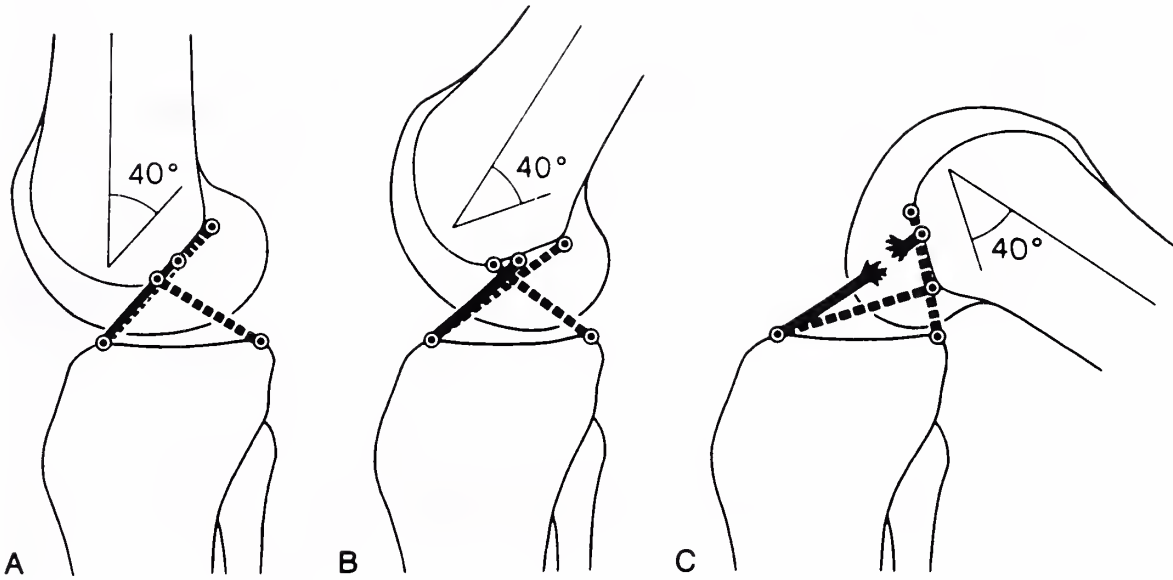
FIG. 13 Improperly inserted, the cruciate ligaments cannot trace out circular arcs during flexion. Thus, an ACL reinserted too far anteriorly (A) becomes lax at about 40° of flexion (B) and at 120 of flexion it is too short and must tear (C). (Muller W, The Knee: Form, Function, and Ligament Reconstruction, Springer-Verlag, Berlin, 1982.)
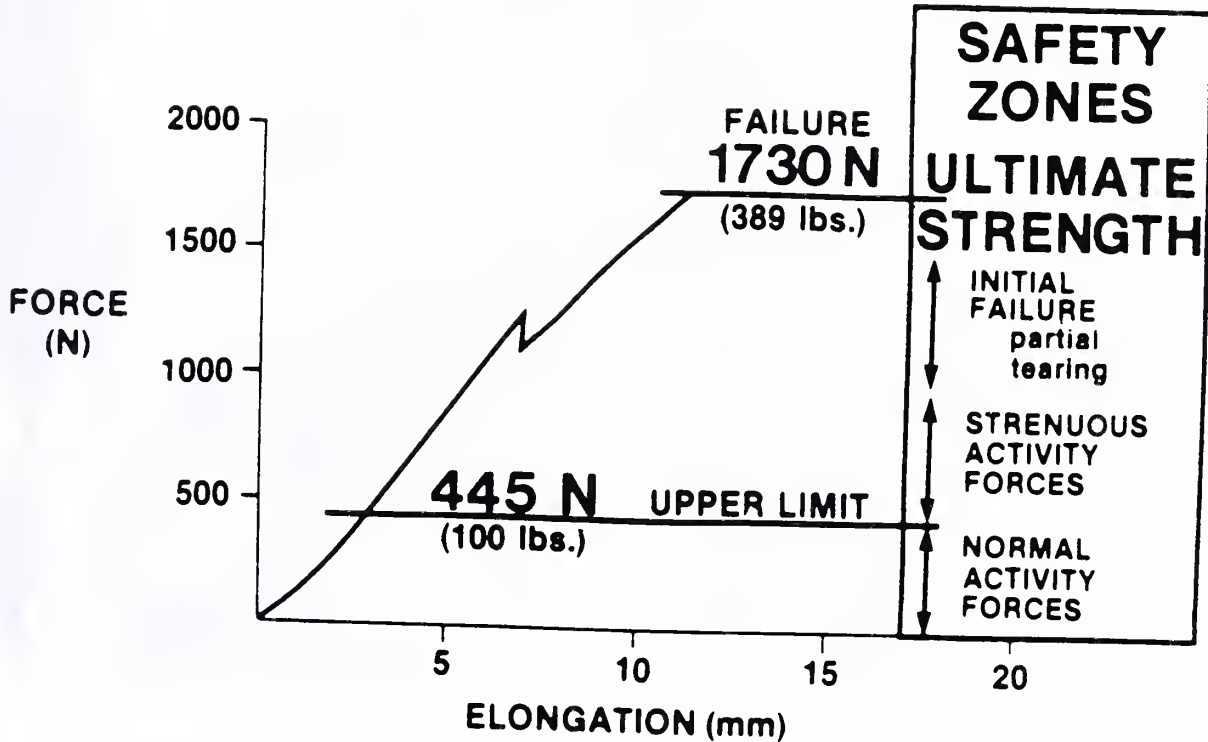
FIG. 14 A hypothetical load-elongation curve for the ACL showing failure at 1730 N (389 lb) with partial tearing occurring at lower loads. (From Noyes FR, at al) Biomechanical analysis of human ligament grafts used in knee ligament repairs and reconstructions, J. Bone Joint Surg. 66A:344-352, 1982; with permission.)
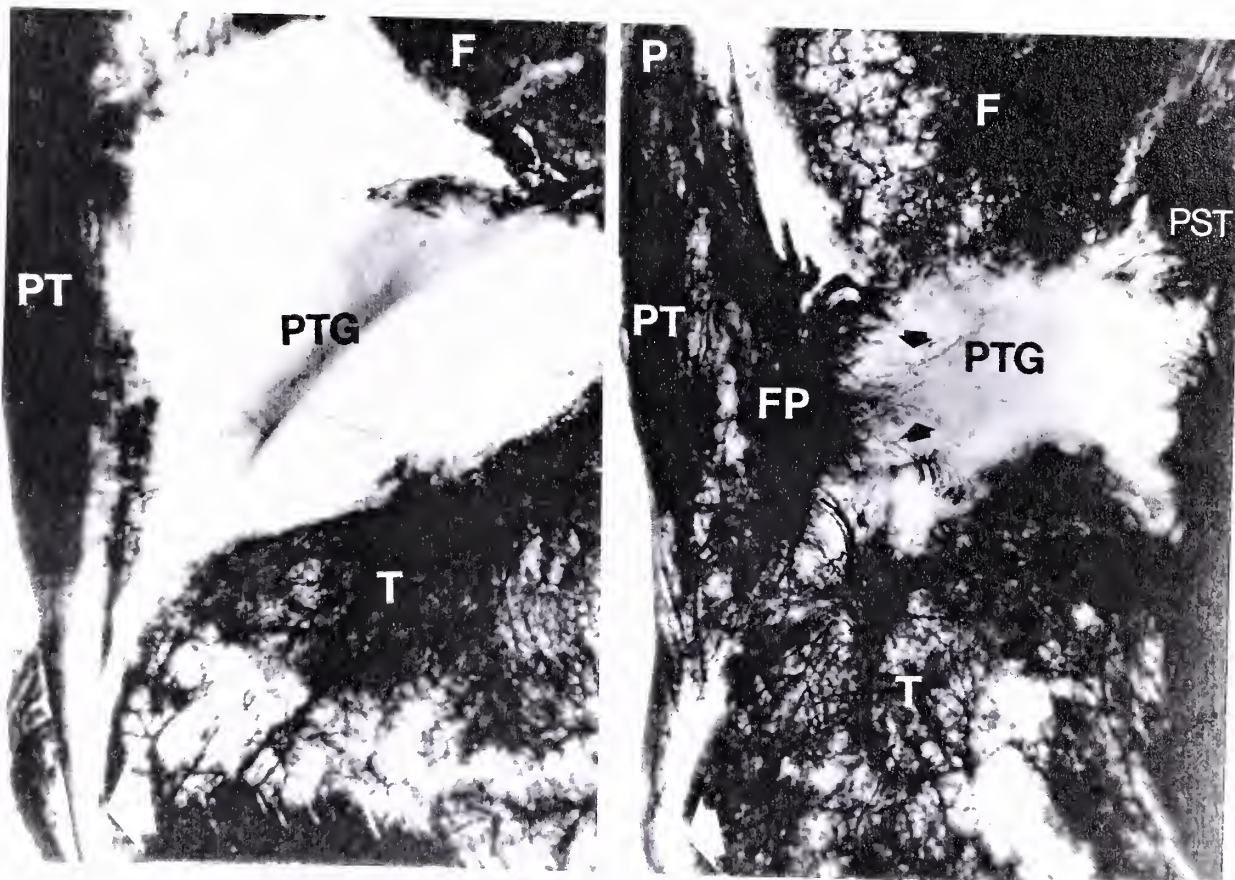
FIG. 15 A five-millimeter-thick sagittal section of a canine knee cleared by the Spalteholz technique, two weeks after replacement with a patellar tendon graft (X2). The graft (PTG shows no evidence of perfused vessels. Note the absence of vessels crossing the tibial attachment of the graft (arrow). (The infrapatellar fat pad and posterior cruciate ligament were removed after clearing to permit better visualization.) F=femur, T=tibia, and PT=patellar tendon. (Arnoczky SP, Tarvin GB, and Marshall JL, Anterior Cruciate Ligament Replacement using Patellar Tendon, J. of Bone & Joint Surg., 64A:217-224, 1982.)

FIG. 16 A five-millimeter-thick sagittal section of a dog's knee six weeks after replacement of the anterior cruciate ligament with a patellar tendon graft (PTG). Note the vascular response of the infrapatellar fat pad (FP) and posterior soft tissues (PST). Vessels from the fat pad can be seen extending over the surface of the patellar tendon graft (arrows) and are part of the vascular synovial envelope. Note that the tibial attachments of the graft (white arrow) do not contribute to any vessels to the graft. F=femur, T=tibia, PT=patellar tendon, and P=patella. (Arnoczky SP, Tarvin GB, and Marshall JL, Anterior Cruciate Ligament Replacement using Patellar Tendon, J. of Bone & Joint Surg., 64A:217-224, 1982.)
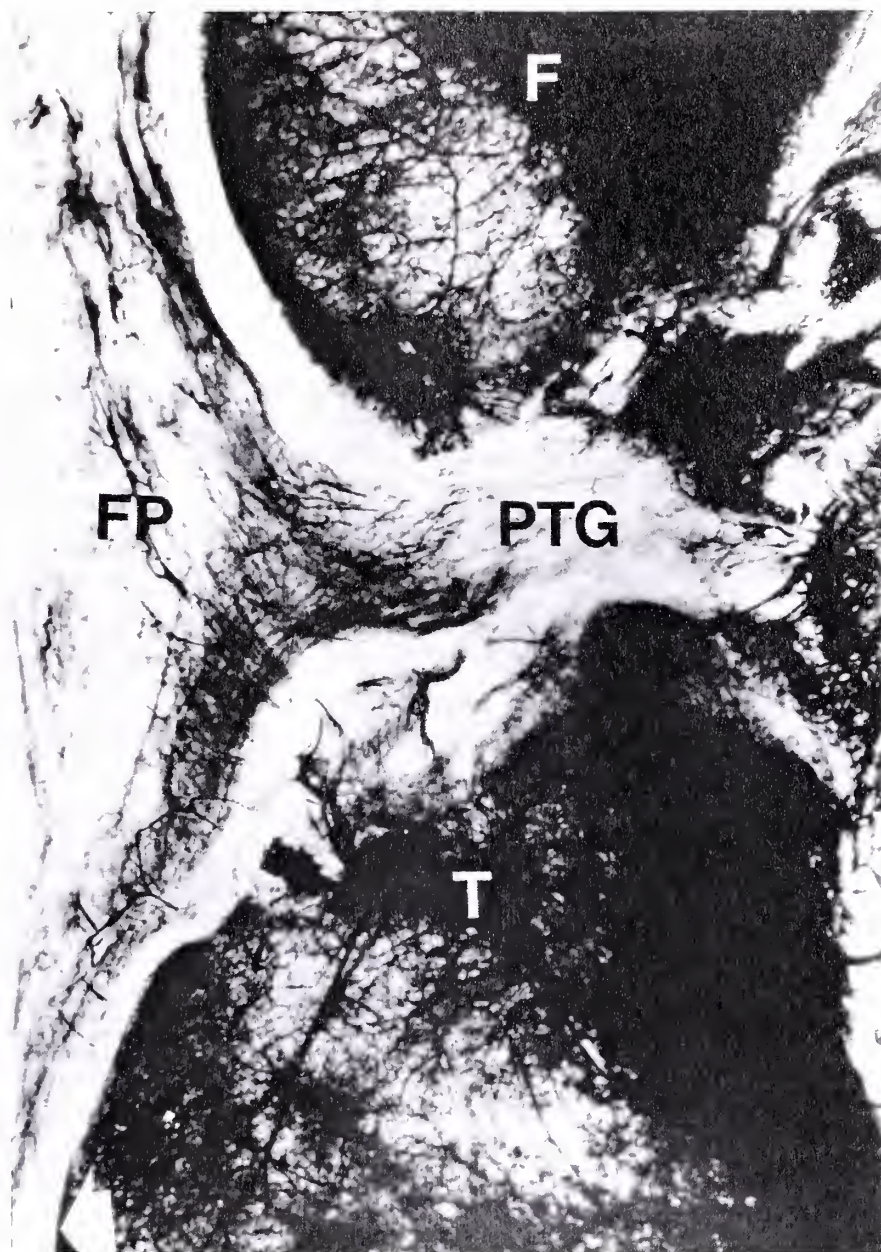
FIG. 17 A five-millimeter-thick sagittal section of a dog's knee twenty-six weeks after replacement of the anterior cruciate ligament with a patellar tendon graft (Spalteholz, X2). The vascular response of the infrapatellar fat pad (FP) as well as of the intrinsic vessels of the graft (PTG) has subsided. Note that even at twenty-six weeks the tibial attachment of the graft (white arrow) contributed no vessels to the revascularization process. (The posterior cruciate ligament wa removed after clearing to permit better visualization.) F=femur and T=tibia. The redundancy of the patellar tendon graft in this specimen is apparent as the graft crosses the joint space and displays a rich vascular attachment to the fat pad. This animal demonstrated five millimeters of anterior drawer when it was killed, but was not clinically lame. (Arnoczky SP, Tarvin GB, and Marshall JL, Anterior Cruciate Ligament Replacement using Patellar Tendon, J. of Bone & Joint Surg., 64A:217-224, 1982.)

Table 10-4. Primary and Secondary Ligamentous Restraints to Laxity Tests

Laxity Test	Primary Restraint				Secondary Restraint		
	Medial	Central	Lateral		Medial	Central	Leteral
A. Anterior Drawer	—	ACL	—	20°/90°	TCL + MM	—	ALS
B. Anterior Drawer + internal rotation	—	ACL	ALS	20°/90°	—	—	FCL + PLS
C. Anterior Drawer + exterior rotation	TCL + MM	ACL	—	20°/90°	PMS	—	—
D. FRD, pivot shift	—	ACL	—	15°	MM + TCL + PMS	—	ALS + FCL
E. Posterior Drawer	—	PCL	—	20°/90°	PMS + TCL	—	FCL + PLS
F. Posterior Drawer + external rotation	—	—	FCL + PLS	30°	—	PCL	—
	—	PCL	FCL + PLS	90°	—	—	—
G. Posterior Drawer + internal rotation	TCL + PMS	—	—	20°	—	ACL + PCL	—
	TCL + POL	PCL	—	90°	—	ACL	—
H. Valgus	TCL + PMS	—	Bone	5°	—	PCL + ACL	—
	TCL	—	Bone	20°	PMS	PCL	—
I. Varus	Bone	—	FCL + PLS	5°	—	ACL + PCL	—
	Bone	—	FCL	20°	—	ACL	PLS
J. External rotation	PMS + TCL	—	FCL + PLS	30°	MM	PCL	—
	MM + TCL	PCL	FCL + PLS	90°	PMS	—	—
K. Internal rotation	TCL + PMS	ACL	ALS	20°	—	PCL	FCL
	TCL + POL	ACL + PCL	ALS	90°	—	—	FCL

ALS, iliotibial band plus anterior plus midlateral capsule
PLS, popliteus, posterolateral capsule
PMS, posterior oblique ligament plus posteromedial capsule
MM, medial meniscus
POL, posterior oblique ligament
TCL, tibial collateral ligament
FCL, fibular collateral ligament
(From ref. 19a, with permission.)

Table 1. Primary and Secondary Ligamentous Restraints to Laxity Tests

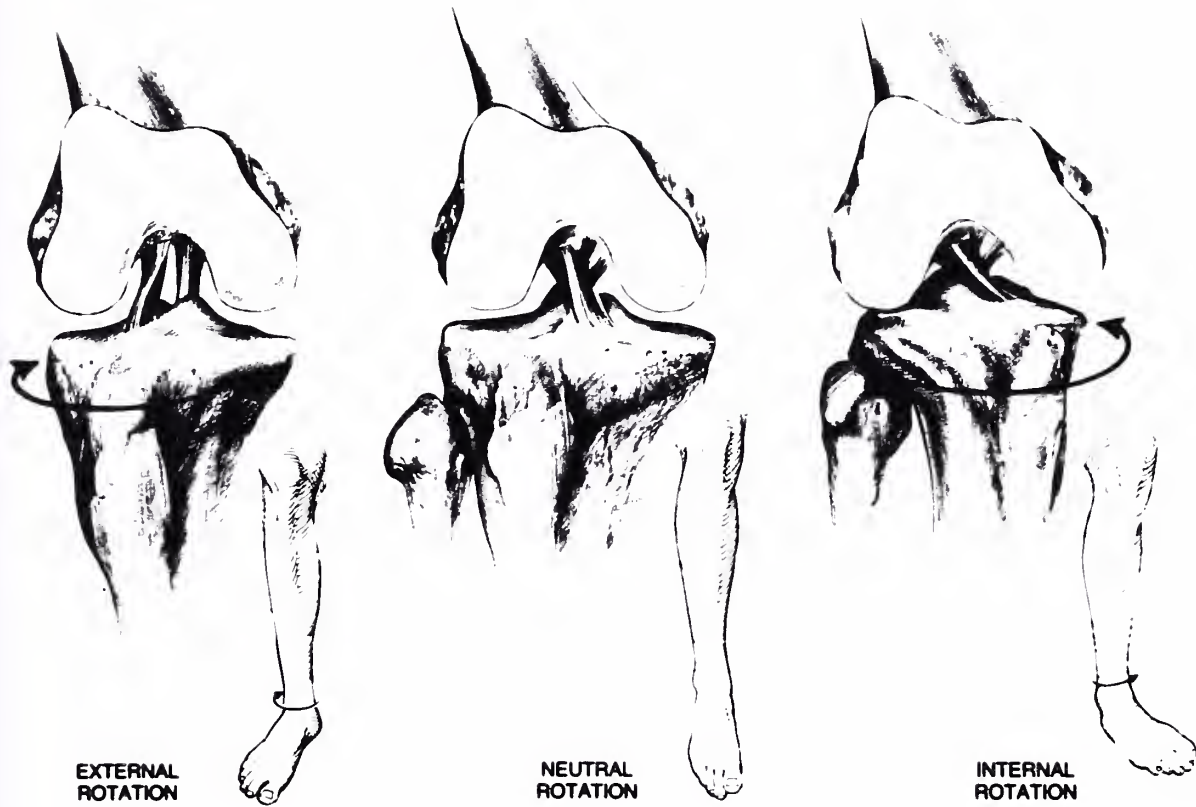(Table 1 - Feagin: The Cruciate Ligaments p. 277)

FIG. 18 Hip and knee flexed 90 ; tibia externally rotated, in neutral position, and internally rotated. Note tightening of the cruciates with internal rotation. (Feagin: The Cruciate Ligaments)
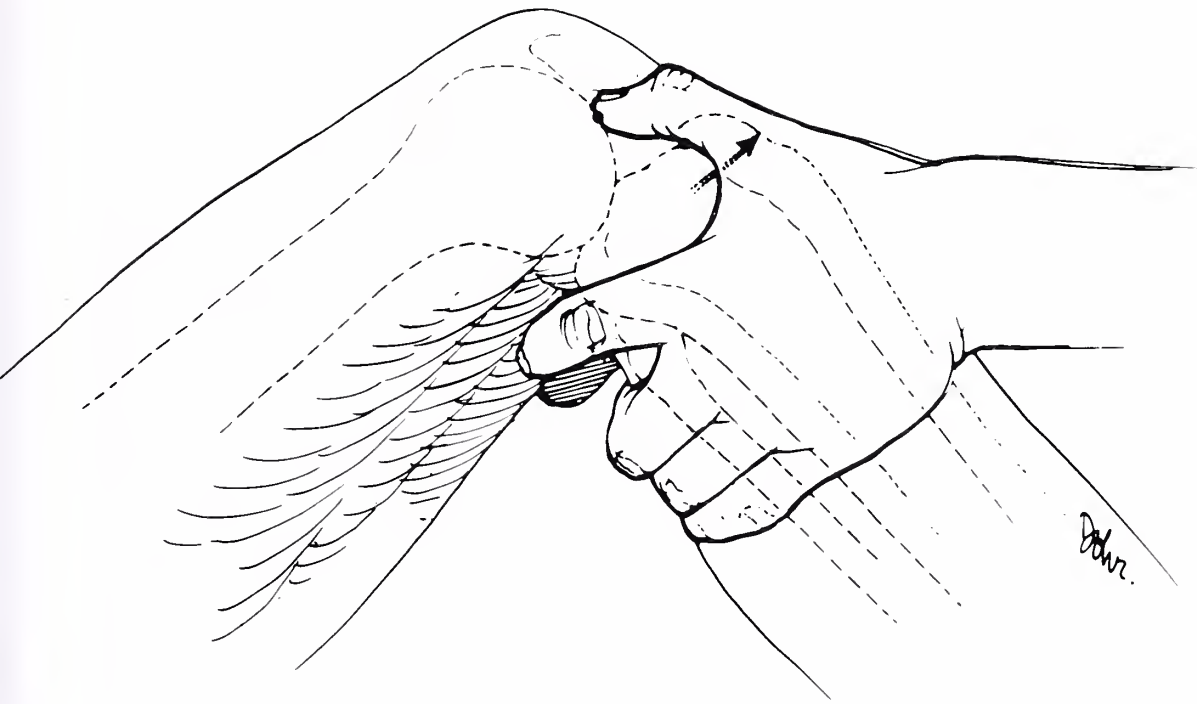
FIG. 19 AP glide with thumbs measuring the translation of the tibia on the femur.
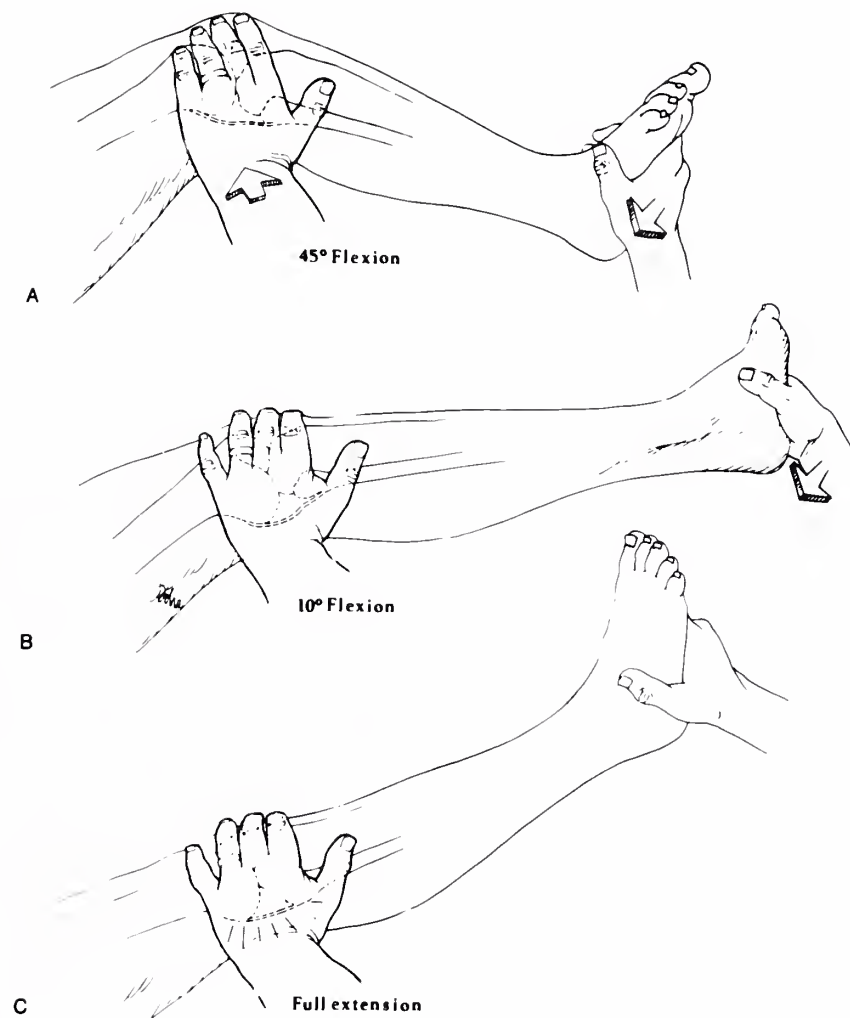
FIG. 20 Losee's test for the pivot shift by extending the knee from a reduced to a subluxed position. (A) The 45 ° flexed knee is reduced with the foot and tibia twisted externally. Push the knee and pull the foot to compress the lateral joint compartment. (B) Let the knee extend while maintaining strong lateral compartment compression. Let the tibia twist internally as the joint subluxes with a thud between 20° and 10° . (C) complete extension quietly reduces the knee as the posterior capsule tightens. (Feagin: The Cruciate Ligaments)
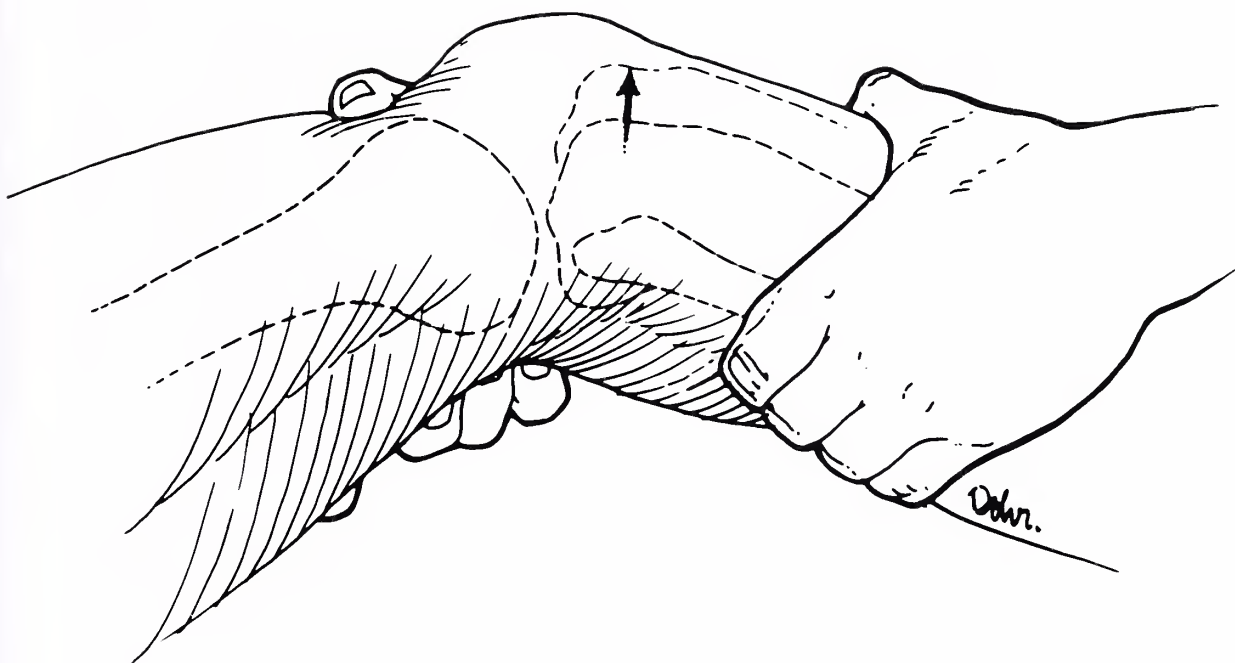
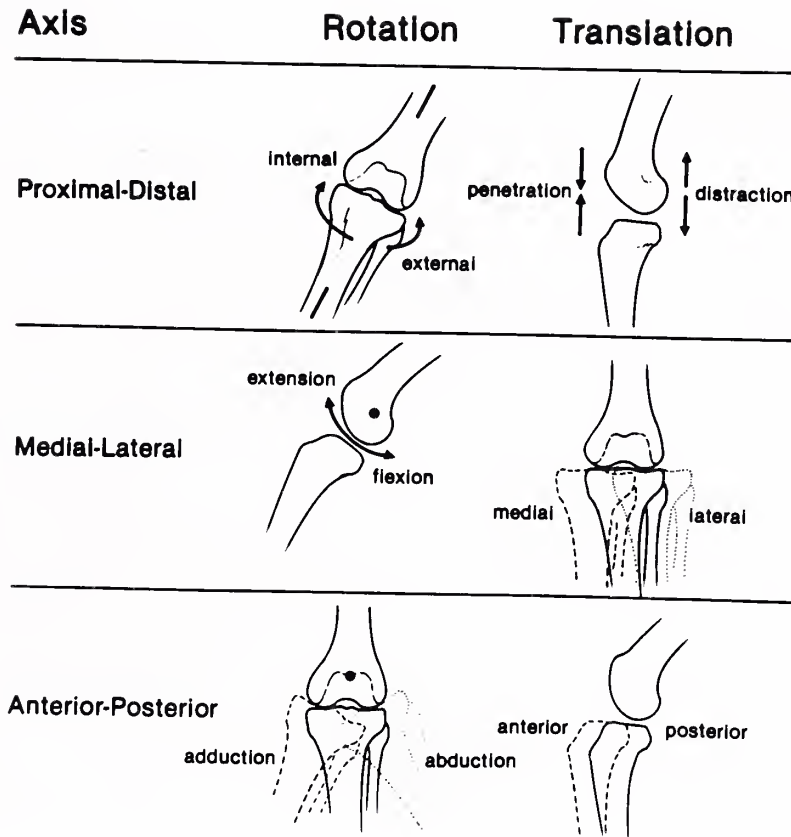FIG. 21 The Lachman test. (Feagin: The Cruciate Ligaments)

FIG. 22 The three knee axes. Around each axis there is rotation and along each axis there is a translation to give a total of six degrees of freedom (6DOF). (Feagin: The Cruciate Ligaments)

FIG. 23 GENUCOM

FIG. 24 and 24A GENUCOM

FIG. 25 An anatomically placed patellar tendon graft with interference fit of bone plugs in an enlarged intercondylar notch to reconstitute the central pivot of the knee. (Feagin: The Cruciate Ligaments, p. 402)

FIG. 26 Marking incisions for the saw cuts are made in the patella and tibial tuberosity projecting from the tendon incisions. (Feagin: The Curciate Ligaments, p. 403)
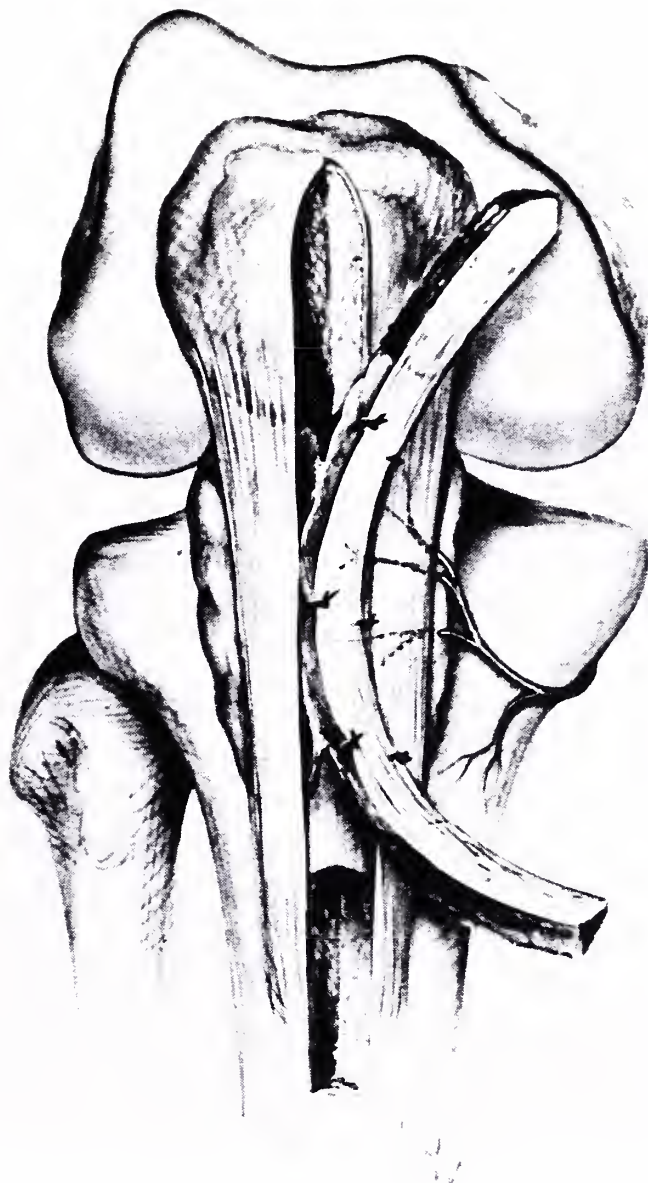
FIG. 27 Exposure to the intercondylar notch is accomplished by making a complete incision through the fat pad laterally, carefully avoiding damage to the lateral meniscus. The blood supply to the graft is thereby maintained from the medial inferior genicular artery. (Feagin: The Cruciate Ligament, p. 403)
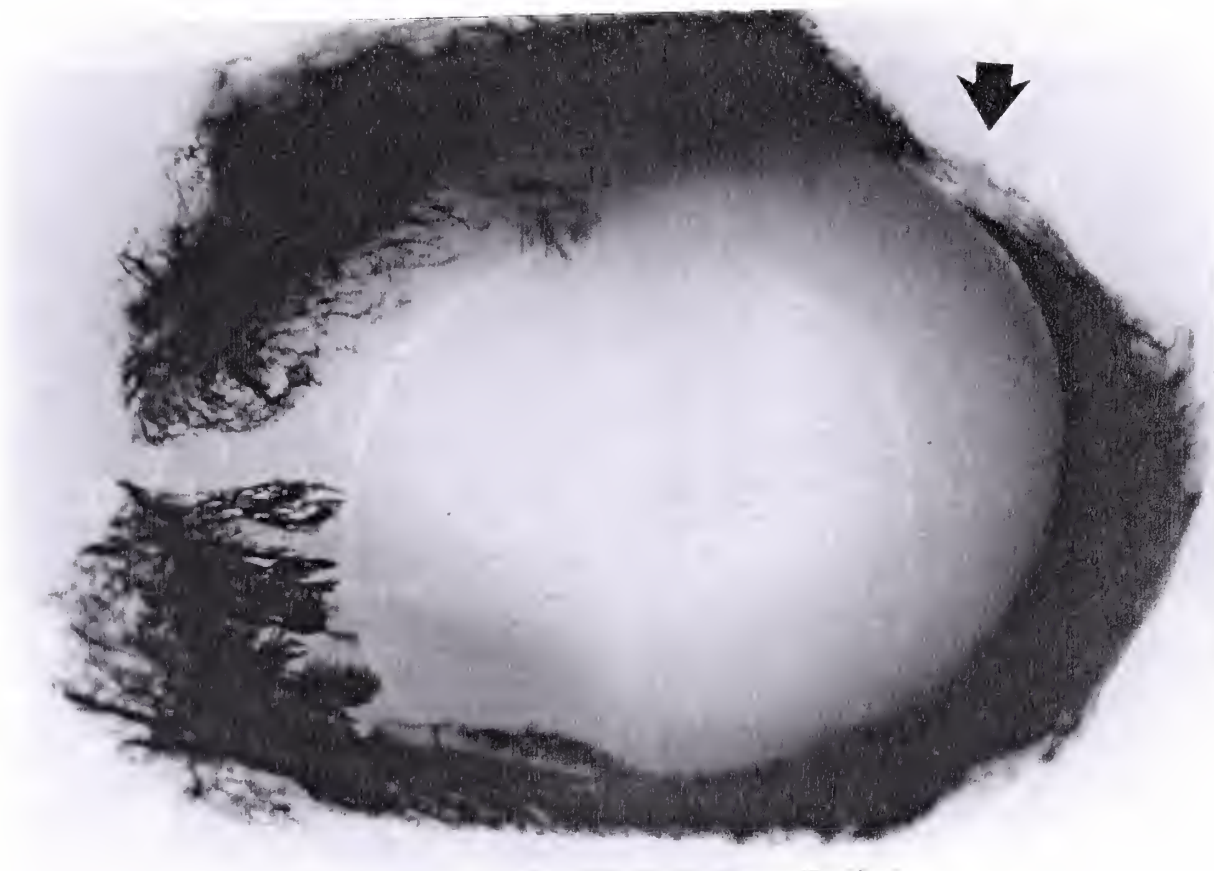
FIG. 28 Superior view of lateral meniscus after vascular perfusion with india ink using the Spalteholtz technique demonstrates vascularity at the periphery of the meniscus as well as increased vascularity at the anterior and posterior horns. The peripheral vasculature is absent at the posterolateral corner (arrow). This represents the region through which the popliteal tendon passes. (Reproduced with permission from Arnoczky SP, Warren RF: Microvasculature of the human meniscus. Am J Sports Med 1982;10:90-95.)

FIG. 29 Classification scheme depicting types of meniscal tears. A, posteromedial; F, posterolateral; O, synovial meniscal junction; I, outer third of the meniscus; 2, middle third of the meniscus; and 3, inner third of the meniscus.
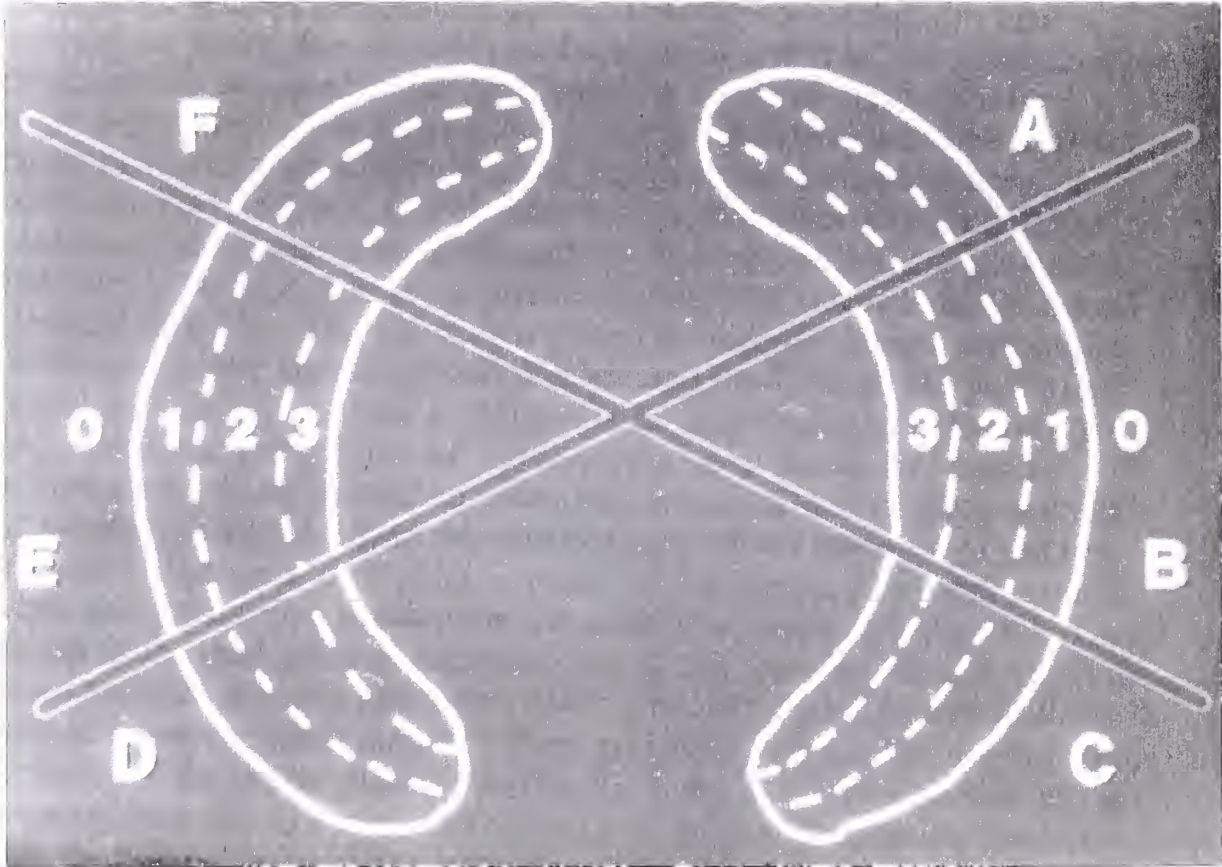
APPENDIX 1
SURGICAL TECHNIQUE

"The first step in an arthroscopic-assisted patellar tendon reconstruction for anterior cruciate instability is a diagnostic arthroscopy ... Once the diagnostic and other appropriate arthoscopic procedures are performed, the anthroscope is placed in the inferior medial patellar portal, and a pituitary rongeur, a curette, and motorized instruments are placed through the medial parapatellar portal to perform a notchplasty and to debride the lateral formal condyle of synovium and scar." A notchplasty is performed to accomodate the graft which is a greater mass than the original ligament. "Once the lateral femoral condyle is debrided so that the entire posterior edge and its junction with the roof of the intercondylar notch can be visualized, a small curette is used to make a small hole for placement of the rear-entry drill guide." "A lateral skin incision is made and carried through the vastus lateralis fascia. The vastus lateralis is then elevated (Fig.5A). The gaf is then placed through the lateral parapatellar portal around the lateral femoral condyle and out through the posterior lateral capsule and soft tissue adjacent to the intermuscular septum (Fig. 6A). Once the femoral tunnel is drilled, the tibial tunnel and patellar tendon graft will be developed. An anterior medial skin incision is made ... and then carried down to the superior level of the insertion of the pes tendons." The tibial drill guide is placed in the medial parapatellar portal while the arthroscope is placed in the inferior medial patellar portal. Once the tip of the drill guide is correctly placed, a K wire is drilled through the guide and into the tibia and then a 10-mm reamer is used to overdrill the K wire to create the tibial tunnel." "Next an isometer is used to determine the accuracy of the tunnel placement (Fig. 9A and 10A). The skin incision is retracted laterally to expose the central one-third of the patellar tendon. This portion of the patellar tendon measuring 10 mm wide, is developed along with its bony attachments. Three No. 5 nonabsorbable sutures are placed through each bone block, and the graft is then pulled through the femoral and the tibial tunnel (Fig. 12A). The surgeons (P.J. and K.L. modified Clancy's technique by holding the bone-tendon-bone graft in place with Kurasaka screws rather than using buttons as Clancy describes. (W.G. Clancy, pp. 14-15, Techniques in Orthop. 1988, 2(4); 13-22)
*REFER TO FIGURES 1A-13A.
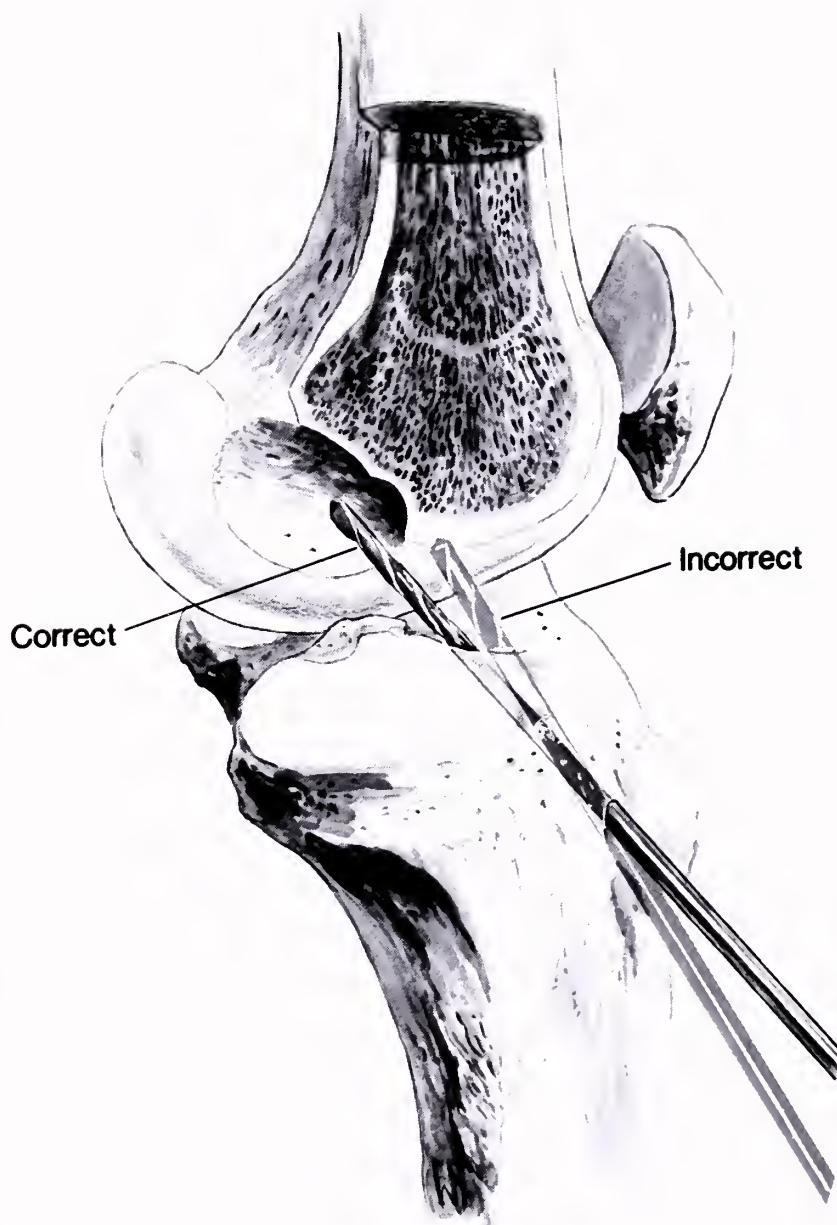
FIG. 1A. The guide is disassembled and removed, leaving the target drill in place. The tip of the target drill is visualized arthroscopically. On extension of the knee, the tip of the target drill should not impinge on the anterior aspect of the intercondylar notch.
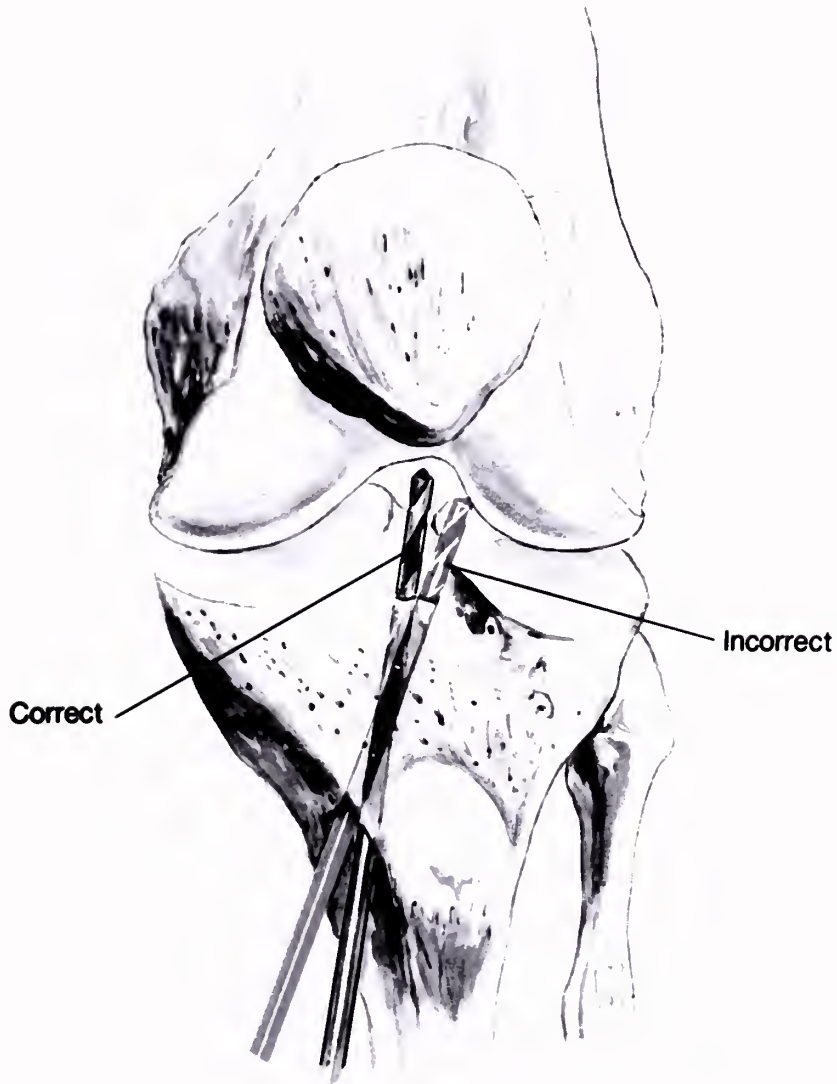
FIG 2A. The target drill should be placed far enough medially to avoid impingement on the lateral femoral condyle. The target drill should align with the intended course of the ACL graft.

FIG. 3A. A tissue protector and cannulated reamer are then placed over the target drill and the tibial tunnel is drilled to the appropriate size.

FIG. 4A. A curette is introduced into the tibial tunnel to chamfer the proximal end of tunnel. chamfering the tunnel edges, particularly the posterolateral aspect, will minimize the abrasion on the graft during range of motion.
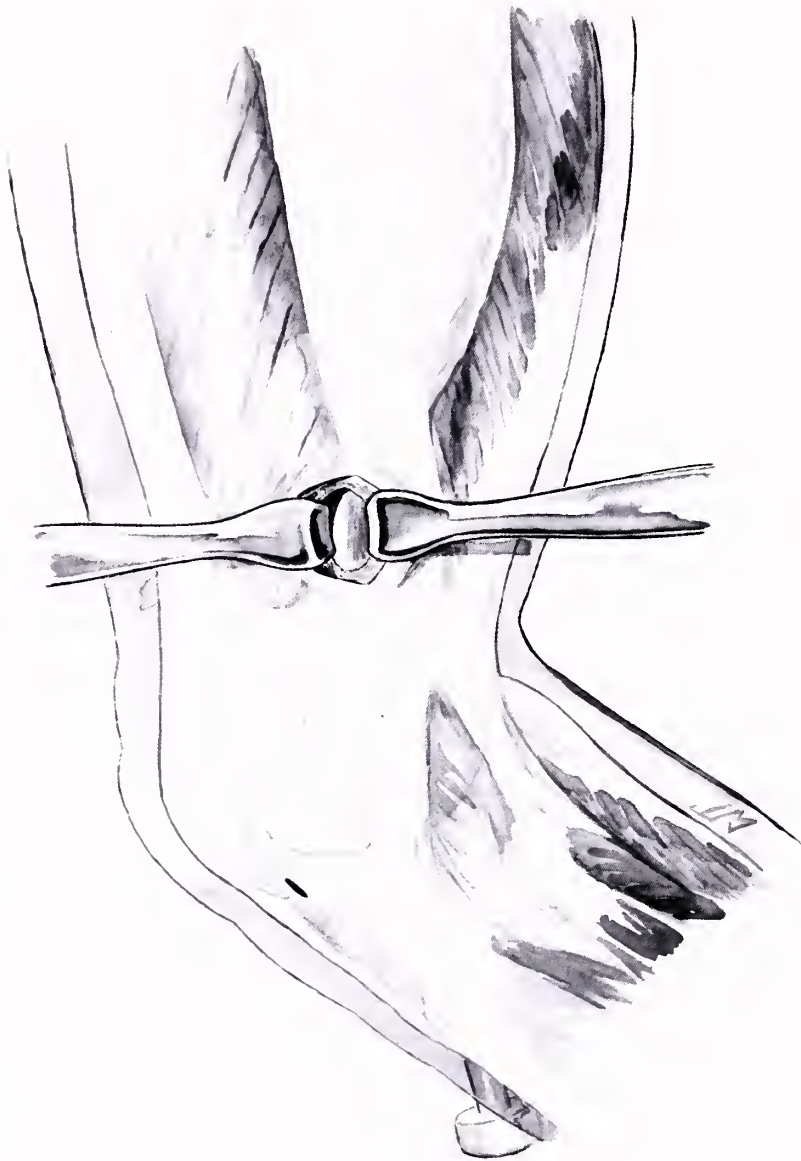
FIG. 5A. The iliotibial band is incised along its anterior margin. The vastus lateralis is retracted anteriorly, exposing the lateral femoral cortex.

The retractors are removed and subcutaneous dissection is carried distally to the posterior edge of the lateral femoral condyle. The iliotibial band is again incised just posterior to its union with the intermuscular septum and anterior to the biceps femoris.
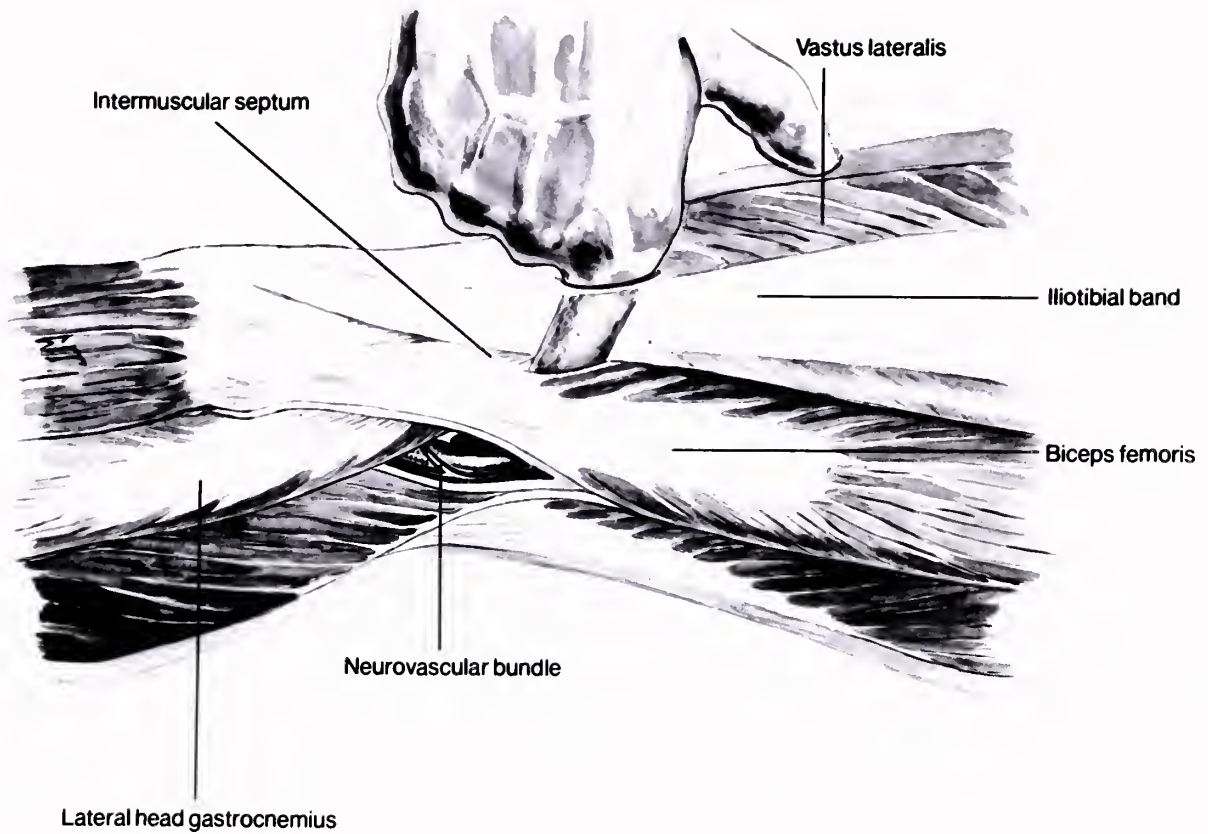
FIG. 6A. Blunt dissection is performed manually, passing a finger posterior to the intermuscular septum, anterior to the biceps femoris, around the lateral head of the gastrocnemius, and into the popliteal fossa.
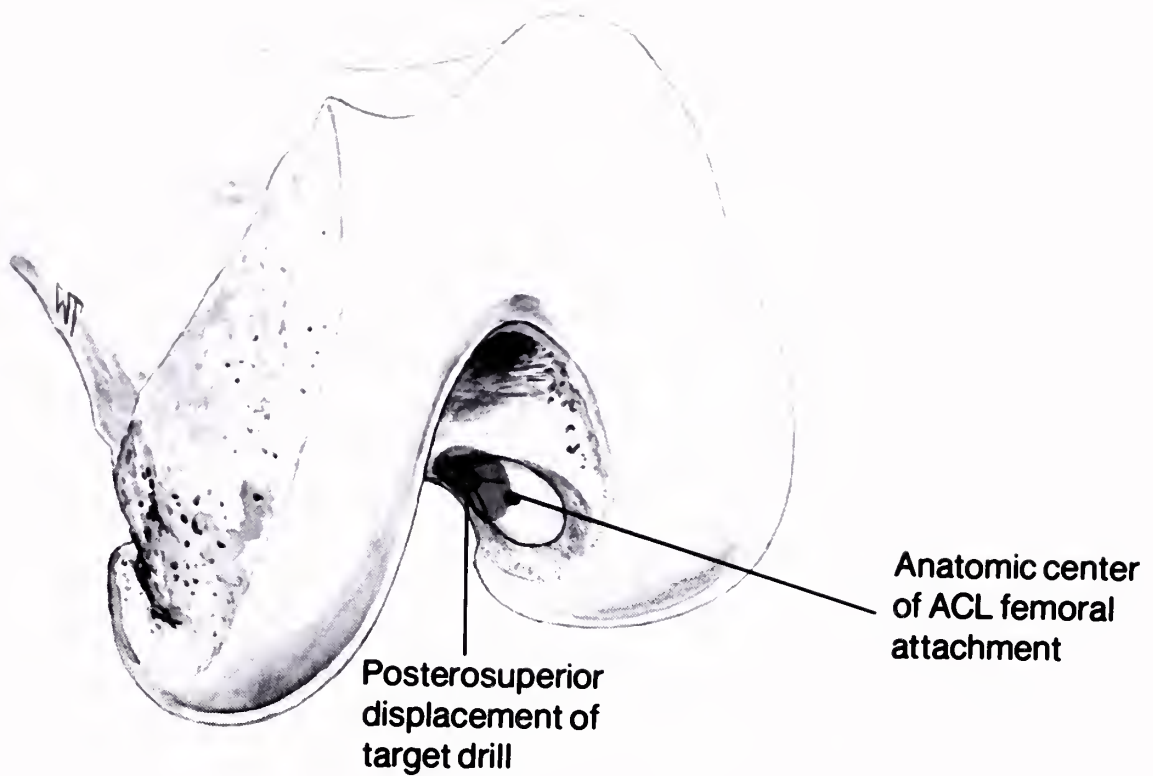
FIG. 7A. The suture and hook are visualized arthroscopically in the intercondylar notch. The tip of the femoral targeting hook is positioned superiorly and posteriorly in the notch, eccentric to the origin of the ACL, as described by Clancy.

FIG. 8A. An additional stab wound is created to accommodate the tip of the clamping device. The guide is clamped firmly in place around the lateral femoral condyle and stability is checked.
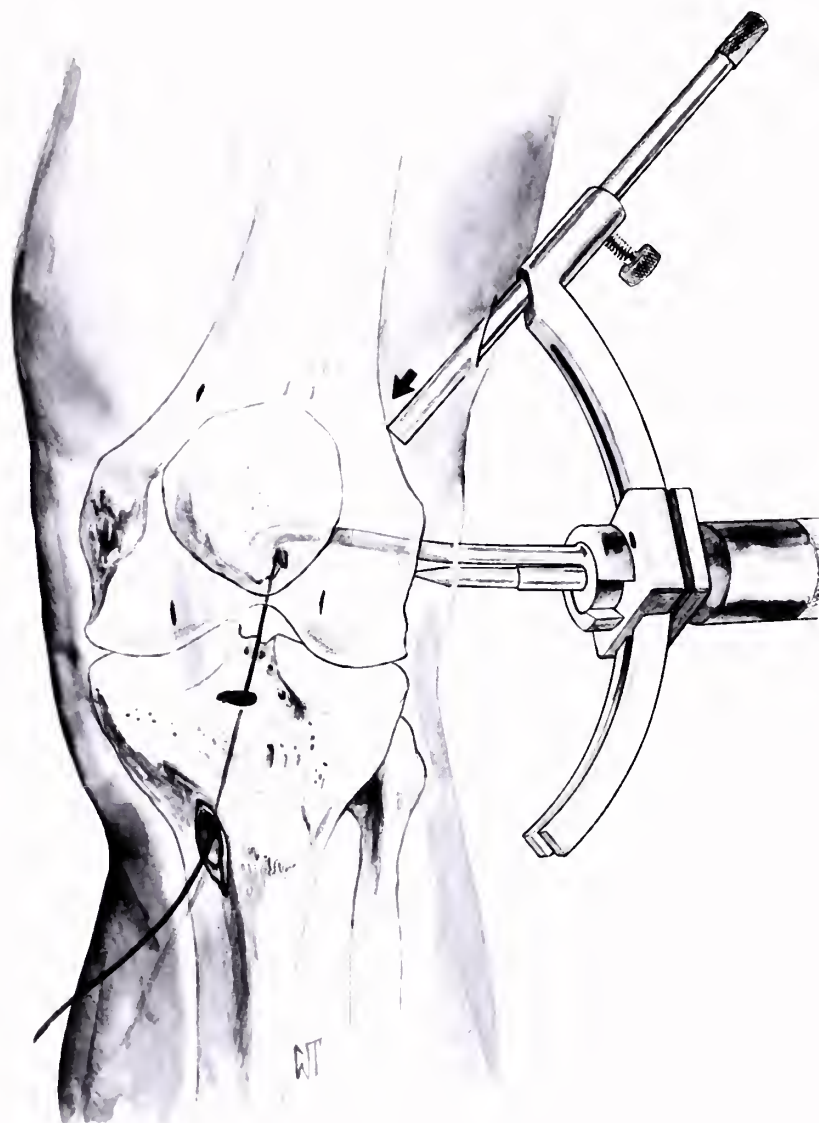
FIG. 9A. Positive excursion in flexion indicates that the targeting hook is placed too far anteriorly and must be repositioned more posteriorly. The ideal location for target drill placement is slightly posterior and superior to the isometric point, since overdrilling will position the tunnel edge more anterior and inferior. This position will be best demonstrated by positive excursion in extension only.

Based on ongoing clinical studies, 3 to 5 mm of total excursion from flexion to extension in a large individual, and 2 to 3 mm in a small individual is well tolerated, since overdrilling will position the femoral tunnel edge slightly anterior and inferior to the tip of the targeting hook(An alternative approach may be to position the femoral targeting hook in the notch until 0-2 mm of total excursion is obtained, and then reposition the hook slightly posterior and superior prior to drilling.)
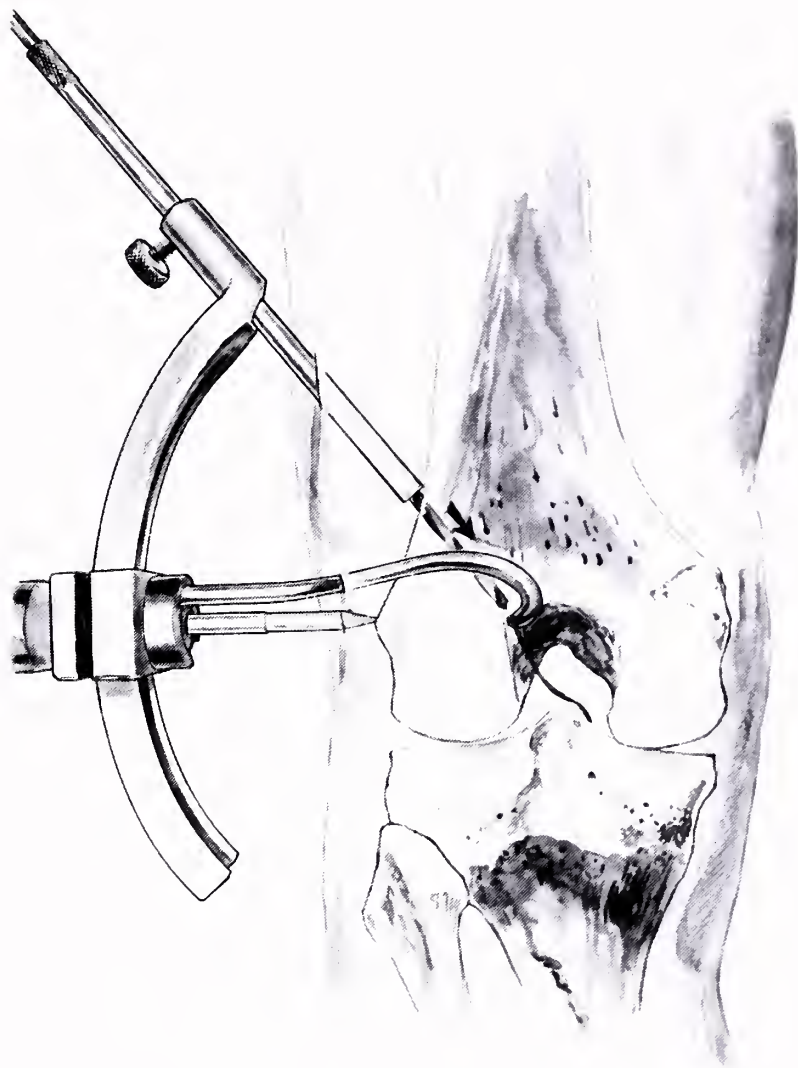
FIG. 10A. A target drill is inserted and drilled to the tip of the femoral targeting hook. Accurate targeting is confirmed arthroscopically.

The guide is disassembled and removed, leaving the target drill in place. The suture is cut from the tip of the targeting hook and left in the knee joint.

FIG. 11A. A tissue protector and cannulated reamer are then placed over the target drill and the femoral tunnel is drilled to the appropriate size.
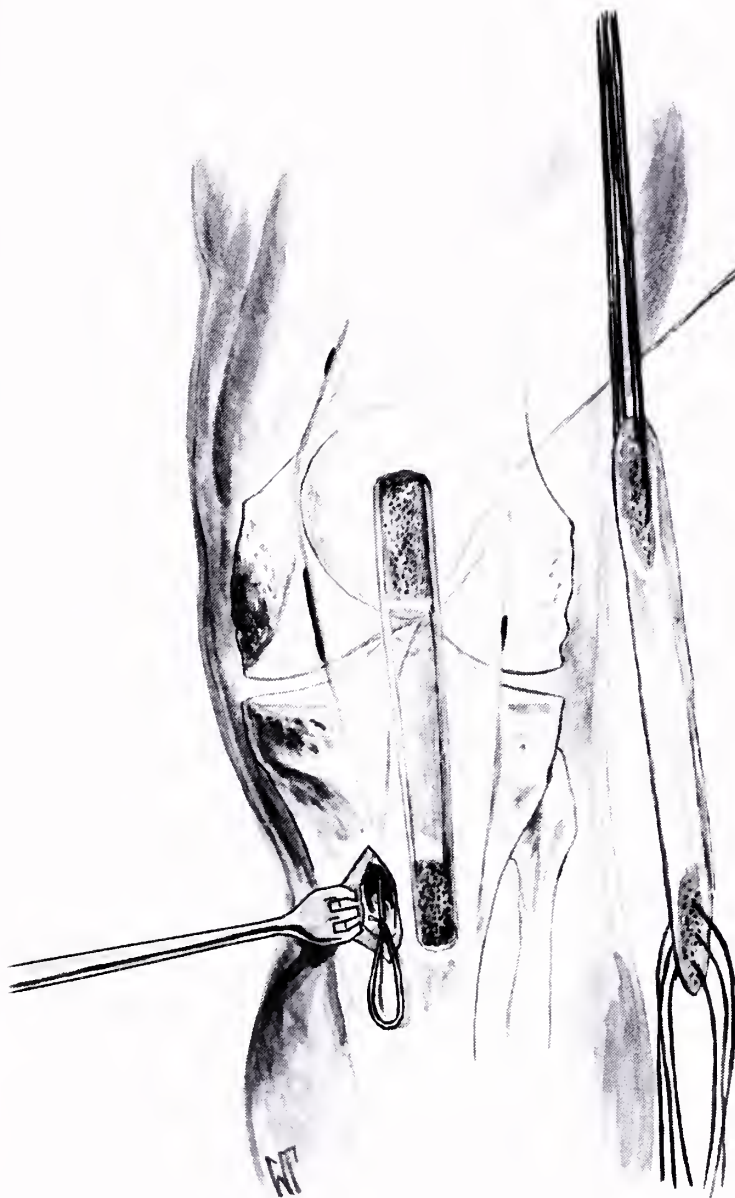
FIG. 12A. The graft fibers are carefully exposed. The patellar bone block is harvested with an oscillating saw and an osteotome. The bone block is trimmed, drilled, and threaded with three #5 ETHIBOND sutures.

⅛ inch target drill bit is more precise than K-wire

Posterior approach allows for accurate anatomic positioning and permits a full range of motion of the knee with the AI GUIDE clamped to the lateral femoral condyle

Isometric graft placement is determined prior to femoral tunnel drilling
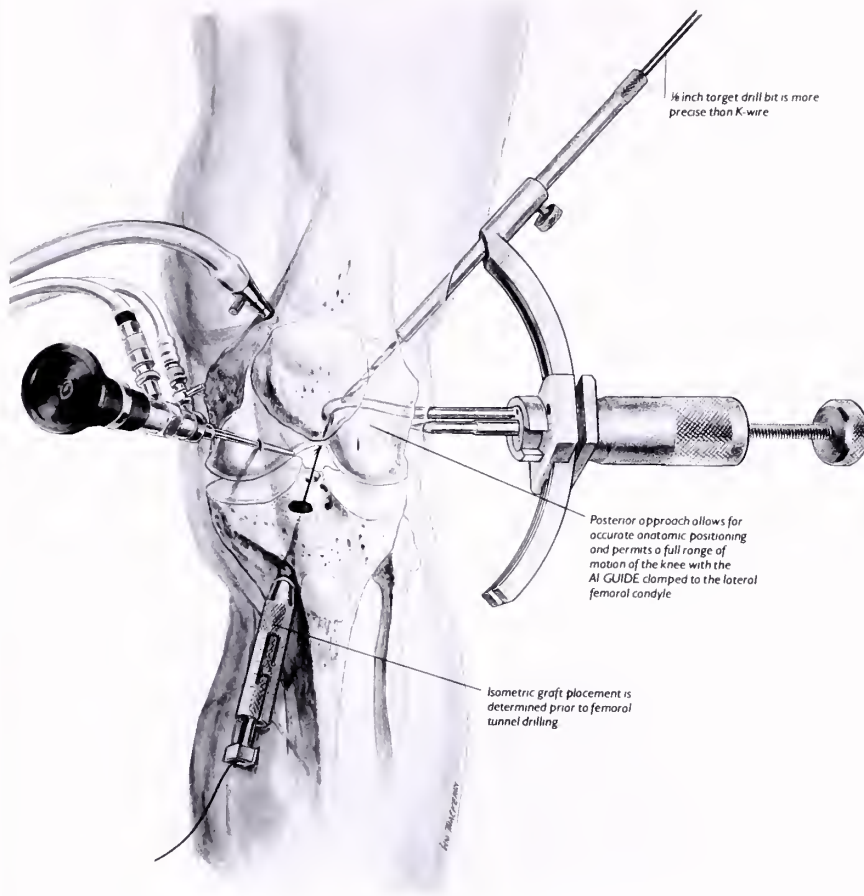
FIG. 13A. The AI Guide: Arthroscopic, Isometric ACL Reconstruction.

APPENDIX 2

CLINICAL ASSESSMENT FORM

ACL EVALUATION ONE PAGE FORM

IDENTIFICATION #:_____

    NAME_____

    AGE_____   SEX: M F   KNEE INVOLVED: RIGHT   LEFT

    SPORT CAUSING INJURY_____

    DATE OF INJURY _____

SYMPTOMS

    KNEE PAIN
            4 none 3 mild 2 moderate(with activity)

            1 severe (at rest & preventing activity)

    GIVING WAY
        8 none

        6 only with cutting (stop & turn) sports

        4 occassional(only with awkward step)

        2 with normal daily activities

    SWELLING
        4 none

        3 strenous activity

        2 with moderate activity

        1 with any activity

FUNCTION

    1 no sports   2 sports activities significantly limited

    3 active, but different sports   4 same sports different performance

    5 equal performance at same sports as before injury

Problem with Specific Activities:

	None	Mild	Moderate	Can't do
Walking				
Running				
Turn/cut				
Jumping				
Stairs				

APPENDIX 3

CHAPTER 8. KNEE JOINT STABILITY TESTS: PHILOSOPHICAL CONSIDERATIONS (from the Genucom Manual)

8.1. INTRODUCTION

Clinical interpretation of classical knee laxity tests has been limited by the lack of precise, accurate data from which the clinician can draw. For example, a 2 or 3 mm difference between the injured knee and the normal knee during an anterior drawer test may represent a positive sign for capsular tear or partial ACL tear. However, this subtle difference is left undetected by the subjective examination in which differences can only be seen when there are 5 or often significantly more millimeters difference between the injured and the uninjured knee. Another associated problem which frequently confuses the clinical examination is the inability of the examiner to precisely reproduce the conditions under which a test is done on the injured and the contralateral leg. For example, during a varus stress test with the knee flexed to approximately 30 degrees, it is extremely difficult to monitor precisely the amount of tibial rotation which occurs in conjunction with this test. Differences of 3 or 4 degrees in the amount of internal tibial rotation, for example, may have significant impact on the interpretation of the varus sign.

This chapter is provided not to have you re-learn concepts of classical laxity tests which you are already fully aware, but to highlight certain aspects of the performance of these tests and their interpretation which can now be discussed because of the reproducible, accurate objective data obtained by performing the tests on the GENUCOM. These discussions are by no means the last word on one or all these tests and will be continually updated as the data base for GENUCOM evaluations expands.

Whereas each test is described in detail in the following sections, all are summarized in one table entitled "Genucom Test Details".

8.2.1. ANTERIOR/POSTERIOR DRAWER TEST CONCEPTS

You may perform a Anterior/Posterior Drawer Test (A/P Test) at any flexion angle between 0 degrees and 90 degrees flexion. As you have seen in the test display, the flexion angle is continuously displayed in a prominent position, allowing you to monitor its value before and

throughout the test. The mean an deviation values for the flexion angle during the test are calculated after the test and form part of the data output.

Although several slightly different definitions of the anterior and posterior drawer test have been forwarded, (differing primarily in the flexion angle at which they are performed) it can be summarized generally as follows: the application, by hand, of a push (posterior) or pull (anterior) force in the vicinity of the proximal tibia and the evaluation of the resulting sliding of the tibia with respect to the femur. This test has been primarily used for the diagnosis of cruciate ligament injury. It is clear, however that at any flexion angle chosen there are soft tissue structure other than the cruciate ligaments which may become stressed by the drawer test. Because of this it is difficult already to precisely diagnose an anterior cruciate (ACL) injury or posterior cruciate ligament (PCL) injury. The GENUCOM improves the quality of your drawer test in a number of ways. For example:

1. You are not left to subjectively evaluate either the amount of force you are applying to the joint, or the resulting subluxation. The GENUCOM provides both to you in a continuous fashion during the test and then, if desired, as an instant plot of A/P force versus sliding at the end of the test.

2. Because the A/P Test is complicated enough by the presence of many soft tissue structures acting as restraints to movement, the GENUCOM removes the previously unknown problems of your applying unwanted compression force or tibial rotation by allowing you to prevent these forces and rotations from occurring during the test, if desired.

The latter are only two well known samples; it is very difficult to say what unwanted loads or movements were over-complicating your A/P Test diagnosis in the past. To use the internal tibial rotation case as an example, it has only recently been brought to light that during an Anterior Drawer Test at 30 degrees flexion angle, the tibia has a natural tendency to rotate internally*, thereby not allowing the ACL to be taut. With the GENUCOM you do not have to let this happen because you can, with your hand and the aid of the video display, sustain non-rotation of the tibia throughout the Drawer Test, thereby obtaining a more true measure of ACL and secondary structure viability.

To summarize the A/P Test background with regard to the GENUCOM, it is left to your discretion as to the exact type of Drawer Test you perform. By all means eliminate the effect of the hamstrings by going

to 15-30 degree flexion; by all means apply a distraction force to minimize interference

*Frank R. Noyes, Wolffe Memorial Lecture. ACSM 30th Annual May 1983.by intercondylar interference. You are free to do what you find necessary and most helpful to diagnose your patient. Just remember that to compare your results in future tests, you will need to know what you did today and the test results given to you by the GENUCOM will permit you to do just that. So be sure to choose an appropriate setting for each force and displacement component before embarking on the test.

8.2.2. INTERPRETING THE RESULTS

By summing the total A/P translation you have a true measure of the total A/P laxity of the joint. This can then be used to (1) compare with previous tests on the same leg, (2) compare with the uninjured contralateral leg of the same patient, (3) compare t a population norm for the same test, or to (4) retain for use in pre-and-post treatment comparisons. Although the bilateral comparison is acknowledged to be perhaps the most definitive indicator of disruption, this comparison is not always possible due to bilateral injury. For this reason FARO has established a program to compile data on normal knees from GENUCOM users, in an effort to establish a standard family of A/P Test plots based on test flexion angle and the amount and type of force application. Then in the absence of any other more indicative comparison, the population standards can be used as a basis to evaluate the extent of damage to the patient. Updated population norms for all GENUCOM tests will be sent to you.

The plot which is presented on the right side of each test result is that of internal and external (I/E) tibial rotation vs I/E tibial moment. The extent of tibial rotation which is allowed has been recognized as a key component in the evaluation of the A/P Test, as previously mentioned. Thus this plot, together with the corresponding mean and deviation values printed in the Table under I/E moment and I/E rotation, give you a permanent record of these factors and this allows you to more clearly evaluate the primary A/P translation versus A/P force results.

When an internal or external rotation is imposed on the tibia prior to an A/P Test the tibia may migrate slightly anteriorly or posteriorly. In interpreting the anterior/posterior laxity seen in this test it may be useful to subtract out the initial migration due to rotation in order to compare the force anterior/posterior laxity with that seen in the neutral or unrotated tibial position. This removes questions concerning the reference position

of the test and concentrates on the ability of the knee to resist forces in the A/P directions.

In addition to the plotted data, the mean and standard deviation values for the remaining force, moment, translation, and rotation components are printed in the center block of data below the main plots. Evaluating these data allows you to check other desired or undesired effects on your test. For example, you may consider a compression force on the joint greater than 3 lb over the entire test as unacceptable when examining the ACL, or that at any point a peak of 3 lb is unacceptable. You can check both these values in the 'mean' and 'sdev' columns of C/D force and thereby decide whether to accept the test.

8.3. DUAL ANTERIOR/POSTERIOR DRAWER TEST

8.3.1. DUAL ANTERIOR/POSTERIOR DRAWER TEST CONCEPTS

The Dual Anterior/Posterior Drawer Test (Dual A/P Test) was developed in order to provide a detailed picture of the difference in anterior and posterior displacement of the centers of the medial and lateral tibial plateaus, independently. It has been suggested in the past by numerous researchers that excessive displacement of a particular tibial plateau is indicative of rotatory instability. The GENUCOM digitization procedure provides a detailed three dimensional picture of the knee which allows it to calculate the centers of both the medial and lateral tibial plateaus. These two points are followed simultaneously by the GENUCOM during the application of the anterior and posterior forces. In this manner the stabilizers in the anterior and posterior portions of the medial and lateral compartments can be examined.

8.3.2. INTERPRETING THE RESULTS

The Dual A/P Test results are presented in a manner similar to the A/P Test except that the anterior and posterior translation are plotted versus the anterior and posterior forces for both the medial and lateral side. The medial side is presented on the left hand graph, for the right or the left leg. The right hand graph presents the A/P displacement versus A/P forces for the lateral side of the knee.

The interpretation of the Dual A/P Test involves both bilateral comparison and comparison to the population norms of the results for both the medial and lateral plateaus. Excessive displacement for the various loads can indicate disruption of various structures depending on the flexion

angle at which the test was performed. Refer to the "Genucom Test Details" table in section 8.10, for a more detailed listing of these structures.

8.4. VARUS/VALGUS STRESS TEST

8.4.1. VARUS/VALGUS STRESS TEST CONCEPTS

Like the A/P Test, the Varus/Valgus Stress Test (V/V Test) has traditionally been performed at different flexion angles. At full knee extension it has been noted that the hamstring muscles and the posterior capsule over compensate for damaged collateral ligaments in resisting V/V moments. At 90 degrees flexion, on the other hand, the femur is often said to rotate to such an extent, under a V/V moment applied to the tibia, as to falsely indicate a positive V/V Test in either direction. Whatever your preference in terms of flexion angle or any other of the force, or displacement components, the basic philosophy behind the V/V test remains as follows:

The application of a moment to the tibia about the A/P axis forcing the tibia to swing toward (Varus) or away (Valgus) from the midline of the body, and the measurement of the ensuing opening (or angulation of the tibia) of the joint on each side. An abnormally large opening or angulation would indicate a positive test and soft tissue damage.

With the GENUCOM you remain free to perform the V/V test in any way comfortable to you given the position of the patient on the chair. Although the GENUCOM will present you with an objective measure of the V/V angulation as a function of the V/V moment, you remain free to palpate the lateral and medial aspects of the joint line as you may be accustomed, to confirm in your own mind the results which will be given.

8.4.2. INTERPRETING THE RESULTS

Of the displacement components available from the V/V Test, the V/V rotation is the primary indicator of excessive joint space opening and hence damaged soft tissue. For this reason the first plot for the V/V Test is that of V/V rotation versus the applied V/V moment. Analogous to the A/P translation in the A/P Test, then, the extent of its V/V rotation for a particular flexion angle and applied moment can be used as the basis for your diagnosis when compared to the contralateral knee or the population norm for the test with similar conditions.

To evaluate the contribution of the anteromedial/lateral or posteromedial/lateral structures to resisting V/V moment (in addition to the

strictly medial and lateral structures), you will want to examine the extent of axial rotation of the tibia before and/or during your V/V Test. For this reason the second plot on the V/V TEst results output is I/E rotation versus I/E moment. During your varus/valgus test the Genucom monitors the I/E rotation of the tibia and if during the varus/valgus angulation the tibial axial rotation (I/E) exceeds 5 degrees from the initially set reference value in either the internal or external direction a warning is given following the test telling you that this rotation has occurred. You have the option then of not saving that test and returning and repeating the varus/valgus stress test or overriding the warning and saving the test with the internal/external rotation flag with it. Note: although V/V angle is plotted versus V/V moment in this test, the numbers placed in the corners of the left-side plot represents millimeters of joint opening rather than angle. This two-phased V/V data presentation allows the user to interpret the results within two different but classical frameworks.

The joint opening is calculated by multiplying the sine of the V/V angle by the distance between the center of the joint and the medial or lateral extremities of the tibia, as measured during the digitization procedure In addition to these two plots you will, as in all test, have the table of mean and deviation values for each force and displacement component to scan and to ensure that the test was performed as desired.

8.5. I/E ROTATION STRESS TEST

8.5.1. I/E ROTATION STRESS TEST CONCEPTS

Performed in conjunction with a compression force on the tibia, the I/E stress test performed at approximately 90 degrees flexion has been known as the Apley compression test. Performed with a distraction force at the same flexion angle it has been known as the Apley distraction test. The former is generally considered to be a test for the meniscii and the latter a test for the collateral ligaments. It is evident that in either of these 2 cases, as well as the case without compression or distraction applied to the joint, that structures other than those mentioned will also be involved in resisting internal or external stress. This point has been amply noted in previous tests involving internal/external stress, in combination with other loads.

8.5.2. INTERPRETING THE RESULTS

Whereas the A/P and the V/V Tests have been discussed at length in the clinical literature, often backed up by operative findings and cadaver studies, the I/E stress test has not received comparable attention. It may

appear that the medial and lateral capsular structures are the first implicated by rotatory stress, followed by the collateral ligaments and the cruciate ligaments, particularly the anterior cruciate during internal rotation. Clearly the meniscii will also play a role in resisting internal and external rotation. On the right side of the graph I/E moment is plotted versus I/E rotation, while on the left side of the plot anterior/posterior translation is plotted versus I/E rotation. The former plot is self-explanatory while the intent of the latter plot, that of the anterior/posterior translation versus rotation, is thought to be a measure of the individual compartment's contribution to rotatory instability in the knee. For example, it has been found in this test that in the normal knee the tibia maintains a centered position during the rotation, that is, there is little anterior or posterior translation of the tibia during the 20 to 30 degree rotation both internally and externally of the tibia. In internal rotation in particular there is very little anterior/posterior translation while in external rotation there maybe 2 to 3 millimeters anterior translation. In the injured knee however, the tibia tends to translate towards a quadrant with less support than in the normal knee. For this reason the 4 quadrants of that plot have been labeled ALRI, AMRI, PMRI and PLRI in conjunction with the instability related to tibial translation into that quadrant.

For example, anterior translation of the tibia beyond that of the normal knee in external rotation suggests anterolateral rotatory instability because of lack of resistance to the anterior translation during external rotation given by the anterolateral quadrant. Posterolateral rotatory instability is indicated as the tibia tends to translate posteriorly on an internal rotation due to lack of support in the posterolateral quadrant.

The numerical summary in the top left and lower right corners of the right side plot indicates the degrees of rotation of the tibia in the external and internal directions respectively for a 6 foot-pound moment in each direction. On the left hand plot the upper left hand corner numerical summary represents the degrees external rotation, while the lower right hand corner represents the A/P translation at 15 degree of internal rotation.

As previously mentioned, the AMRI, ALRI, PMRI and PLRI headings have been added in the 4 quadrants of the left side plot on the I/E stress test output. These are placed such that if the tibia translates (A/P) abnormally (versus the normal knee) into that quadrant, then the instability listed in that quadrant is indicated.

8.6. GENU RECURVATUM/SCREW HOME TEST

8.6.1. GENU RECURVATUM/SCREW HOME TEST CONCEPTS

Genu recurvatum in the acute patient has been associated with tears of the anterior cruciate ligament, and in the chronic patient with tears to the posterior cruciate ligament. The screw home phenomenon in the normal knee is a well recognized function. These two concepts have been joined together in a single test, the Genu Recurvatum/Screw Home Test, on the GENUCOM.

The patient begins with the knee at 90 degrees of flexion, and is asked to extend his leg to maximum extension, at which time the examiner then supports the leg behind the foot and the patient is asked to relax. The examiner then forces the leg into hyperextension to a predetermined load level and the recurvatum is measured. Although the screw home measured during extension by the patient is certainly not under weight bearing conditions, there are clearly discernible features in the pattern of internal and external rotation of the tibia which occurs during this extension.

8.6.2. INTERPRETING THE RESULTS

The left side of the graph shows a plot of anterior/posterior force versus flexion angle while the right hand shows a graph of internal/external rotation versus flexion angle. The amount of recurvatum is determined on the left hand plot and this is the amount of extension beyond zero degrees which is allowed under a minimum anterior force of 21 lbs. This 21 lb level ensures that adequate force has been applied to produce any recurvatum. In the patient with a 'tight' normal knee, which may not extend to 0 degrees of flexion, the comparison of course should be with the normal knee rather than with the 0 degree line. The flexion angle associated with the 21 lb anterior force is posted in the top left corner of the left plot.

The right hand plot shows the gradual internal rotation of the tibial from approximately 90 degrees to approximately 20 to 30 degrees, followed by an external rotation to the limit of extension. Early results show that the degree of internal rotation is significantly more for female normal knees than male normals. The clinical significance of this test clearly involves active structures as well as the passive ligamentous structures, and will be determined only after significant additional testing is performed. The Genucom computes the screw home angle as the difference between the maximum internal rotation and the tibial rotation

angle at the most extended position (before the examiner applied force). This angle is posted in the top left corner of the right side plot.

8.7. PIVOT SHIFT TESTS

8.7.1. PIVOT SHIFT TEST CONCEPTS

On the main menu for the classical laxity tests the medial and lateral pivot shift tests are designated as two separate entities. The rationale behind each is the same. Having the ability to look at the medial and lateral tibial plateaus allows us to examine the lateral and medial stability of the knee independently. This forms the basis for the medial and lateral pivot shift tests as performed on the Genucom.

Traditionally, the pivot shift test or more commonly the lateral pivot shift test involves an attempt to dislocating the lateral tibial plateau with respect to the femoral condyle and then eliciting a reduction of the joint due to flexion of the knee by flexing the knee toward 20 to 30 degrees of flexion. The focus of the pivot shift test in the Genucom is to start from the relatively normal configuration of the knee at some flexion angle between 20 and 30 degrees and then, in the case of the lateral tibial plateau, applying an anterior force as in the Lachman test, followed by an internal rotation thereby causing the tibial plateau to come forward and rotate out of its normal configuration with respect to the femoral condyle. Subsequently this force and rotation are released and a posterior force is applied followed by an external rotation, checking subluxation of the lateral tibial plateau in the posterior compartment. In this way a complete measure of the lateral stability of the lateral tibial plateau is objectively evaluated in the same region as the classical pivot shift test would be performed.

Similarly on the medial tibial plateau antero- and posteromedial instability can be evaluated by the application first of an anterior force followed by an external rotation and a posterior force followed by an internal rotation while looking at the medial tibial plateau.

8.7.2. INTERPRETING THE RESULTS

For both the medial and lateral pivot shift tests the tibial plateau of concern is shown in terms of its straight A/P laxity on the right side plot and its rotatory laxity following an A/P translation on the left side plot. Assuming that the data for the Lachman has already been quantified then the focus on this test usually is placed on the rotatory instability of the knee

as represented by anterior/posterior translation of the tibial plateau with rotation above and beyond that due to the straight Lachman test.

The number which appears on the top left corner of the left side plot represents the anterior translation of the tibial plateau of concern due the combined force and rotation. Similarly the number in the lower right corner of the left side plot represents the posterior translation of the plateau resulting from the posterior force and the rotation. The A/P translation is calculated at 15 degrees of rotation in either direction or 3/5 of the I/E rotation scale maximum.

Hence the pivot shift test for either the medial or lateral side represents the total subluxation of the plateaus while separating the straight instability from the rotatory instability thus allowing the examiner to establish the relative contributions of these two instability types.

8.8. FUNCTIONAL MODE

The GENUCOM makes provisions for the performance of the classical laxity tests in a functional mode. This allows the tests to be performed while the patient actively resists the applied stress. Although the stresses which are applied to the knee in this mode are significantly higher than in the passive mode, they are not of the same scale as the physiological forces seen during walking or running. However, they do provide a measure of the patient's ability to stabilize his knee in directions known to be excessively lax due to ligamentous damages. It has been shown, for example, that muscles used in internal/external rotation of the lower leg can be effectively evaluated in that mode at 90 degrees flexion.*

GENUCOM results have shown that patients exhibiting excessive laxity during the I/E Stress Test can reduce the difference in laxity from the injured to the normal knee to zero during a functional test, in the case of athletically active patients, and to approximate one half of the passive laxity difference in the non-athletically active patient. Clearly there is a definitive measurable effect of these structures during a functional I/E stress on the GENUCOM. This represents only one example of clinically significant functional testing on the GENUCOM.

The protocol for the functional testing requires one significant change from the passive protocol and that is in the area of soft tissue compensation. Recall from Sec. 2.2.4. that the soft tissue compensation requires the examiner to apply forces to the distal femur in such a manner as to teach the GENUCOM how much femoral motion occurs within the thigh and the thigh restraint mechanism on the GENUCOM. Subsequently

during the actual laxity evaluations this femoral motion is subtracted from combined tibo/femoral motion by the GENUCOM to give the true tibial femoral relatively displacement. Because the soft tissue around the femur is of significantly different stiffness when the musculature is actin, there is a corresponding change in the motion of the femur inside this soft tissue.

For these reasons it is necessary that if functional testing is to be performed, then soft tissue compensation must be performed with the thigh muscles tense. Once the patient has tensed the thigh muscles then the soft tissue compensation can proceed as indicated in Section 2.2.4.

Following soft tissue compensation the patient may relax until the time that the actual laxity test has begun, that is just before the examiner presses the right foot switch to gather data. At that time, the examiner should indicate to the patient to tense the thigh muscles.

As more and more functional laxity tests are suggested, the functional testing protocol will expand quickly and extensively, and for this reason FARO will constantly update this section of your operator's manual with new information as it become available.

*Osternig, L.R. et al., Relationships Between Tibial Rotary Torque and knee Flexion/Extension after Tendon transplant Surgery, Arch. Phys. Med. Rehabil., Vol. 62, 381-5, 1981.

8.9. GENUCOM TEST DETAILS SUMMARY TABLE

GENUCOM TEST DETAILS			
TEST NAME	**TECHNIQUE**	**INSTABILITY TYPE**	**STRUCTURES INVOLVED** (see numbered list below)
A/P Drawer	Straight A/P force applied by hand	Straight A/P	Anterior 8 9 3 17, 1 Posterior 6, 11, 5
A/P Drawer with Internal Rotation	Minimum -10° internal tibial rotation, followed by straight A/P force	Anterior: anterolateral, posterior Posterior: posteromedial, straight posterior	Anterior 17, 5, 18 1 Posterior 6-10, 5
A/P Drawer with External Rotation	Minimum + 10° external tibial rotation, followed by straight A/P force	Anterior: anteromedial, straight anterior Posterior: posterolateral	Anterior 3, 1, 4 Posterior 11-16
Dual A/P Drawer	Straight A/P force applied by hand	Medial/lateral plateau imbalance	Anterior, medial 3, 8, 9, 4, 1 lateral 17, 15, 16, 18, 1 Posterior, medial 6-10, 5 / lateral 11-16
Lachman (30° A/P Drawer)	Straight A/P force applied by hand	Straight A/P	Anterior 1, 2, 17 Posterior 5, 6, 11, 13
Dual Lachman	Straight A/P force applied by hand	Medial/lateral plateau imbalance	Anterior, medial 1, 3, 4 lateral, 1, 17, 18 Posterior, medial 5-7, 10 / lateral 5 11-16
Varus/Valgus Stress (0° - 10°)	Varus/valgus stress applied while restricting tibial and femoral axial rotation	Varus: posterolateral, ACL Valgus: posteromedial, ACL	Varus 11-14, 5, 1 Valgus 6, 7, 10, 5, 1
Varus/Valgus Stress (20° - 30°)	Varus/valgus stress applied while restricting tibial and femoral axial rotation	Varus: lateral Valgus: medial	Varus 17, 18, 13, 1 Valgus 3, 4
I/E Rotation Stress (80°)	Forced internal tibial rotation, release, forced external rotation	Internal: anterolateral, posteromedial External: anteromedial, posterolateral	Internal 17, 18, 5-7, 10 External 3, 4, 1, 8-10, 11-14
I/E Rotation Stress (20°)	Forced internal tibial rotation, release, forced external rotation	Internal: anterolateral, posteromedial External: anteromedial, posterolateral	Internal 11-17, 5, 6 External 3, 4, 1, 10, 11-14
Medial Pivot Shift	Straight anterior force then external rotation, release, straight posterior force then internal rotation	Anterior: anteromedial rotatory, anterior Posterior: posteromedial rotatory, posterior	Anterior 1-4 Posterior 5-10
Lateral Pivot Shift	Straight anterior force then internal rotation, release, straight posterior force then external rotation	Anterior: anterolateral rotatory, anterior Posterior: posterolateral rotatory, posterior	Anterior 1, 17-19 Posterior 11-16, 5
Genu Recurvatum/ Screw Home	Active leg extension by patient, relax, anterior force at heel by examiner to elicit hyperextension	Recurvatum: hyperextension Screw Home: active rotatory instability	Recurvatum 1, 5-7, 11-14 Screw Home 1
Axial Alignment	3D digitization of tibial crest, medial/lateral knee joint line, and anterosuperior iliac spine	Physiological knee valgus	Varus/valgus deviation
Hypermobility	Straight medial/ lateral force exerted to both sides of patella	Medial/lateral	Passive 21-25 Active 20-25
Q-angle	3D digitization of tibial crest, medial/lateral patella and anterosuperior iliac spine	Quadriceps/patellar misalignment	20-25
Patellar tracking	Active flexion/extension by patient with tracking attachment on patella	Patellar tracking	20, 21, 24, 25, 21, 22

1 Anterior Cruciate	8 Post Medial Menisco/Tib	15 Post Lateral Menisco/Tib	22 Patellofemoral Lig
2 Ant Medial Meniscus	9 Post Medial Menisco/Fem	16 Post Lateral Menisco/Fem	23 Patellotibial Lig
3 Mid 1/3 Medial Capsule	10 Oblique Popliteal	17 Mid 1/3 Lateral Capsule	24 Medial Fem Condyle
4 Medial Collateral	11 Arcuate	18 Iliotibial Band	25 Lateral Fem Condyle
5 Posterior Cruciate	12 Biceps Fem Tendon	19 Ant Lateral Meniscus	
6 Medial Capsule — Post Oblique	13 Lateral Collateral	20 Quadriceps Group	
7 Semimembranosus	14 Popliteal Tendon	21 Patellar Tendon	

8.10. REFERENCES

The data presented in the test details in Section 8.10. is based on the information contained in many articles written in the journals of orthopedic surgery. A comprehensive list of references regarding knee examination can be found in the GENUCOM Knee Analysis System Bibliography of Knee Joint Examination and Related Topics. Among the major contributors to the classification of knee injuries on the basis of the classical stability tests, and whose results are used in Table 8.10 are:

1.   Medical and Anterior Instability of the Knee, An Anatomical and Clinical Study Using Stress Machines, John C. Kennedy, Peter J. Fowler, The Journal of Bone and Joint Surgery, Vol. 53-A, No. 7, October 1971, pp. 1257-70.

2.   Pivot Shift: A Clinical Sign of Symptomatic Anterior Cruciate Insufficiency R.D. Galway, A. Beaupre, D.L. MacIntosh, The Journal of Bone and Joint Surgery, Vol. 54-B, No. 4, 1972, pp. 763-4.

3.   Clinical Diagnosis of Anterior Cruciate ligament Instability in the Athlete, Joseph S. Torg, Wayne Conrad, Vickie Kalen, The American Journal of Sports medicine, Vol. 4, No. 2, March/April 1976, pp. 84-93.

4.   Classification of Knee Ligament Instabilities, Part I. The Medial Compartment and Cruciate Ligaments, Jack C. Hughston, James R. Andrews, Mervyn J. Cross, Arnaldo Moschi, The Journal of Bone and Joint Surgery, Vol. 58-A, No. 2, March 1976, pp. 159-72.

5.   Classification of Knee Ligament Instabilities, Part II. The Lateral Compartment, Jack C. Hughston, James R. Andrews, Mervyn J. Cross,

Arnoldo Moschi, The Journal of Bone and Joint Surgery, Vol. 58-A, No. 2, March 1976, pp. 173-9.

6.   Ligamentous Restraints to Anterior Posterior Drawer in the Human knee, David L. butler, Frank R. Noyes, Edward S. Grood, The Journal of Bone and Joint Surgery, Vol. 62-A, No. 2, March 1980, pp. 259-70.

7.   Clinical Laxity Tests and Functional Stability of the Knee: Biomechanical Concepts, Frank R. Noyes, Edward S. Grood, David S. Butler, Mehrdad Malek, Clinical Orthopedics and Related Research, No. 146, January/February 1980, pp. 84-9.

8.   The Posterolateral Drawer Test and External Rotational Test for Posterolateral Rotatory Instability of the Knee, Jack C. Hughston, Lyle A. Norwood, Clinical Orthopaedics and Related Research, 1980, pp. 82-7.

9.   Acute Straight Lateral Instability of the Knee, Jesse C. DeLee, Marke B. Riley, Charles A. Rockwood, Jr., The American Journal of Sports Medicine, Vol. II, No. 6, 1983, pp. 404-11.

10.   Physical Examination in the Diagnosis of Rotatory Instability, Robert L. Larson, Clinical Orthopaedics and Related Research, January/February 1983, pp. 38-43.

8.11. LIMITATION OF LIABILITY

The following is an excerpt from the "Purchase Conditions," attached to your order form:

FARO shall not be responsible under any circumstances for special, incidental or consequential damages, including but not limited to, injury to or death of any patient, operator, or other person, damage or loss resulting from inability to use the System, increased operating costs, loss of production, loss of anticipated profits, damage to property, or other special, incidental or consequential damages of any nature arising from any cause whatsoever whether based in contract, tort (including negligence, or any other theory of law, consists of the obligation to repair or replace defective components in the System subject to the limitations set out above in this section.

This disclaimer of liability for consequential damage extends to any such special, incidental or consequential damages which may be suffered by third parties, either caused directly or indirectly resulting from test results or data produced by the System or any component thereof and the Purchaser agrees to indemnify and save FARO harmless from any such claims made by third parties.

APPENDIX 4
IV. CONSENT FOR PARTICIPATION IN A RESEARCH PROJECT

YALE UNIVERSITY SCHOOL OF MEDICINE - YALE NEW HAVEN HOSPITAL

Invitation to Participate and Description of Project:

You are invited to participate in a study of the diagnostic value of magnetic resonance imaging (MRI). You have been chosen for this study because you (1) have a condition which we and your doctor think might be evaluated better, or more safely, or both, by MRI than by other available methods of (2) you are participating in a study of the diagnostic value and capability of MRI to demonstrate normal anatomy.

The MRI is done as follows: You will lie on a firm plastic table inside a doughnut-shaped magnet. You will be asked to lie as still as possible. Neither you nor the magnet will move. You will feel no unusual sensations, although the magnet is noisy during the examination. The technologist and doctor operating the scanner will be able to see you, and you and they can maintain contact by voice as well. The total time for the examination will be about one hour.

In this study, MRI pictures will be made of one or more regions of your body, which will be selected based on your symptoms or the results of other tests which you have had. The MRI study is in addition to and will usually not replace other tests which you might have; it is possible, though, that the results of the MRI study might prompt your doctor to order additional tests. You will be asked to have MRI pictures on one or more occasions, depending on your health status, treatment, or test results.

MRI uses magnetism and radio waves to make pictures. If you have a pacemaker or some types of metallic implant, you may be excluded form the study due to possible effects of magnetic fields on the pacemaker or implant. Be sure to tell us if you know or think you have a pacemaker or metallic implant (such as an aneurysm clip, heart valve, etc.)

Except for pacemakers and some types of metallic implants (other types appear to pose no hazard), we know of no health hazard from the MRI examination. You will fill out the attached safety questionnaire to make sure there is no hazard to you from metallic implants. It is possible that MRI may have effects which we don't yet know about. Although thousands of people have been exposed to the magnetic and radio fields we do not have long term experience with the health effects of this technique. Even though we have no reason to suspect any adverse effects you should be aware that problems could occur in the future. Yale-New Haven Hospital cannot assume responsibility for paying for medical therapy of such effects. No painful procedures are involved, although lying on the scanning table may be slightly uncomfortable. WE know of no evidence whatever that your condition will get better or worse due to having the MRI examination. A few people may experience claustrophobia (being anxious from being in an enclosed place) when they are lying in the scanner. If you are prone to claustrophobia, let us know. If you feel anxious during the test, you will be able to tell us, and we will stop the test if you wish. Although we know of no fetal hazards from MRI, we have chosen not to examine pregnant women yet. Please tell us if you might be pregnant.

The MRI pictures might be of direct benefit to you in helping diagnose your condition or evaluate your response to treatment. If MRI is found to be useful for your condition or other conditions, it is possible that you or others might benefit in the future from this knowledge.

Neither you nor your insurance company will be charged for the MRI examination.

In all records from this study, your name will be available only to the doctors and researchers, and agents of the Food and Drug Administration and will not be used in any scientific reports of the study.

You are completely free to choose whether or not to participate in this study. If you do decide to participate, you may change your mind and withdraw at any time. If you decide not to participate, or if you withdraw, it will not affect your relationship with us, your doctors, or this hospital.

YALE-NEW HAVEN HOSPITAL MAGNETIC RESONANCE CENTER

PATIENT HISTORY AND SAFETY SCREENING

PLEASE CHECK IF YOU HAVE ANY OF THESE ITEMS:

THE FOLLOWING ITEMS MAY BE HAZARDOUS TO YOUR SAFETY

                                        YES   NO   DO NOT KNOW

1. Cardiac Pacemaker
2. Brain clip (Aneurysm clip)
3. Vascular filter, umbrella
4. Metal fragments-in the head, eye or skin
5. Shrapnel
6. Neurostimulators
7. Heart valve
8. Infusion pump
9. Electrodes
10. Cochlear or stapes implant
11. Any prosthesis or artificial implant
12. Shunt, spinal or ventricular

THE FOLLOWING ITEMS MAY INTERFERE WITH IMAGING

1. Coronary artery clips (bypass surgery)
2. Other surgical clips
3. Hearing aids
4. IUD
5. Hickman or Broviac catheters
6. Joint replacements
7. Metal rods, plates, pins, screws, nails
            or clips
8. Harrington rods
9. Wire sutures
10. Dentures
11. Eye make-up

- -

Previous operations?
When _____

Is there any possibility that you might be
  pregnant?
Do you work with metal (e.g. welder) or have
  a war or gun injury?

Signature of patient:

_____

Signature of parent or guardian:

_____

Date: _____

Please feel free to ask questions about anything related to this form or this study which you don't understand. Take as long as you need to decide whether or not to participate in this study.

Authorization: I have read this form and decided

that _____ will participate in the
                (name of subject)

project described above. Its general purposes, the particular of involvement and possible hazards and inconveniences have been explained to my satisfaction. My signature also indicates that I have received a copy of this consent form.

_____
                                    Signature

_____
                                    Relationship (self, parent,
guardian)

_____
                                    Date

_____
Signature of Principal Investigator   Telephone   _____

                        o r

_____
Signature of Person Obtaining Consent   Telephone   _____

If you have further questions about this project or your rights as a research subject or if you have a research related injury, please contact the principal investigator,

_____
at _____.
   (telephone)

THIS FORM IS NOT VALID UNLESS THE FOLLOWING BOX
HAS BEEN COMPLETED IN THE HIC OFFICE

THIS FORM IS VALID ONLY UNTIL

_____(date).

APPENDIX 5
MRI

Magnetic Resonance imaging utilizes the phenomenon of nuclear magnetic resonance of hydrogen photons (water and fat) when placed in the magnetic field and irradiated with radiofrequency at the Larmor frequency. This specific resonance frequency is $w = \gamma B_o$ where w^o is the Larmor frequency, γ the gyromagnetic ratio for the hydrogen and B_o the static magnetic field strength.

Spin echo imaging utilizes pulses of radio frequency to excite the protons to resonance (90 degree pulse) and refocuses the energy (180 degree pulse) to allow measurement of the tissue energy level at specific time points following the exciting pulse (90 degree pulse. Echo time (TE) is the time between the 90 degree pulse and the apearance of the echo. To obtain significant information to reconstruct the entire image, the specific pulse sequence is repeated 128, 192, or 256 times depending on the matrix size chosen. The time between the exciting pulses is the TR (repetition time). For "T1 weighted" sequences. TR is +/- 400-800 msecs, TE is 20 msecs and for "T2 weighted" sequences TR is 2000-3000 msec, and TE is 80-100 msecs for a typical sequence. The image obtained with the TR 2000 and TE 20 is a "balanced" or "proton density" image. Reconstruction is most commonly performed using two-dimensional Fourier transformation techniques. In Fourier imaging, phase- and frequency encoding gradients are applied to spatially encode the NMR signals. The NEX is the number of times the entire sequence is repeated to improve the signal to noise ratio of the acquired time signal intensity(S.I.) according to $S.I. = \sqrt{NEX}/2$. Saturation pulses are used to decrease artifacts due to blood flow during the time of imaging.

APPENDIX 6

ACL GRAFT FORM

Name: ....   **Date:**   L   R

ACL Graft   **# post-op days:** ....

Signal Intensity

- ▉ normal tendon
- ░ >tendon < or = musc
- ▨ > musc & < fluid
- ☐ fluid
- ▦ metal artifact
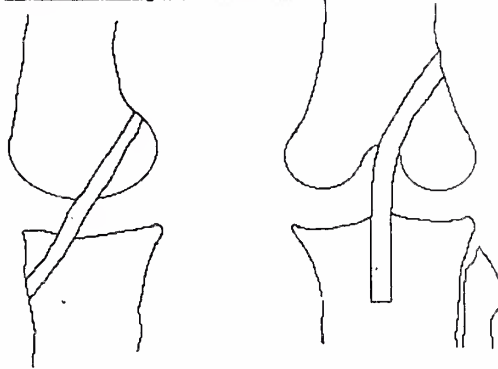
MENISCUS   Medial

....

Lateral

EFFUSION

trace

small

moderate

large

Measurements in mm.

a1
a2
a3
b
d

patella

c1
c2
c3
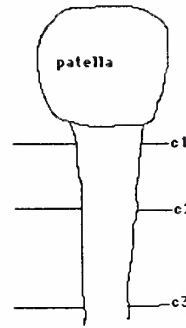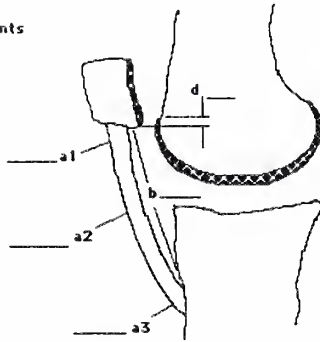
HYALINE CARTILAGE   Patello-femoral

....

Tibio-femoral

Comments: ....

APPENDIX 7

GRAFT STANDARD EXAMPLES
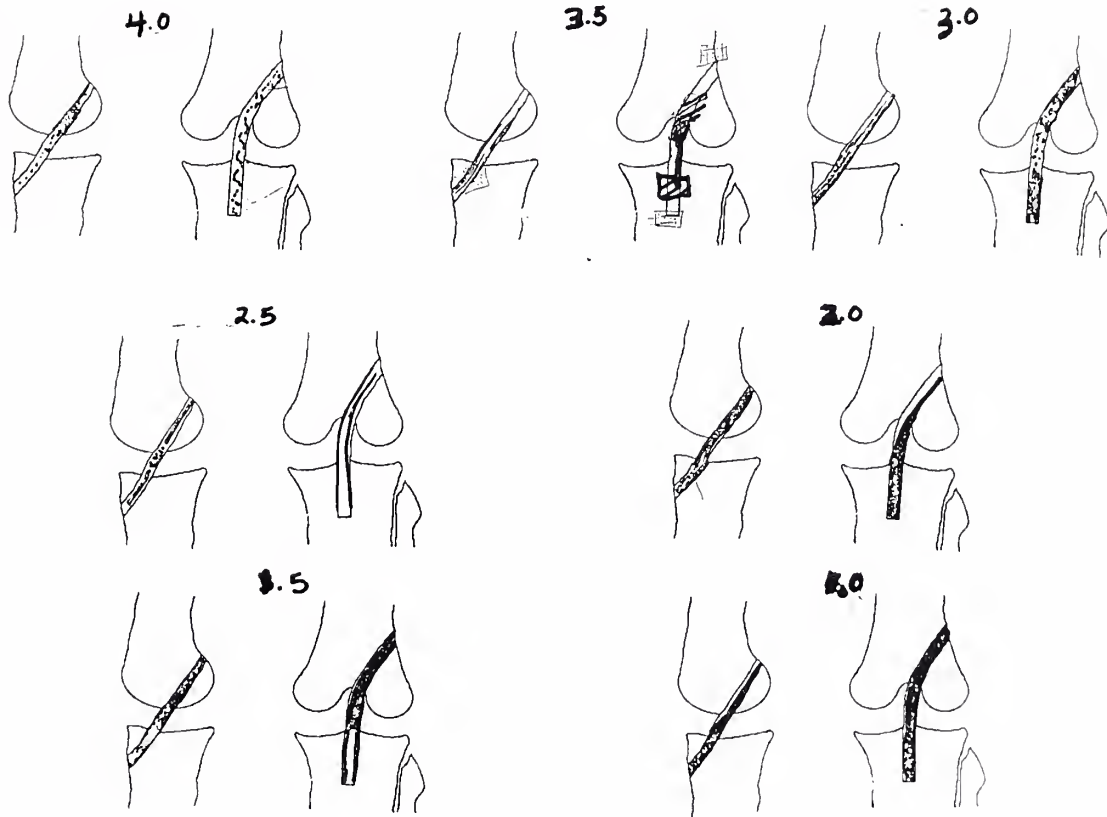
APPENDIX 8

STATISTICAL ANALYSIS

The data comparing changes over time were analyzed with a Repeated Measures Analysis of Variance (ANOVA). The p values indicate whether a statistically significant difference exists between any of the groups (eg. Time 1, Time 2, Time 3). If a significant difference does exist, then a "post-hoc" test was performed to determine which groups are different. The "post-hoc" test used in this study was the Fisher's Least Significant (LSD) tests. The specified significance level used to identify differences was 95%. The data comparing two groups to determine if a significant difference exists between them was analyzed using the paired T-test.

Repeated Measures Analysis of Variance(ANOVA)
This test is used to compare three or more groups of numeric data. This test computes the mean and standard deviation for each group, then uses a formuls to determine the p value. When the p value is less than 0.05 the null hypothesis can be rejected at a 95% confidence level.

Fisher's Least Significant Difference Test
The least significant difference (LSD) procedure is a Student's t test using a pooled error variance, is valid only when making independent comparisons or comparisons planned before the data are analyzed. A difference between any two means that exceeds a least significant difference (LSD) is considered significant at the level of significance used in computing the LSD. The LSD procedure usually is used only when the overall analysis of variance leads to a significant variance.
     Post-hoc Tests
     Post-hoc tests are conducted by computing the appropriate univariate t ratio on the appropriate combination of the outcome measures, but the resulting value of such a post-hoc t is compared with that value of T(the square root of t^2) that would have just barely achieved statistical significance.

Paired T-Test
This test is used to compare two paired groups of numeric data. A p value is calculated with the null hypothesis being that the average difference is equal to zero. If the average difference is significantly different from zero, the null hypothesis is rejected and a statistically signinficant difference is said to exist.

APPENDIX 9
GRAPHS
(Refer to Table 3)
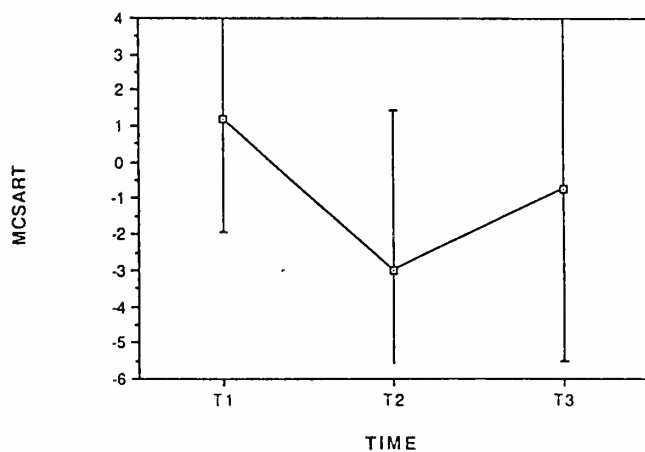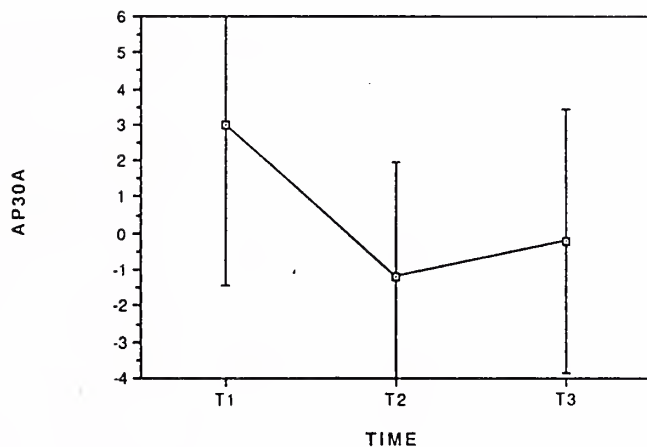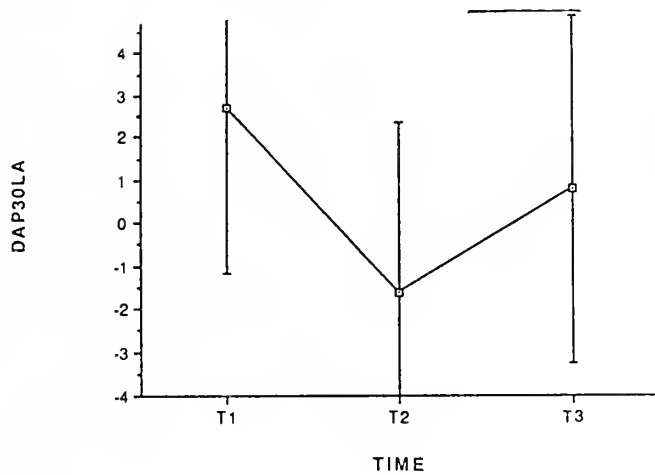
*DAP30LA = Dual anterior-posterior drawer at thirty degrees (lateral-anterior)
*AP30A = Anterior-posterior drawer at thirty degrees (anterior)
*MCSART = Medial compartment subluxation with anterior rotation

THEORY OF OPERATION

As the patient applies force to the arm of the KIN-COM, three signals are generated. These signals, FORCE, ANGLE, and VELOCITY are fed back to the computer for processing. In turn, the computer sends a signal back to the control circuitry to adjust the velocity, or force of the arm, depending on the type of treatment used.

The FORCE signal is generated in the loadcell, located on the actuator arm. During an exercise the patient applies force directly to the loadcell. A very small voltage is generated, which is representative of the amount of force applied, and is passed down to the PCB10. There, it is amplified and passed on to the interface box, located behind the computer. In the interface box, the FORCE signal is converted from an analoge, to a digital signal and is passed through COM1 to the computer. In the computer the signal is processed and displayed.

The VELOCITY signal is generated by a tachometer located inside the head of the KIN-COM. This signal passes through the PCB10 up to the interface box. VELOCITY, like the FORCE signal is converted to a digital signal and passed through COM1 to the computer to be processed and displayed.

With the FORCE and VELOCITY information, the computer can calculate the speed at which the arm is moving and the amount of force being applied by the patient.

The ANGLE information is generated by a servo pot also located in the head of the KIN-COM. This signal is passed up to the PCB10 and up to the interface box where it is converted to a digital signal. The signal is then passed, in digital form, through COM1 to the computer for processing.

With the ANGLE information, the computer detects the direction of travel, and the location angle of the actuator arm. This information is used to determine the selected start and stop angles and also for error sensing.

The computer processes the FORCE, VELOCITY, and ANGLE signals for display on the CRT. The information is also used to control the force and velocity of the actuator arm. This is done by controlling the hydraulic system in the KIN-COM. To control the hydraulic system, a servo valve is placed between the pump and the actuator. This valve is controlled by the amount of electric current flowing to it. The computer generates the servo control signal and passes it to the servo drive circuitry on the PCB10. There it is amplified and passe to the serve valve. Based on information gathered, the computer opens or closes this valve to change the force and/or velocity of the actuator arm. These changes are made 100 times per second to compensate for any changes in the force or velocity.

APPENDIX 11

GLOSSARY-BIOMECHANICAL TERMS

Abduction -- Motion away from the midline

Acceleration -- The change in velocity of a body divided by the time over which change occurs

Adduction -- Motion toward the midline

Agonistic muscles -- Muscles which initiate and carry out motion

Bending -- A loading mode in which a load is applied to a structure in a manner that causes it to bend about an axis, subjecting the structure to a combination of tension and compression

Brittleness -- The quality whereby a material exhibits little deformation before failure

Concentric work -- Work produced by a muscle when it is contracting and its length is shortening

Creep -- Progressive deformation of soft tissues due to constant low loading over an extended period of time

Degrees of freedom -- The number of ways in which a body can move

Direction of displacement -- The direction of change in position of the contact points of two surfaces

Eccentric work -- Work produced by a a muscle when its length is increasing

Elasticity -- Property of a material which allows the material to return to is original shape and size after being deformed

Extension -- The position of the joints of the extremities and back when one stands at rest, or the direction of motion which tends to restore this position; the opposite of flexion

Flexion -- Movement involving the bending of a joint whereby the angle between the bones is diminished; the opposite of extension

Load-deformation curve -- A curve which plots the deformation of a structure when the structure is loaded in a known direction

Range of motion -- The range of translation and rotation of a joint for each of its 6 degrees of freedom

Resiliency -- The capacity of a strained body to recover its size and shape after deformation

Rotation -- Motion in which all points describe circular arcs about an immovable line or axis

Screw-home mechanism -- A combination of knee extension and external rotation of the tibia

Shear -- A loading mode in which a load is applied parallel to the surface of the structure, causing internal angular deformation

Shear modulus -- An innate property of a deformable body that indicates how much resistance the body presents when an attempt is made to shear it, represented by the slope of the shear stress-strain curve; related to the modulus of elasticity

Shear strain -- The amount of angular deformation of a structure under shear loading (See strain)

Speed of loading -- The rate at which load is applied to a structure

Stiffness -- controls how much additional joint movement is required (after the ligament has become taut to create a force large enough for the ligament to resist the applied load

Strain -- Deformation (lengthening or shortening) of a body divided by its original length

Strain rate -- The speed at which a strain-producing load is applied

Stress -- Load per unit area which develops on a plan surface within a structure in response to externally applied loads

Tension -- A loading mode in which equal and opposite loads are applied away from the surface of a structure, resulting in lengthening and narrowing

Wear -- The removal of material from solid surfaces by mechanical action

APPENDIX 12

ACL PROTOCOL

ACL PROTOCOL

Intra-articular repair/reconstruction with high strength, rigidly fixated graft with no meniscal repair and no extra-articular repair.

The following are guidelines for rehabilitation of patients post ACL reconstruction. Normal variations will occur, but patients should be relatively close to these criteria within the given time.

TIME	BRACE ROM	GUIDELINES
Day 5 to week 2	$10-70^o$	Passive Range of Motion (PROM) Quad/ham co-contractions, prone Active Range of Motion (AROM) self-assisted sitting PROM, quad Electrical Muscle Stimulation (EMS) patella mobilization. Anti-inflammatory & edema reduction modalities as appropriate.
2 wks to 4 wks post-op	$0-90^o$	Toe touch weightbearing-1/4 weightbearing: Quad sets, A/AROM, Bilateral leg raises, Quad/hamstring co-contractions, calf exercises, gluteal sets, Upper Body Ergometer, upper cycle, upper body exercises sitting, EMS with SLR if tolerated, patella mobilizations, active and assisted PNF diagonals with brace, anti-inflammatory and edema reduction modalities as appropriate. Begin stationary bike partial ROM and progress; Eagle multi-hip; patella mobilization.
4 wks to 6 wks post-op	$0-100^o$	1/2-3/4 Weight Bearing: Quad sets, AROM, EMS with SLR, hip exercises with weight above knee; BLR to Single Leg Raise above the knee; PNF exercises with no quad resistance patella mobilizations, stress prone AROM to tolerance, sitting active extension to brace ROM only, continue anti-inflammatory edema reduction modalities as appropriate. Eagle multi-hip; Full revolution stationary bike when ready (no extension past 30). Add Eagle hamstring curls. Begin Eagle Leg Press or Total Gym to weight bearing level only. McConnell taping prn
6 wks to 8 wks post-op	Full ROM	Weight to tolerance: As above; add Eagle Hamstring curls, concept II rower for ROM only. Begin Eagle leg press to body weight. Begin prone KinCom isokinetic hamstrings as tolerated.
10 wks- 12 wks post-op tolerance.	Full ROM or consider	Weight to tolerance: As above; begin KinCom concentric/eccentric quads with proximal shin pad placement (avoid terminal 35° of extension) KinCom isokinetic leg press. Speed to tolerance. Hamstrings prone with speed to Nordic track when Full weight bearing.
12 wks- 16 wks	Full ROM	Full Body Weight: As above; Build in proprioceptive facilitation as tolerated; i.e., BAPS, vestibular board, etc. Stool-stepping for eccentrics (10-12 wks if Full weight bearing early).

TIME	BRACE ROM	GUIDELINES
16 wks- 24 wks post-op	—	As above; Progress to tolerance. May begin running straight ahead; job only with brace on level surface or treadmill.
24 wks- 28 wks post-op	—	As above; Increase to tolerance. Begin side to side drills drills with brace.
28 wks- 32 wks post-op	—	Add more aggressive running. Same exercises. KinCom test 6 months post-op
32 wks- 40 wks post-op	—	More running with brace. Add figure eights, springing, and continue strengthening exercises.
40 wks- 52 wks post-op	—	Progress back to full activities. KinCom test at 1 year post-op; strength should be within 80% of the uninvolved leg. Goal of 1:1 Ham./Quad ratio.

Patients should be progressed to tolerance and advanced as they are ready, provided graft precautions are observed at all times. Report significant deviations to the patient's physician.

Patients who also have a meniscal repair should be kept non-weight-bearing for 6 weeks or as specified by the patient's physician.

Revised 8/90: RJ/TP

BLR = Bent Leg Raises
ROM = Range of Motion
SLR = Straight Leg Raises
ex's = exercises
EMS = Electrical Muscle Stimulation
PNF = Proprioceptive Neuromuscular Facilitation

APPENDIX 13

MRI EXAMPLES
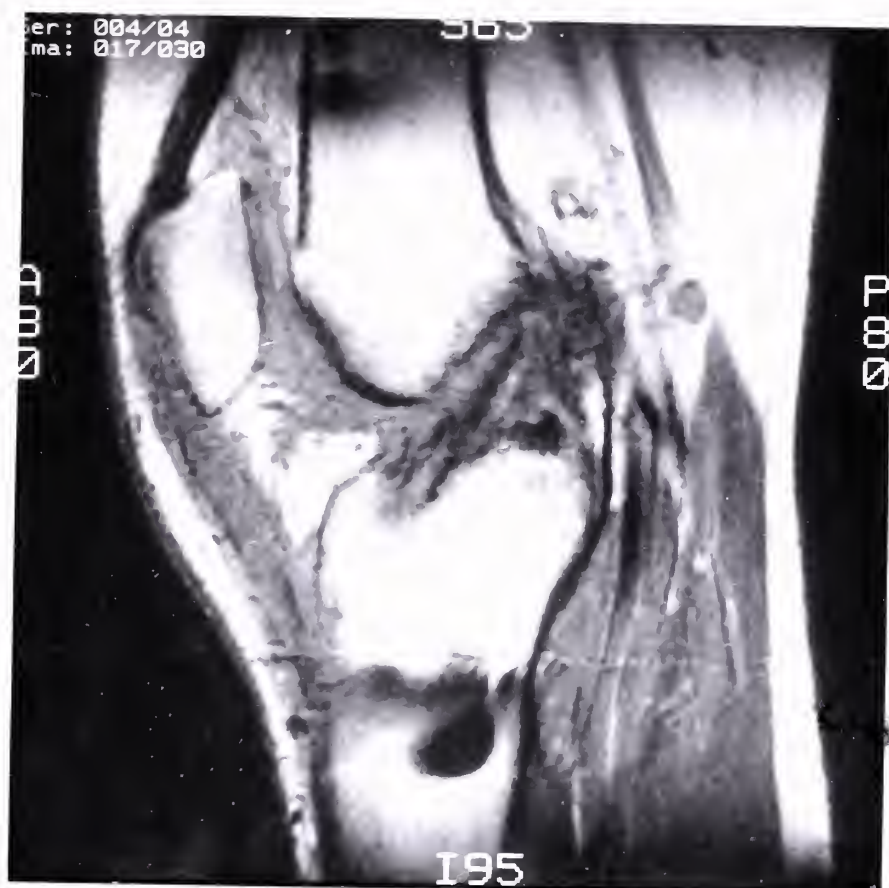
FIGURE 13-1A-D
     The following MR images are representative examples of patients in this study. All of these images are sagittal views of the same patient at different intervals after ACL reconstruction.
     Figure 13-1A is a patient 1.5 months after reconstruction. This proton density weighted image demonstrates the ACL graft's increased signal intensity relative to the ACL graft 1.5 months after reconstruction.

Figure 13-1B illustrates the same patient six months after reconstruction. This proton density weighted image demonstrates the ACL graft's increased signal intensity relative to normal tendon and relative to the ACL graft 1.5 months after reconstruction.
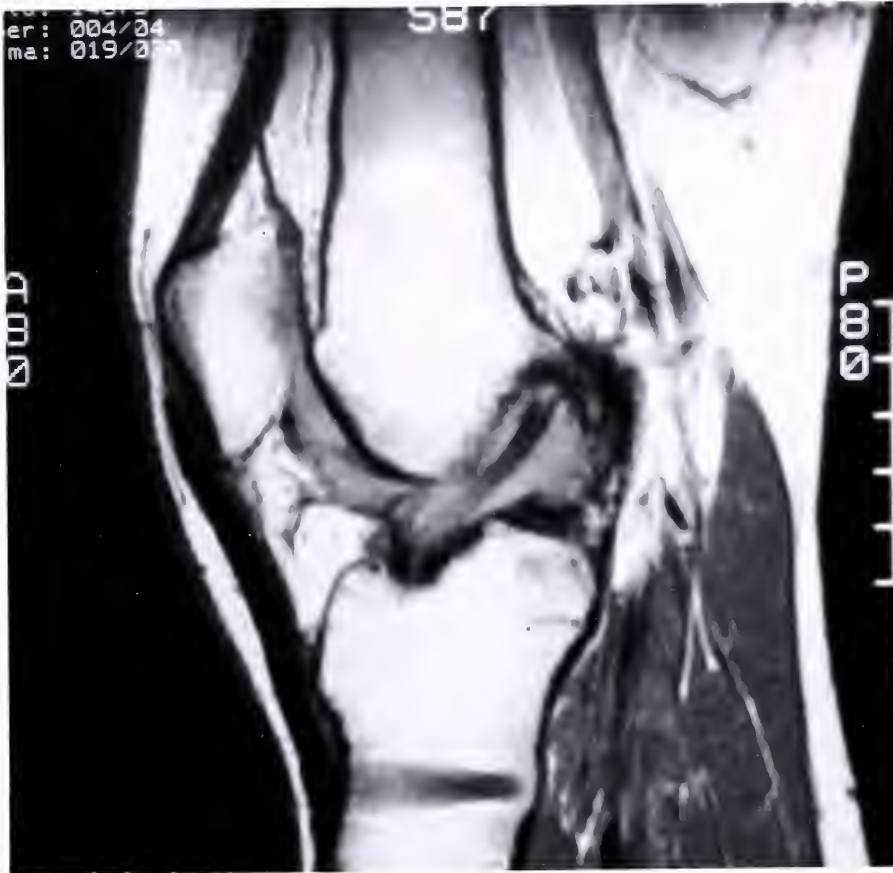
Figures 13-1C is a proton density weighted image of the same patient twelve months after reconstruction. The image is shown to illustrate the persistent increased signal intensity relative to normal tendon.

Figure 13-1D illustrates the same patient twenty-four months after reconstruction. This proton density weighted image demonstrates the ACL graft's increased signal intensity relative to normal tendon and relative to the ACL graft 1.5 months after reconstruction.

REFERENCES

1. Amiel D, Frank C, Harwood F, Fronek J, Akeson WH, <u>Tendons and Ligaments: A Morphological and Biochemical Comparison</u>, **J. Orthop. Res.**, 1(3):257 (1984).

2. Amiel D, Kleiner JB, Roux RD, Harwood FL, Akeson WH, <u>The Phenomenon of "Ligamentization": Anterior Cruciate Ligament Reconstruction with Autogenous Patellar Tendon</u>, J. Orthop. Res., 4(2):162 (1986).

3. Anderson AF, Lipscomb AB, <u>Analysis of Rehabilitation Techniques after Anterior Cruciate Reconstruction</u>, Am. J. **Sports Medicine**, 17(2):154 (1989).

4. Arnoczky SP, <u>Anatomy of the Anterior Cruciate Ligament</u>, Clin. **Ortho.**, 172:19 (1983).

5. Arnoczky SP, Tarvin GB, Marshall JL, <u>Anterior Cruciate Ligament Replacement Using Patellar Tendon</u>, **J. Bone Joint Surg.**, 64-A(2):217 (1982).

6. Arnoczky SP, Warren RF, <u>The Microculature of the Meniscus and its Response to Injury: An Experimental Study in the Dog</u>, **Am. J. Sports Med.**, 11:131 (1983).

7. Arnoczky SP, Warren RF, Spivak JM, <u>Meniscal Repair Using an Exogenous Fibrin Clot</u>, **J. Bone Joint Surg.**, 70A:1209 (1988).

8. Arviddson E, Ericksson E, Haggmark T, Johnson RJ, <u>Isokinetic Thigh Muscle Strength after Ligament Reconstruction in the Knee Joint: Results from a 5-10 Year Follow-up after Reconstruction of the Anterior Cruciate Ligament in the Knee Joint</u>, **Int. J. Sports Med.**, 2:7-11, (1981).

9. Bennett JG, Stauber WT, <u>Evaluation and Treatment of Anterior Knee Pain Using Eccentric Exercise</u>, **Med. & Sci. in Sports & Exercise**, 18(5):526-30 (1986).

10. Bergfeld JA, Clancy WG, DeHaven KE, Johnson RJ, <u>Injury to the Anterior Cruciate Ligament: A Round Table</u>, **Phys. Sports Med.** 10:47-59 (1982).

11. Bessette GC, Hunter RE, <u>The Anterior Cruciate Ligament</u>, **Orthopedics**, 13(5):551 (May 1990).

12. Butler DL, Grood ES, Noyes FR, Sodd AN, <u>On the Interpretation of Our Anterior Cruciate Ligament Data</u>, **Clin. Ortho.**, 196:26 (1985).

13. Campbell WC, <u>Reconstruction of the Ligaments of the Knee Joint</u>, **Surg. Gynecol. Obstet.**, 62:964 (1936).

14.   Cabaud HE, Rodkey WG, Philosophy and Rationale for the
       Management of Anterior Cruciate Injuries and the Resultant
       Deficiencies, Clinics in Sports Med., 4(2):313 (1985).

15.   Clancy WG, Narechania RG, Rosenberg TD, Gmeiner JG, Wisnefke
       DD, Lange TA, Anterior and Posterior Cruciate Ligament
       Reconstruction in Rhesus Monkeys, J. Bone Joint Surg.,
       63A:1270 (1981).

16.   Clancy WG Jr., Anterior Cruciate Ligament Functional
       Instability, Clin. Ortho., 172:102 (1983).

17.   Clancy WG, Arthroscopic Anterior Cruciate Ligament
       Reconstruction with Patellar Tendon, Tech. Orthop., 2(4):13
       (1988).

18.   Collins HR, Hughston JC, DeHaven KE, Bergfeld JA, Evarts, C,
       The Meniscus as a Cruciate Ligament Substitute, J. Sports
       Med., 2:11 (1974).

19.   Currier DP, Positioning for Knee Strengthening Exercises,
       Physical Ther., 57(2):148 (1977).

20.   DeHaven KE, Arthroscopy in the Diagnosis and Management of
       the Anterior Cruciate Ligament Deficient Knee, Clin. Ortho.
       172 (1983).

21.   DeHaven, KE, Diagnosis of Acute Knee Injuries with
       Hemarthrosis, Am. J. Sports Med., 8:9 (1980).

22.   DeHaven KE, Decision-Making Factors in the Treatment of
       Meniscus Lesions, Clin. Ortho., 252:49.

23.   Deutsch AL, Mink JH, Fox JM, Arnoczky SP, Rothman
       BJ, Stoller DW, Cannon WD Jr., Peripheral Meniscal Tears: MR
       Findings After Conservative Treatment or Arthroscopic Repair,
       Radiology, 176:486 (1990).

24.   Donaldson WF, Warren RF, Wickiewicz TA, A Comparison of Acute
       Anterior Cruciate Ligament Examination, Am. J. Sports Med.,
       13(1):5 (1985).

25.   Durkan JA, Wynne GF, Haggerty JF, Extraarticular
       Reconstruction of the Anterior Cruciate Ligament Insufficient
       Knee. A Long-Term Analysis of the Ellison Procedure, Am. J.
       Sports Med., 17(1):112 (1989).

26.   Dye SF, Cannon WD Jr., Anatomy and Biomechanics of the
       Anterior Cruciate Ligament, Clinics in Sports Med., 7(4):715
       (1988).

27.   Ellison, A.E., Distal Iliotibial-Band Transfer for
       Anterolateral Rotatory Instability of the Knee, J. Bone Joint
       Surg., 61A:330 (1979).

28. Ellison AE, Berg EE, <u>Embryology, Anatomy, and Function of the Anterior Cruciate Ligament</u>, **Ortho. Clin.**, 16(1):3 (1985).

29. Engebretsen L, Benum P, Fasting O, Molster A, Strand T, <u>A Prospective, Randomized Study of Three Surgical Techniques for Treatment of Acute Ruptures of the Anterior Cruciate Ligament</u>, **Am. J. Sports Med.**, 18(6):585 (1990).

30. Eriksson E, <u>Reconstruction of the Anterior Cruciate Ligament</u>, **Orthro. Clin.**, 7:167 (1976).

31. Farrell M, Richards JG, <u>Analysis of the Reliability and Validity of the Kinetic Communicator Exercise Device</u>, **Med. & Sci. in Sports & Exercise**, 18(1):44 (1986).

32. Feagin JA, Walton WC, <u>Isolated Tear of the Anterior Cruciate Ligament: 5-Year Follow-Up Study</u>, **Am. J. Sports Med.**, 4:95 (1976).

33. Feagin JA Jr., <u>Operative Treatment of Acute and Chronic Knee Problems</u>, **Clinics in Sports Med.**, 4(2):325 (1985).

34. Feagin JA, Blake WP, <u>Postoperative Evaluation and Result Recording in the Anterior Cruciate Ligament Reconstructed Knee</u>, **Clin. Ortho.** 172:143 (1983).

35. Feagin JA, Cabaud HE, Curt WW, <u>The Anterior Cruciate Ligament Radiographic and Clinical Signs of Successful Repairs</u>, **Clin. Ortho.**, 54 (1982).

36. Feagin, John A., <u>The Crucial Ligaments</u>, Churchill, Livingstone, New York, N.Y. (1988).

37. Fell HB, Canti RG, <u>Experiments on the Development in Vitro of the Avaian Knee-Joint</u>, **Proc. R. Soc. Land (Biol.)**, 116:316 (1934).

38. Ferkel, Markoff, Fox, DelPizzo, Friedman, Stryker, Strun, <u>Instrumented Clinical Knee Testing for A-P Stability. How, What, Why?</u> Center for Disorders of the Knee.

39. Ferkel RD, Fox JM, Del Pizzo W, Friedman MJ, Snyder SJ, Dorey F, Kasimian D, <u>Reconstruction of the Anterior Cruciate Ligament Using a Torn Meniscus</u>, **J. Bone Joint Surg.**, 70-A(5):715 (1988).

40. Ferkel RD, Markolf K, Goodfellow D, Sprague NA, Friedman MJ, Dorey F, Fox JM, Del Pizzo W, <u>Treatment of the Anterior Cruciate Ligament-absent Knee with Associated Meniscal Tears</u>, **Clin. Ortho.**, 222:239 (1987).

41. Feto JW, Marshall JL, <u>The Natural History and Diagnosis of the Anterior Cruciate Ligament Insufficiency</u>, **Clin. Ortho.**, 147:29 (1980).

42.   Fowler PJ, Regan WD, The Patient With Symptomatic Chronic Anterior Cruciate Ligament Insufficiency, Am. J. Sports Med., 15(4):321 (1987).

43.   Frank C, Amiel D, Woo SL-Y, Akeson W, Normal Ligament Properties and Ligament Healing, Clin. Ortho., 196:15 (1985).

44.   Franke K, Cruciate Experience in 130 Cruciate Ligament Reconstructions, Ortho. Clin., 7:191 (1976).

45.   Frazer, HA, Diagnostic Imaging, Imaging News, 33 (1990).

46.   Friedman MJ, Arthroscopic Semitendinosus (Gracilis) Reconstruction for Anterior Cruciate Ligament Deficiency, Techniques Orthop., 2(4):74 (1988).

47.   Furman W, Marshall JL, Girgis FG, The Anterior Cruciate Ligament: A Functional Analysis Based on Postmortam Studies, J. Bone Joint Surg. 58:179 (1976).

48.   Gersoff WK, Clancy WG Jr., Diagnosis of Acute and Chronic Anterior Cruciate Ligament Tears, Clinics in Sports Med., 7(4):727 (Oct. 1988).

49.   Giove TP, Miller SJ, Kent B, Sanford T, Stanford, Garrick J, Non-Operative Treatment of the Torn Anterior Cruciate Ligament, J. Bone Joint Surg., 65A(2):184 (1983).

50.   Girgis FG, Marshall JL, AlMonajem ARS: The Cruciate Ligaments of the Knee Joint. Anatomical Functional and Experimental Analysis, Clin. Ortho. 106:216 (1975).

51.   Glashow JL, Katz, R., Schneider, M., and Scott, N, Double-Blind Assessment of the Value of Magnetic Resonance Imaging in the Diagnosis of Anterior Cruciate and Meniscal Lesions, Vol. 71-A(1), January 1989.

52.   Glousman R, Shields C, Kerlan R, Jobe F, Lambardo S, Yocum L, Tibone J, Gambardella R, Gore-Tex Prosthetic Ligament in Anterior Cruciate Deficient Knees, Am. J. Sports Med., 16(4):321.

53.   Granberry WM, Noble PC, and Woods GW, Evaluation of Electrogoniometric Instrument for Measurement of Laxity of the Knee, J. Bone Joint Surg., 72-A:1316 (1990).

54.   Gray DJ, Gardner E, Prenatal Development of the Knee Joint, Am. J. Anat., 86:235 (1950).

55.   Grood, ES, Noyes, FR: Cruciate Ligament Prosthesis: Strength, Creep and Fatigue Properties. J. Bone Joint Surg. 58:1083 (1976).

56.   Hageman PA, Gillaspie DM, Hill LD, <u>Effects of Speed and Limb Dominance on Eccentric and Concentric Isokinetic Testing of the Knee</u>, **J. of Ortho. and Sports Phy. Ther.**, 10(2):59 (1988).

57.   Harding B, Black T, Bruulsema A, Maxwell B, Stratford P, <u>Reliability of a Reciprocal Test Protocol Performed on the Kinetic Communicator: An Isokinetic Test of Knee Extensor and Flexor Strength</u>, **J. of Ortho. & Sports Phy. Ther.**, 10(6):218 (1988).

58.   Harter RA, Osterning LR, Singer KM, James SL, Larson RL, Jones DC, <u>Long-Term Evaluation of Knee Stability and Function Following Surgical Reconstruction for Anterior Cruciate Ligament Insufficiency</u>, **Am. J. Sports Med.**, 16(5):434 (1988).

59.   Henning, CE, Lynch, MA, Glick, KR. <u>An In Vivo Strain Gauge Study of Elongation of the Anterior Cruciate Ligament</u>, **Am. J. Sports Med.**, 13:22 (1985).

60.   Highenboten CL, Jackson AW, Meske NB, <u>Concentric and Eccentric Torque Comparisons for Knee Extension and Flexion in Young Adult Males and Females Using the Kinetic Communicator</u>, **Am. J. Sports Med.**, 16(3):234 (1988).

61.   Highgenboten, "The reliability of the Genucom System" Presented at the Second European Congress of Knee Surgery and Anthroscopy, Basel, Switzerland, September 29, 1986, **Am. J. Surg. Med.** 16(3):234 (1988).

62.   Hislop HJ, Perrine JJ, <u>The Isokinetic Concept of Exercise</u>, **Physical Ther.**, 47(2):114-17.

63.   Howell S, Clark J, Blaiser RD, <u>Serial Magnetic Resonance Imaging of Hamstring Anterior Cruciate Ligament Autografts During the First Year of Implantation</u>, **Am. J. Sports Med.**, 19:42 (1991).

64.   Jackson DW, Jennings LD, <u>Arthroscopically Assisted Reconstruction of the Anterior Cruciate Ligament Using a Patella Tendon Bone Autograft</u>, **Clinics in Sports Med.**, 7(4):785 (1988).

65.   Jackson DW, Jennings LD, RM Maywood, Berger PE, <u>Magnetic Resonance Imaging of the Knee</u>, **Am. J. Sports Med.**, 16(1):29 (1988).

66.   Johnson, Robert J., M.D., The Anterior Cruciate Ligament
        Problem, **Clin. Ortho.** 172:14 (1983).

67.   Johnson RJ, Eriksson E, Haggmark T, Pope MH, Five- to Ten-
        Year Follow-Up Evaluation After Reconstruction of the
        Anterior Cruciate Ligament, **Clin. Ortho.**, 183:122 (1984).

68.   Jones KG, Reconstruction of the Anterior Cruciate Ligament
        (Using the Central One Third of the Patellar Ligament), J.
        Joint Bone Surg., 52A:1302 (1970).

69.   Kapandu JA, The Physiology of the Joints, Vol. II Churchill
        Livingston, New York (1970).

70.   Kennedy JC, Stewart R, Walker D.M., Anterolateral Rotatory
        Instability of the Knee Joint. An Early Experience of the
        Ellison Procedure, **J. Bone Joint Surg.**, 60A:1031 (1978).

71.   King S, Butterwick DJ, Cuerrier JP, The Anterior Cruciate
        Ligament: A Review of Recent Concepts, **J. Ortho. & Sports
        Phy. Ther.**, 8(3):110 (1986).

72.   Kleiner JB, Amiel D, Harwood FL, Akeson WH, Early Histologic,
        Metabolic, and Vascular Assessment of Anterior Cruciate
        Ligament Autografts, **J. of Ortho. Res.**, 7:235 (1989).

73.   Kornblatt I, Warren RF, Wickiewicz TL, Long-term Followup of
        Anterior Cruciate Ligament Reconstruction Using the
        Quadriceps Tendon Substitution for Chronic Anterior Cruciate
        Ligament Insufficiency, **Am. J. Sports Med.**, 16(5):444
        (1988).

74.   Lambert KL, Vascularized Patellar Tendon Graft with Rigid
        Internal Fixation for Anterior Cruciate Ligament
        Insufficiency, **Clin. Ortho.**, 172:78 (1983).

75.   LeStrange NR, "The Anterior Cruciate Ligament: Evaluating
        and managing acute injuries," **Medical Student** 16(4):4 (1990).

76.   Lipke JM, Janecki CJ, Nelson CL, McLeod P, Thompson C,
        Thompson J, Haynes DW: The Role of Incompetance of the
        Anterior Cruciate and Lateral Ligaments in Anterolateral and
        Anteromedial Instability, **J. Bone Joint Surg.**, 63:954
        (1981).

77.   LoPresti C, Kirkendale D, Street G, Dudley AW, Quadriceps
        Insufficiency Following Repair of the Anterior Cruciate
        Ligament, **J. Ortho. & Sports Phy. Ther.**, 9(7):245 (1988).

78.   Lorentzon R, Elmquist LG, Sjostro M, Markku F, Fuglmeyer A,
        Thigh Musculature in Relation to Chronic Anterior Cruciate
        Ligament Tear and Muscle Size, Morphology, and Mechanical
        Output Before Reconstruction, **Am. J. Sports Med.**, 17(3):423
        (1989).

79.   MacIntosh DL, Darby TA, <u>Lateral Substitution Reconstruction.</u> <u>In Proceedings of the Canadian Orthopedic Association</u>, J. **Bone Joint Surg.**, 58B:142 (1976).

80.   Malcolm LL, Daniel DM, Stone ML, Sachs R, <u>The Measurement of</u> <u>Anterior Knee Laxity After ACL Reconstructive Surgery</u>, Clin. **Ortho.**, 186 (1985).

81.   Maltry JA, Nobel, PC, Woods GW, Alexander JW, Feldman GW, Tullos HS, <u>External Stabilization of the Anterior Cruciate</u> <u>Ligament Deficient Knee During Rehabilitation</u>, **Am. J. Sports Med.**, 17(4):550 (1989).

82.   Markolf KL, Kochan A, Amstutz HC, <u>Measurement of Knee</u> <u>Stiffness and Laxity in Patients With Documented Absence of</u> <u>Anterior Cruciate Ligament</u>, **J. Bone Joint Surg.**, 66-A(2):242 (1984).

83.   Markolf KL, Amstutz HC, <u>The Clinical Relevance of</u> <u>Instrumented Testing for ACL Insufficiency</u>, Clin. Ortho., 223:198 (1987).

84.   <u>MRI of the Musculoskeletal System</u>, Mink JW, Deutsch AL, Raven Press, New York, N.Y. (1990).

85.   Meyers JF, St. Pierre R.K., Sutter JS, Caspari RB, Whipple TL, <u>Arthroscopic Evaluation of Anterior Cruciate Ligament</u> <u>Reconstructions</u>, **Arthroscopy**, 2(3):155 (1986).

86.   Moeser P, Bechtold RE, Clark T, Rovere G, Karstaedt N, Wolfman N, <u>MR Imaging of Anterior Cruciate Ligament Repair</u>, **J. Computer Assisted Tomography**, 13(1):105 (1989).

87.   Muller W, <u>The Knee: Form, Function and Ligament</u> <u>Reconstruction</u>, New York, NY, Springer-Verlga (1983).

88.   Nicholas JS, Minkoff J, <u>Iliotibial Band Transfer Through the</u> <u>Intercondylar Notch for Combined Anterior Instability (ITPT</u> <u>Procedure)</u>, **Am. J. Sports Med.**, 6:341 (1978).

89.   Norwood LA, Cross MJ, <u>Anterior Cruciate Ligament: Functional</u> <u>Anatomy of Its Bundles in Rotatory Instabilities</u>, **Am. J. Sports Med.**, 7(1):23.

90.   Noyes FR, Butler DL, Grood ES, Zernicke R, Hefzy M., <u>Biomechanical Analysis of Human Ligaments Used In Knee-</u> <u>Ligament Repairs and Reconstructions</u>, J. Bone Joint Surg., 66A:344 (1984).

91. Noyes FR, Butler DL, Paulos LE, Grood ES, Intra-Articular Cruciate Reconstruction. I: Perspectives on Graft Strength, Vascularization, and Immediate Motion after Replacement, **Clin. Ortho.**, 172:71 (1983).

92. Noyes FR, Mangine RE, Barber S, Early Knee Motion After Open and Arthroscopic Anterior Cruciate Ligament Reconstruction, **Am. J. Sports Med.**, 15(2):149 (1987).

93. Odensten M, Gillquist I, Functional Anatomy of the Anterior Cruciate Ligament and a Rationale for Reconstruction, J. Bone Joint Surg., 67A:257 (1985).

94. O'Donahue DH, A Method for Replacement of the Anterior Cruciate Ligament of the Knee, J. Bone Joint Surg., 45A:905 (1963).

95. O'Donaghue DH, Surgical Treatment of Fresh Injuries to the Major Ligaments of the Knee, J. Bone Joint Surg. 32A:721 (1950).

96. Oliver JH, Coughlin LP, Objective Knee Evaluation Using the Genucom Knee Analysis System, **Am. J. Sports Med.**, 15(6):571 (1987).

97. Pavlov H, The Radiographic Diagnosis of the Anterior Cruciate Ligament Deficient Knee, **Clin. Ortho.**, 172:57 (1983).

98. Penner DA, Daniel DM, Wood P, Mishra D, An In Vitro Study of Anterior Cruciate Ligament Graft Placement and Isometry, **Am. J. Sports Med.**, 16(3):238 (1988).

99. Rak KM, Gillogly SD, Schaefer RA, Yakes WF, Liljedahl RR, Anterior Cruciate Ligament Reconstruction: Evaluation with MR Imaging, **Radiology**, 178:553 (1991).

100. Reicher MA, Hartzman S, Bassett LW, Mandelbaum B, Duckwiler G, Gold RH, MR Imaging of the Knee, **Radiology**, 162:547 (1987).

101. Robson MA, Ruptured Cruciate Ligaments and Their Repair By Operation, **Ann. Surg.**, 37: 716 (1903).

102. Roth JH, Kennedy JC, Lockstadt H, McCallum CL, Dunning IA, Intra-Articular Supplementation By Transfer of the Biceps Femoris Tendon, **J. Bone Joint Surg.**, 69-A(2):275 (1987).

103. Sandbert R, Balkfors B, The Durability of Anterior Cruciate Ligament Reconstruction with the Patellar Tendon, **Am. J. Sports Med.**, 16(4):341 (1988).

104. Schultz RA, Miller DC, Kerr CS, Micheli L, <u>Mechanoreptors in Human Cruciate Ligaments</u>, **J. Bone Joint Surg.**, 66A:1072 (1984).

105. Seto JL, Orofino AS, Morrissey MC, Medeiros JM, Mason WJ, <u>Assessment of Quadriceps/Hamstring Strength, Knee Ligament Stability, Functional and Sports Activity Levels Five Years After Anterior Cruciate Ligament Reconstruction</u>, **Am. J. Sports Med.**, 16(2):170 (1988).

106. Shields CL, Silva I, Yee L, Brewster C, <u>Evaluation of Residual Instability after Arthroscopic Meniscectomy in Anterior Cruciate Deficient Knees</u>, **Am. J. Sports Med.**, 15(2):129 (1987).

107. Smith Alwyn S, <u>The Diagnosis and Treatment of Injuries to the Crucial Ligaments</u>, **Brit. J. Surg.**, 6:176 (1918).

108. Snook George A, <u>A Short History of the Anterior Cruciate Ligament and the Treatment of Tears</u>, **Clin. Ortho.**, 172:11 (1983).

109. Solomonow M, Baratta R, Shou BH, Shoji H, Bose W, Beck C, D'Ambrosia R, <u>The Synergistic Action of the Anterior Cruciate Ligament and Thigh Muscles in Maintaining Joint Stability</u>, **Am. J. Sports Med.**, 15(3):207 (1987).

110. Solonen KA, Hjelt L, Rinne T, <u>Anterior Cruciate Ligament Reconstruction by a Vascularized and Innervated Graft</u>, **Acta Orthop. Scand.**, 39:82 (1968).

111. Stark J, <u>Two Cases of Ruptured Cruciate Ligaments of the Knee Joint</u>, **Edinburgh Med. Surg.**, 74:267 (1850).

112. Steiner ME, Brown C, Zarins B, Brownstein B, Koval PS, and Stone P, <u>Measurement of Anterior-Posterior Displacement of the Knee</u>, **J. Bone Joint Surg.**, 72-A:1307 (1990).

113. Strum GM, Fox JM, Ferkel RD, Dorey FH, Del Pizzo W, Friedman MJ, Snyder SJ, Markoff K, <u>Intraarticular Versus Intraarticular and Extraarticular Reconstruction for Chronic Anterior Cruciate Ligament Instability</u>, **Clin. Ortho.** 245:188 (1989).

114. Stull P, "Recovery from Arthroscopic Knee Surgery -- A Prospective Randomized Trial Comparing Supervised Outpatient Physical Therapy and Unsupervised Home Exercise in Patient after Arthroscopic Partial Meniscectomy," Yale Medical School Thesis, 1988.

115. Svelstrup H, Hoshizake TB, Vagenas G, "Asymmetrical Knee Laxities: An Objective Evaluation" Presented at the North American Conference on Biomechanics, Sept. 1986.

116. Thomas NP, Jackson AM, Afeborgh PM, <u>Congenital Absence of the Anterior Cruciate Ligament</u>, **J. Bone Joint Surg.**, 67B:572 (1985).

117. Tibone JE, Antich TJ, Perry J, Moynes D, <u>Functional Analysis of Untreated and Reconstructed Posterior Cruciate Ligament Injuries</u>, **Am. J. Sports Med.**, 16(3):217 (1988).

118. Tillberg B, <u>The Late Repair of Torn Cruciate Ligaments Using Menisci</u>, **J. Joint Bone Surg.**, 59B:15 (1977).

119. Van Rens TJG, van den Berg AF, Huiskes R, and Kuypers W, <u>Substitution of the Anterior Cruciate Ligament: A Long-Term Histologic and Biomechanical Study with Antogenous Pedicled Grafts of the Iliotibial Band in Dogs</u>, **Arthroscopy**, 2(3):139 (1986).

120. Walsh JJ, <u>Meniscal Reconstruction of the Anterior Cruciate Ligament</u>, **Clin. Ortho.**, 89:171 (1972).

121. Warman M, "The Effect of Treadmill Exercise on Genucom -- Measured Knee Ligament Laxity," Yale Medical School Thesis, 1990.

122. Warren RF, Marshall JL, <u>Injuries of the Anterior Cruciate and Medial Collateral Ligments of the Knee: A Retrospective Analysis of Clinical Records -- Part I</u>, **Clin. Ortho.**, 136:191 (1978).

123. Wojtys E, Wilson M, Buckwalter K, Braunstein E, Martel W, <u>Magnetic Resonance Imaging of Knee Hyaline Cartilage and Intraarticular Pathology</u>, **Am. J. Sports Med.**, 15(5):455 (1987).

124. Woods GW, Chapman DR, <u>Repairable Posterior Meniscocapsular Disruption in Anterior Cruciate Ligament Injuries</u>, **Am. J. Sports Med.**, 12:381 (1984).

125. Wroble RR, Grood ES, Noyes FR, Schmitt DJ, <u>Reproducibility of Genucom Knee Analysis System Testing</u>, **Am. J. Sports Med.**, 18(4):387 (1990).

126. Yahia L-H, Drouin G, <u>Miscroscopical Investigation of Canine Anterior Cruciate Ligament and Patellar Tendon: Collagen Fascicle Morphology and Architecture</u>, **J. Ortho. Res.**, 7:243 (1989).

127. Yoshiya S, Andrich JT, Manley MT, Bauer TW, <u>Graft Tension in Anterior Cruciate Ligament Reconstruction. An In Vivo Study in Dogs</u>, **Am. J. Sports Med.**, 15(5):464 (1987).

128. Zimny ML, Schutte M, Dabezies E, <u>Mechanoreceptors in the Human Anterior Cruciate Ligament</u>, **Anat. Res.**, 214(2):204 (1986).

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