REVISION ANTERIOR CRUCIATE LIGAMENT SURGERY

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The importance of the anterior cruciate ligament (ACL) to the maintenance of normal knee function is now well accepted.[94] An untreated ACL tear can lead to recurrent giving-way episodes, damage to the menisci and articular cartilage, with progression to osteoarthritis in some patients.[9, 38, 41, 42, 91, 94, 106, 141] The poor long-term results of nonoperative treatment, primary repair, and extra-articular reconstruction have led to intra-articular ACL reconstruction becoming the surgical procedure of choice for an athletically active patient with a functionally unstable knee.* Reconstruction of the ACL is one of the most commonly performed orthopedic operations. According to the National Center of Health Statistics, in 1991 approximately 63,000 ACL reconstructions were performed in the United States. By current industry estimates, over 100,000 ACL reconstructions are performed annually in the United States.

The success rate of primary ACL reconstruction has been reported to range from 75% to 93%, good or excellent results with respect to relief of giving-way symptoms, restoration of functional stability, and return to normal or near normal activity levels.† Given the reported success rates, a significant number of patients who undergo ACL reconstruction may have a less than satisfactory outcome. What qualifies as a unsatisfac-

*References 2, 4, 9, 12–14, 27, 32, 38, 41, 42, 61, 73, 74, 85, 91, 100, 106, 109–112, 130–132, 141, 152.

†References 2, 4, 5, 9, 12–14, 16, 27, 32, 34, 53, 56, 57, 61, 74, 79, 85, 98–100, 109, 111, 112, 130–132.

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tory result or "failure" after ACL reconstruction, however, has not been well defined or agreed on. Johnson and Fu[69] have defined a failed ACL reconstruction as a knee that demonstrates recurrent pathologic laxity that was present prior to surgery, or a stable knee that has a range of motion from 10 to 120 degrees of flexion that is stiff and painful even with activities of daily living. Although graft failure is the most common cause of failed ACL surgery, it is important to keep in mind that non–graft-related conditions may also result in persistent complaints and an unsatisfactory outcome. Failed ACL surgery can be classified into one of the following four categories with the potential for overlap among the categories in some cases[69]:

1. Loss of motion or arthrofibrosis
2. Extensor mechanism dysfunction
3. Arthritis
4. Recurrent patholaxity (graft failure)

It is important to characterize and categorize properly the residual complaints following the original ACL reconstruction in order to prevent a second ligament operation directed at improving anterior laxity of the knee from being performed, only to result in the patient continuing to experience the original non–graft-related complaints.

Given today's emphasis on maintaining fitness and the participation of all age groups in physical activities that place the ACL at risk for injury, the number of primary ACL reconstructions performed can be expected to continue to increase. Although surgical and rehabilitation advancements have improved the success rate of primary ACL reconstruction, the increasing number of primary reconstructions being performed can be expected to lead to an increasing number of patients with the potential need for revision ACL surgery.[44, 47, 59, 60, 120, 132, 134, 136]

In general, the results of revision ACL reconstruction do not appear to be as favorable as those of primary reconstructions.* The success rate of revision ACL reconstruction is determined by many factors including the origin of the primary failure, the preoperative laxity of the knee, the status of the secondary restraints, menisci and articular cartilage, and patient motivation and compliance.† In order to maximize the success of revision ACL surgery, a methodic and organized approach is required. This article reviews the etiology of failed ACL surgery, discusses preoperative evaluation and planning, and reviews some of the technical considerations of revision ACL surgery.

ETIOLOGY OF FAILED ACL SURGERY

Loss of Motion

Loss of motion is one of the most common complications following knee ligament surgery.‡ The incidence of loss of motion following ACL

*References 71, 72, 103, 117, 126, 151, 157, 158.
†References 68, 70, 124, 155, 157.
‡References 15, 48, 52, 64, 105, 113, 115, 122.

surgery has been reported to range from 5.5% to 24%.[52, 64, 122] The large variation in the reported incidence reflects differences in the criteria used to define the problem, differences between studies in the timing of surgery, and differences in surgical technique and postoperative rehabilitation. Delaying acute surgery, immediate postoperative motion with emphasis on full extension, patellar mobilization, early quadriceps exercises, and immediate weight bearing after ACL surgery have all been shown to reduce the incidence of loss of motion.[52, 64, 132, 134, 137]

Loss of motion can involve extension or flexion. Loss of extension is typically more disabling than loss of flexion.[52, 64, 122] Patients with a loss of extension tend to ambulate with an abnormal bent-leg gait pattern, have limited improvement with physical therapy, and develop anterior knee pain and quadriceps muscle weakness.[52, 64, 122] Loss of flexion rarely causes functional problems unless the knee fails to flex to at least 120 degrees.[64] Loss of flexion primarily interferes with activities, such as running, stair-climbing, squatting, kneeling, and sitting.

The origin of loss of motion is multifactorial and includes impingement, capsulitis, concomitant ligament surgery, errors in surgical technique, immobilization, reflex sympathetic dystrophy, and infection.[15, 52, 64] Current treatment is directed at prevention. Impingement results from a physical block in the intercondylar notch, which prevents full extension of the knee.[52, 64] Impingement can be caused by intercondylar notch scarring (the cause in approximately 50% of the cases); a cyclops lesion; an anteriorly placed ACL graft; or an inadequate notchplasty.[52, 64] Patients with loss of motion secondary to impingement usually complain of morning stiffness, which improves with motion as the day goes on. The knee is minimally swollen and demonstrates a loss of terminal extension. Flexion and patellar mobility are usually normal. In the early stages treatment consist of serial extension casting or a drop-out cast, and quadriceps strengthening exercises.[64, 105, 113] Surgical intervention is usually necessary in the later stages of the disease process, and consists of arthroscopic debridement of the tissue impinging in the notch, revision notchplasty, followed by serial extension casting or a drop-out cast (Fig. 1).[15, 52, 64, 105]

Capsulitis is defined as periarticular inflammation and swelling, and results in the development of adhesions and intra-articular scar formation.[52, 64] Capsulitis typically causes a loss of both flexion and extension, and also results in a restriction of patellar mobility.[64] The loss of extension and decrease in patellar mobility can lead to quadriceps weakness with loss of pull through the extensor mechanism. If unrecognized and untreated, the loss in pull through the extensor mechanism may lead to an adaptive shortening of the patellar tendon, patella baja, and the development of a infrapatellar contracture syndrome.[15, 108, 113, 115]

Capsulitis can be either primary or secondary.[64] Primary capsulitis is defined as an exaggeration of the normal inflammatory process caused by surgery or trauma. Primary capsulitis is a diagnosis of exclusion after secondary causes have been eliminated. Secondary causes of capsulitis

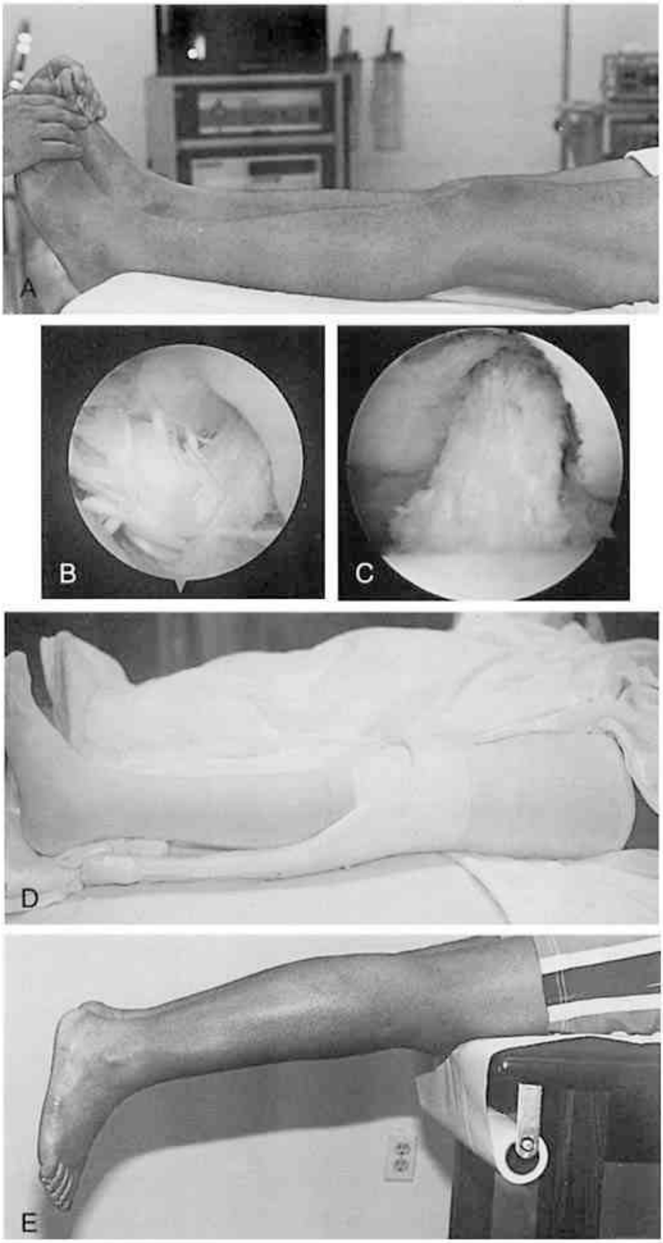


Figure 1. *A*, Loss of extension following anterior cruciate ligament (ACL) reconstruction with autogenous patellar tendon graft. The patient complained of stiffness, anterior knee pain, quadriceps muscle weakness, and walked with a bent-leg gait pattern. *B*, Arthroscopic examination revealed fraying of the anterior fibers of the ACL graft secondary to roof impingement which produced a cyclops lesion. *C*, The cyclops lesion and frayed ACL graft fibers were excised, and revision notchplasty was performed. *D*, A drop-out cast was used to maintain extension in the postoperative period. *E*, Three-month follow-up examination, demonstrating full restoration of extension.

include surgery performed during the acute inflammatory stage following injury, improper surgical technique, and postoperative immobilization or restriction of motion. Secondary capsulitis can be prevented by appropriate timing of surgery, proper surgical technique, immediate motion, early quadriceps muscle exercises, patellar mobilization, and early weight bearing after surgery.

Patients with capsulitis usually complain of constant pain and stiffness. Examination usually demonstrates an actively inflamed and diffusely swollen knee, a quadriceps lag, a loss of more than 10 degrees of extension and 25 degrees of flexion, and limited patella mobility. Treatment of capsulitis depends on the stage of the process. In the early stages of the disease process the goals of treatment are to reduce pain and inflammation and to restore motion and quadriceps strength.[52, 64]

The primary goal during the early stages of the disease is directed at reduction of the inflammatory reaction. Aggressive forceful manipulations should be avoided during this stage because they may further stimulate the inflammatory process. Cryotherapy, anti-inflammatory medications, gentle stretching exercises, and overnight splinting in extension are prescribed. Once the inflammatory state of the knee has calmed down, and the patient has entered the fibrotic phase of the disease process, arthroscopic debridement and lateral release should be considered. Extension should be obtained first, followed by flexion (Fig. 2). The final and most advanced phase of capsulitis is the infrapatellar contracture syndrome.[15, 108, 113, 115] If the disease progresses to this stage, open debridement and open releases are usually required to restore a functional range of motion.[108, 113, 115]

The etiology of the loss of motion can usually be determined by a thorough history and physical examination. Before considering revision ligament surgery, correct identification of the origin of the loss of motion is critical so that a logical treatment plan can be formulated. It is especially important to determine the inflammatory state of the knee before considering surgical intervention. Surgical intervention should be avoided when the knee is in a highly inflamed state to avoid further stimulating the inflammatory process and further compromising the range of motion.

Nonanatomic graft placement is often accompanied by a loss of motion. In these cases a maximal, painless range of motion should be restored prior to considering revision ligament surgery. Therefore, a staged approach is required in most cases. The goal of the first stage is to obtain a painless, functional range of motion, and the second stage addresses any residual instability if present (Fig. 3).

Extensor Mechanism Dysfunction

Dysfunction of the extensor mechanism in an otherwise stable knee can also lead to failure of the original ACL reconstruction. Extensor mechanism dysfunction includes anterior knee pain; quadriceps muscle weakness; patellar tendinitis; problems secondary to harvesting (patellar

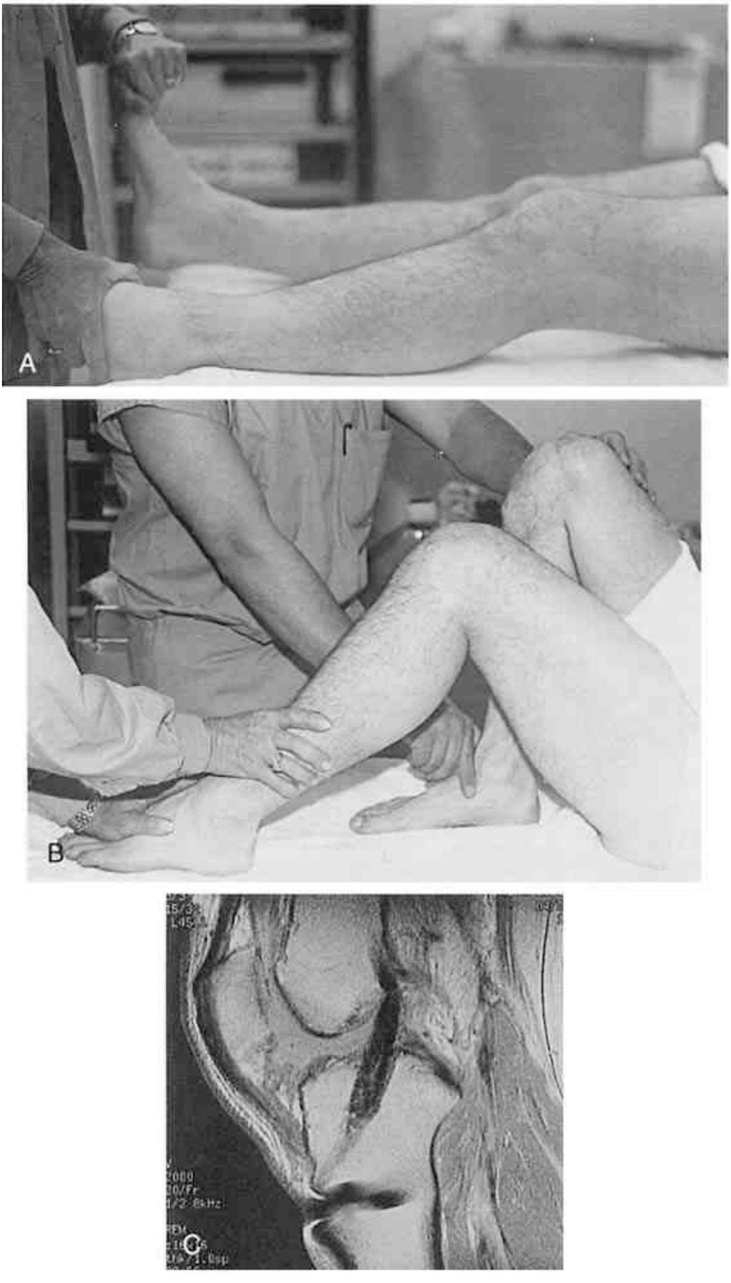


Figure 2. *See legend on opposite page*

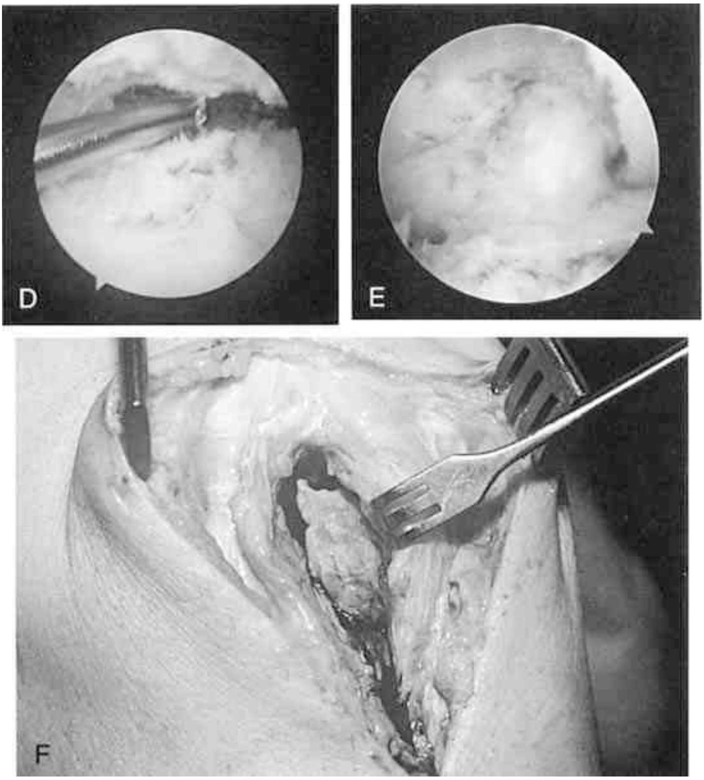


Figure 2. *A* and *B*, Secondary capsulitis (acute surgery), resulting in loss of both extension and flexion. The patient underwent acute ACL reconstruction with a quadrupled hamstring tendon graft before achieving full extension. *C*, The quadrupled hamstring tendon ACL graft has a normal MRI appearance. Non–graft-related problems can be a cause of failed ACL surgery. *D*, Arthroscopic débridement of adhesions in the suprapatellar pouch. An arthroscopic lateral release was required to restore patellar mobility. *E*, Revision notchplasty was required to restore extension. The ACL graft was normal. *F*, Excision of the fibrotic fat pad was required to restore normal gliding between the patellar tendon and the anterior surface of the tibia.

fracture, extensor mechanism rupture, donor site pain); and the infrapatellar contracture syndrome.* As mentioned previously, there is often an overlap between loss of motion and extensor mechanism dysfunction (Fig. 4).

Anterior knee pain is one of the most common complications following ACL reconstruction.[3, 122] The incidence of anterior knee pain following ACL reconstruction has been reported to range from 3% to 47%.† The large variation in incidence reported in various clinical studies reflects differences in the preoperative status of the patellofemoral joint,

*References 3, 15, 17, 18, 20–22, 30, 31, 36, 39, 48, 51, 75, 77, 78, 86, 87, 90, 119, 127, 139, 156.

†References 3, 5, 12–14, 34, 85, 100, 109, 122, 156.

Figure 3. *A*, Loss of flexion secondary to nonanatomic graft placement. *B*, Lateral radiograph demonstrates anterior placement of the femoral tunnel. Anterior placement of the femoral tunnel results in the ACL graft lengthening with flexion. An improperly positioned ACL graft can act as a check rein, capturing the knee with a resultant loss of motion.

ACL graft source, surgical technique, postoperative rehabilitation, and differences in the criteria used to define the problem. The origin of anterior knee pain following ACL reconstruction is multifactorial. Reported risk factors include a history of preoperative anterior knee pain, pre-existing articular cartilage injury to the patellofemoral joint, ACL graft source, improper surgical technique, postoperative immobilization, graft impingement, flexion contracture, and aggressive use of open chain exercises.[3, 122, 134, 136, 156] Improvements in surgical technique and preoperative and postoperative rehabilitation have significantly reduced the incidence of anterior knee pain following ACL reconstruction.[136]

Figure 4. *A* and *B*, Intraoperative patellar fracture treated with internal fixation and bone grafting. *C*, Infrapatellar contracture syndrome. Note the anterior placement of both the tibial and femoral tunnels. The patient complained of anterior knee pain and loss of motion. This case illustrates an overlap between extensor mechanism dysfunction and loss of motion in patients with failed ACL surgery.

During the first year after surgery, extensor mechanism dysfunction associated with harvest of the bone-patellar tendon-bone autograft, such as patellar fracture, extensor mechanism rupture, patellar tendinitis, donor site pain, and anterior knee pain secondary to abnormalities of patellar tracking and contracture of the extensor mechanism, are usually manifested. Fortunately, many of these complications can be prevented by proper surgical technique, bone grafting and protection of the patellar harvest site, emphasis on immediate full knee extension, patellar mobilization, prevention of quadriceps muscle shutdown, and avoidance of early open-chain exercises.[25, 37, 122, 132, 136] Clinical studies have shown that anterior knee pain and donor site pain tend to improve with time.[14, 34]

Because the incidence of extensor mechanism dysfunction appears to be higher following patellar tendon grafts compared with hamstring tendon grafts and allografts, selection of an alternative ACL graft in high-risk patients may decrease the incidence of this complication.* Noyes and Barber[98] have suggested that relative contraindications to use of an autogenous patellar tendon graft include a narrow patellar tendon from which an 8- to 10-mm graft cannot be taken without undue compromise of the remaining patellar tendon, malalignment of the extensor mechanism, previous harvest of a autogenous patellar tendon graft, and severe patellofemoral or tibiofemoral osteoarthrosis.

Arthritis

One of the goal of ACL reconstruction is to prevent or delay the development of osteoarthritis. The development of osteoarthritis following ACL reconstruction is related to many factors, including injury to the articular cartilage and menisci sustained at the time of the initial traumatic event, progressive damage to joint and secondary restraints from repeated giving-way episodes prior to surgery, previous meniscectomy, and restoration of functional stability without restoration of normal kinematics and osseous homeostasis (Fig. 5).[38, 40]

At the time of the initial traumatic injury to the ACL, other structures are often damaged. In approximately 80% of acute ACL injuries bone bruises are present.[143, 144] These injuries typically occur at the middle portion of the lateral femoral condyle and the posterolateral aspect of the lateral tibial plateau. Bone bruises are thought to represent trabecular microfractures from blunt trauma and may result not only in injury to the bone marrow but injury to the overlying articular cartilage as well. Although the articular cartilage may not appear to be damaged visibly, a bone bruise may result in changes to the articular cartilage at the biochemical, histologic, and ultrastructural level. These changes may result in future cartilage degeneration even after a successful ACL reconstruction.

Whether the onset or progression of osteoarthritis following an otherwise successful ACL reconstruction should result in the ACL reconstruction being classified as a failure is controversial. Many of the preexisting conditions that may contribute to the development of osteoarthritis cannot be expected to be corrected by an ACL reconstruction, and therefore the final knee rating may not reflect the actual result of the ACL reconstruction.[69, 100] In a patient with recurrent instability and pain secondary to articular cartilage damage, it is important to determine which of these two symptoms is the primary complaint. Revision liga-

*References 5, 74, 75, 77, 78, 85, 103.

Figure 5. Subtotal medial meniscectomy was performed prior to ACL reconstruction. Following ACL reconstruction, the patient had full range of motion, normal AP laxity, and no longer complained of instability; however, the patient was unable to return to their preinjury level of activity because of recurrent swelling and medial compartment pain.

ment surgery in a patient whose primary complaint is pain likely results in continued symptoms and an unsatisfactory outcome.

Recurrent Patholaxity (Graft Failure)

The incidence of graft failure following primary ACL reconstruction has been reported to range from 0.7% to 8%.[53, 56, 57, 68, 74] The patient with recurrent patholaxity usually presents with instability symptoms similar to those experienced before the primary reconstruction. The University of Pittsburgh has developed a classification system in an attempt to define factors that can lead to recurrent patholaxity after primary ACL reconstruction.[69] In this classification system the three general categories responsible for graft failure are (1) errors in surgical technique, (2) failure of graft incorporation, and (3) trauma.

To maximize the chances for success and to avoid repeating errors that led to failure of the primary ACL reconstruction, it is important to identify the etiology of graft failure prior to undertaking revision ACL surgery.

MECHANISMS OF GRAFT FAILURE

Errors in Surgical Technique

Errors in surgical technique are the most frequent cause of graft failure.[68–70, 72, 126, 151, 155, 157, 158] Nonanatomic graft placement is the most common surgical error responsible for failure of the primary ACL graft.* An improperly positioned femoral or tibial tunnel results in excessive length changes of the ACL graft as the knee moves through a range of motion.[44, 47, 55, 94, 138] Because biologic ACL grafts can accommodate only small changes in length before plastically deforming, improper graft placement results in the graft stretching and becoming lax with time, leading to recurrent patholaxity and instability. Alternatively, the graft functions as a check-rein and captures the knee, resulting in a loss of motion.[94] Either one of these situations may result in failure of the ACL reconstruction.

Because the ACL femoral attachment site is close to the axis of rotation of the knee, small changes in the position of the femoral tunnel have a profound effect on graft length-tension relationships.[44, 47, 55, 94, 138] Anterior placement of the femoral tunnel is the most common error in surgical technique (Fig. 6). Incorrect placement of the femoral tunnel is most often caused by the surgeon's failure to adequately visualize the "over-the-top" position. Anterior placement of the femoral tunnel and

*References 66–72, 126, 151, 155, 157.

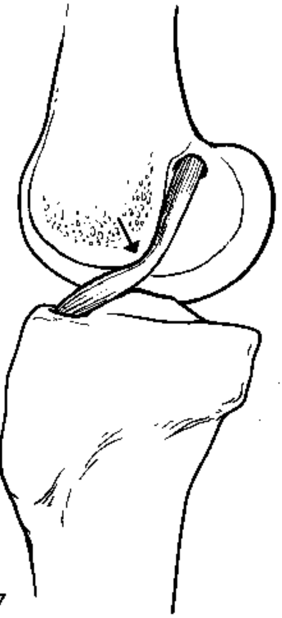
Figure 6. *A*, Anterior placement of the femoral tunnel (error in surgical technique). *B*, Arthroscopic appearance of the failed ACL graft.

fixation of the graft between 0 and 30 degrees of extension results in the graft lengthening and developing increased tension as the knee is flexed.[29, 94] Increased tension in the graft as the knee is flexed can result in graft fixation failure, stretching of the ACL graft with the development of recurrent patholaxity, and loss of flexion with increased stress on the articular surfaces. Conversely, a too posterior placement of the femoral tunnel with tensioning of the graft in flexion results in excessive lengthening of the graft and increased graft tension as the knee is extended.

Although originally thought to be of less importance to the success of ACL surgery, placement of the tibial tunnel has subsequently been shown to have a profound effect on the results of ACL reconstruction.[59, 60, 62] Placement of the tibial tunnel in the eccentric anteromedial position as described by Clancy et al[32] has been shown to cause impingement of the ACL graft against the roof of the intercondylar notch as the knee is extended (Fig. 7). The clinical manifestations of graft impingement include an effusion, loss of extension, and progressive graft failure.[59, 60, 62] A lateral radiograph taken with the knee in maximum hyperextension or an MR imaging scan through the sagittal plane of the ACL graft can be helpful in demonstrating the presence of graft impingement.[59, 62] Graft impingement can be avoided by positioning the tibial tunnel posterior to the slope of the intercondylar roof with the knee in maximum hyperextension.[19, 58, 59, 60, 62] In most knees an impingement-free tibial tunnel can be produced by positioning the center of the tunnel at the junction of the middle and posterior third of the ACL footprint.[65] Knees that have a vertical intercondylar roof or significant hyperextension require the tibial tunnel to be placed in a more posterior location or removal of bone from the roof of the intercondylar notch in order to avoid graft impingement in extension.[19, 58] A too posterior placement of the tibial tunnel, however, can result in excessive laxity of the ACL graft in flexion, or a vertically oriented graft that experiences higher tensile forces and is biomechanically less effective in resisting anterior translation of the tibia. A too medial placement of the tibial tunnel can result in damage to the articular cartilage of the medial tibial plateau and impingement of the graft against the posterior cruciate ligament (Fig. 8). A too lateral placement can result in the graft impinging against the medial aspect of the lateral femoral condyle (Fig. 9).[96]

Inadequate Notchplasty

Most ACL replacement grafts are larger than the size of the original ACL.[23, 54, 104] A large graft placed in a small notch results in the graft impinging against the roof of the intercondylar notch or the inner wall of the lateral femoral condyle.[150] Impingement has been shown to lead to gradual attrition of the ACL graft and eventually graft failure.[62] Impingement can also compromise the biologic incorporation of the graft.[35]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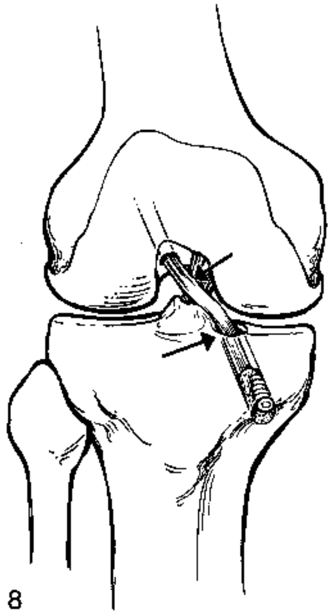
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Figure 7. Anterior placement of the tibial tunnel results in premature contact of the ACL graft with the intercondylar notch as the knee is extended, causing a loss of extension or failure of ACL graft.

Figure 8. Too medial placement of the tibial tunnel can result in damage to the articular cartilage of the medial tibial plateau and impingement of the ACL graft against the posterior cruciate ligament (PCL).

Graft Tension

At the present time, the optimal intraoperative tension that should be applied to the ACL graft is unknown.[8, 26, 29, 162, 163] Optimal graft tension is dependent on a number of factors including the amount of preoperative laxity, the type of graft used, graft placement, the type of graft fixation used, and the knee flexion angle at the time of graft fixation. Of these variables, graft placement and the knee flexion angle at the time of fixation appear to be the most critical.[29] Because ACL grafts do not tighten with time, undertensioning of the graft results in residual patholaxity. Therefore, it is important that the ACL graft be fixed under adequate tension at the time of implantation. Overtensioning of the graft, however, has been associated with delayed graft incorporation, myxoid degeneration, decreased graft strength, and overconstraint of the joint.[163] Overconstraint of the joint may result in a loss of joint motion or increased joint contact pressures, which may accelerate joint wear and lead to osteoarthritis.[125]

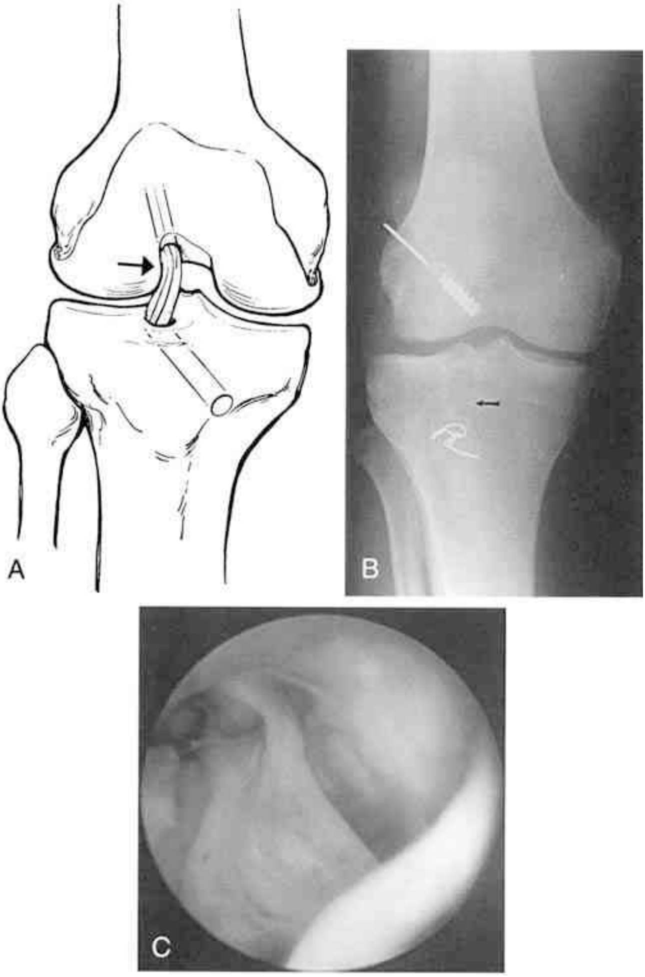

Figure 9. *A*, Lateral placement of the tibial tunnel can result in the ACL graft impinging against the medial side of the lateral femoral condyle. *B*, Lateral placement of the tibial tunnel *(arrow)*, resulting in graft impingement against the inner wall of the lateral femoral condyle and subsequent graft failure. *C*, Arthroscopic appearance of the failed ACL graft.

Graft Fixation

The initial graft fixation strength must be secure enough to prevent graft elongation at the graft fixation sites until the fixation sites are healed.[28] Graft fixation strength depends on the type of graft, the type of fixation device used, and bone quality at the fixation sites.[28] Interference screw fixation has been demonstrated to be the strongest and stiffest fixation device for patellar tendon grafts.[80, 149] Potential pitfalls of

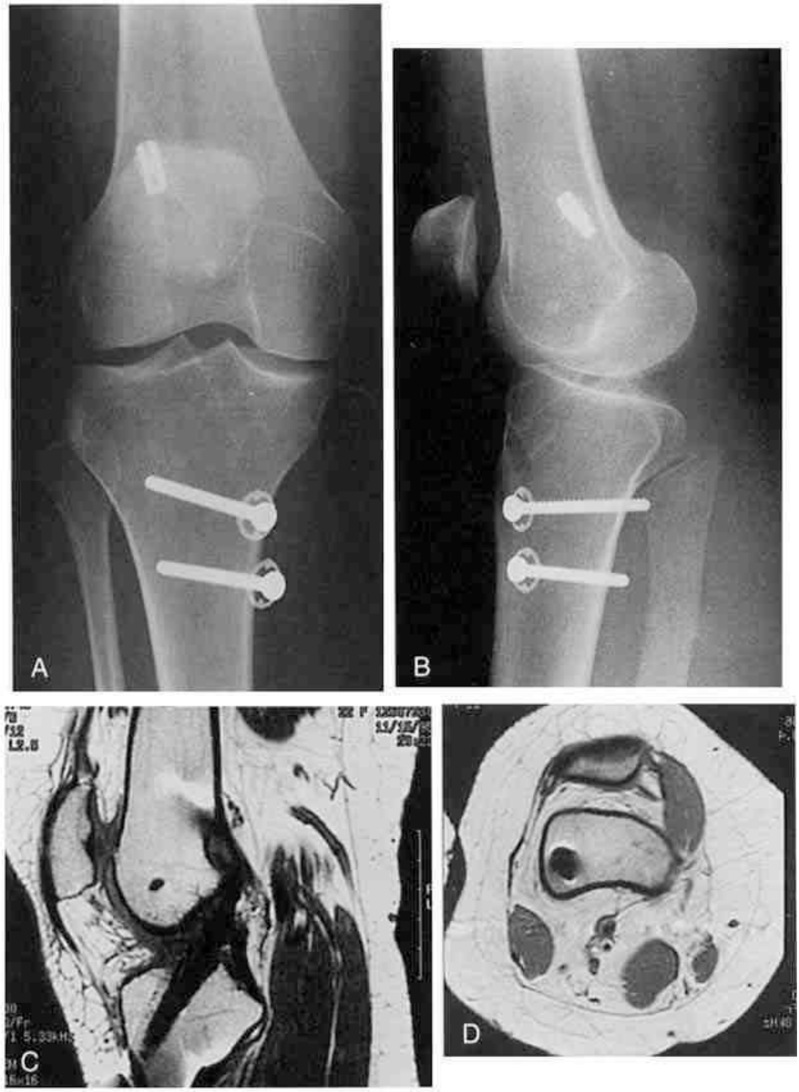

Figure 10. *A* and *B*, Recurrent patholaxity secondary to graft fixation failure. ACL reconstruction was performed with doubled semitendinosus and gracilis tendons fixed in the femur with a Mitek Ligament Anchor (Mitek Surgical Products, Inc., Norwood, MA). According to the patient, the knee never felt stable after surgery. Examination 6 weeks postoperatively revealed +3 Lachman and a +3 pivot shift. AP and lateral radiographs demonstrate correct placement of the femoral and tibial tunnels. *C*, Sagittal MR imaging demonstrates proper placement of the tibial and femoral tunnels. *D*, Axial MR imaging demonstrates the ligament anchor to lie inside the femoral cortex. Fixation failure (error in surgical techniques) resulted from failure of the ligament fixation device to anchor on the cortex of the lateral femur. The ligament anchor migrated through the cancellous bone, resulting in a loss of fixation.

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interference screw fixation include graft-tunnel mismatch, nonparallel screw placement, bone block fracture, graft laceration, laceration of the tensioning sutures, and loss of fixation in osteopenic bone.[11, 33, 89] If unrecognized at the time of the primary ACL reconstruction, any of the previous conditions could compromise graft fixation strength and lead to an early failure of the ACL reconstruction.[33]

Although the fixation strength of hamstring tendon grafts has been reported to equal or exceed that of patellar tendon grafts fixed with interference screws, many hamstring tendon graft fixation techniques have been demonstrated to have longer elongations to failure and less stiffness.[24, 149] Aggressive rehabilitation of a hamstring ACL reconstruction in which a more compliant type of fixation has been utilized (sutures tied around a post) could result in excessive graft elongation and early graft failure. Regardless of the type of graft and the method of fixation used, the graft fixation device must be properly inserted and must prevent loss of the initially applied graft tension until the fixation sites heal (Fig. 10).

Failure to Recognize or Address Associated Instabilities

Failure to recognize or treat secondary restraints to anterior tibial translation can subject the newly reconstructed ACL to increased tensile forces, which may result graft failure. Schepsis et al[126] reported that

Figure 10 *(Continued). E,* Because the anchor was buried in the distal femur and could not be removed easily without a significant loss of bone stock, the revision procedure was performed using a patellar tendon graft placed in the over-the-top position. The bone block *(arrows)* was fixed to the femur by tying sutures around an AO cancellous screw and a washer.

failure to recognize or address associated ligamentous instability at the time of the primary ACL reconstruction was the origin of failure in 15% of the failed ACL reconstructions that they revised. In this series, associated instabilities were the second most frequent error in surgical technique leading to failure of the primary ACL reconstruction (nonanatomic tunnel placement was the number one error).

Posterolateral instability is probably the most common unrecognized and untreated associated patholaxity. Gersoff and Clancy[45] have estimated that associated posterolateral laxity is present in 10% to 15% of chronic ACL deficient knees. Untreated posterolateral instability may result in continued complaints of the knee "giving-way backward" owing to increased hyperextension and varus recurvatum.[97] Posterolateral instability may also result in failure of the ACL reconstruction secondary to the excessive tensile forces placed on the ACL graft as a result of the tendency for the knee to go into hyperextension and lateral joint opening.[97]

In a study of chronic ACL-deficient knees reconstructed with a central-third patellar tendon graft, O'Brien et al[109] noted that 17 of 19 knees with greater that a 3-mm side-to-side KT-1000 difference had associated ligamentous instabilities. Of these 17 knees, 11 had increased external tibial rotation at 30 degrees of flexion. Increased external tibial rotation at 30 degrees of flexion has been shown to be the most sensitive test for injury to the posterolateral structures.[46, 50, 107] Based on their results, O'Brien et al[109] recommend that an increase in external tibial rotation of more than 10 degrees compared with the opposite side be addressed at the time of ACL reconstruction.

Unrecognized or untreated injury to the medial ligamentous structures may also result in failure of the primary ACL reconstruction. The superficial medial collateral ligament (MCL), posterior oblique ligament (POL), and the posterior horn of the medial meniscus are secondary restraints to anterior tibial translation on the medial side of the knee.[94, 107, 114] The popularity and ease of arthroscopy-assisted ACL reconstruction, along with the high incidence of stiffness reported following combined ACL reconstruction and repair of the medial structures, has led to a de-emphasis on surgical repair of the medial structures.[133, 134]

Although good clinical results have been reported with nonoperative treatment of grade III MCL tears in knees with combined ACL and MCL injuries, it should be recognized that not all grade III medial sided injuries have such a favorable result.[16, 135] A grade III MCL tear associated with a complete tear of the POL and the meniscofemoral and meniscotibial ligaments results in the loss of the important "brake-stop" effect of the posterior horn of the medial meniscus.[94] Failure to restore this important brake-stop mechanism by repair of the posteromedial structures may subject the ACL graft to increased forces and result in graft failure.

Graft Material

The type of graft used to perform the primary ACL reconstruction may also play a role in the failure of the reconstruction. At the present time, the central-third bone-patellar tendon-bone graft is the most widely used autograft to replace a torn ACL. One advantage of the patellar tendon is the ability to obtain rigid initial fixation at both ends of the graft using interference screw fixation of the bone blocks.[80, 149] Harvest of undersized or poor quality bone blocks, however, may provide inadequate purchase for the interference screw, compromising graft fixation.

Although initially thought to be too weak, recent studies have demonstrated that equally tensioned four-stranded hamstring tendon grafts are the strongest and stiffest autografts currently available.[23, 54] Use of single-stranded hamstring grafts or unequally tensioned four-stranded hamstring grafts in the chronic ACL-deficient knee with lax secondary restraints, however, may provide inadequate initial graft strength, potentially leading to failure of the primary reconstruction.[23, 74]

The use of allograft tissue offers many advantages including decreased surgical time, smaller incisions, less surgical dissection, variable graft sizes and shapes, and no donor site morbidity.[123, 128] At the present time, however, most allograft tissue is irradiated to decrease the risks of disease transmission.[128] The dose of radiation required to neutralize the AIDS virus has been demonstrated to weaken the graft by approximately 27%.[118] Use of irradiated allografts has been associated with a higher rate of unacceptable arthrometer results in chronic ACL-deficient knees.[98, 123] Histologic and biomechanical studies have also demonstrated that biologic incorporation of allograft tissue is delayed compared with autograft tissue.[66] The secondary effects of irradiation and delayed biologic incorporation may place allograft ACL reconstructions at increased risk for failure, particularly in the chronic ACL-deficient knee.

Failure of Graft Incorporation

The ultimate success of any biologic ACL replacement graft depends on the ability of the replacement tissue to survive and maintain its initial biomechanical properties in the intra-articular environment of the knee, and incorporate with the host. Graft incorporation is known to be influenced by various mechanical factors, such as graft placement, graft impingement, graft tensioning, stress shielding of the graft, and deleterious stresses applied to the graft in the early healing phase.[35, 69] Little is known, however, about the biologic variables that control the rate and extent of ACL graft incorporation.

Experimental studies have shown that both autograft and allograft tissues undergo the same biologic process of graft incorporation, which consists of graft necrosis, revascularization, cellular repopulation with cells of extrinsic origin, collagen deposition, and graft maturation and remodeling.[6, 7, 10, 66] This complex biologic healing response has been called *ligamentization* because it results in a replacement structure that grossly resembles the normal ACL.[6, 7] Jackson et al[66] using a goat model have demonstrated that the time course and extent of graft remodeling are slower and less complete in allografts compared with autografts. In this study the allografts were also found to be inferior biomechanically to autografts. The biologic factors responsible for the delayed graft incorporation of allografts have not been identified at the present time.

Trauma

Factors that can lead to traumatic failure of the primary reconstruction include overaggressive rehabilitation, premature return to athletics before graft incorporation is completed and neurophysiology control of the lower extremity is re-established, and a significant reinjury after initial functional stability was restored and full activities were resumed. The common factor responsible for traumatic graft failures is the inability of the ACL graft to withstand the tensile loads applied to it during the particular stage when it is injured.

Although probably not the most common cause of traumatic failure, an overaggressive rehabilitation program can apply tensile loads that may injure or stretch the immature graft during the early healing period. An overaggressive rehabilitation program can also result in fixation-site failure and an early loss of stability. Traumatic graft failure may also occur when a patient attempts to return to strenuous activities before the graft has incorporated or neurophysiologic control of the leg is re-established. Although the use of an accelerated rehabilitation program has significantly reduced postoperative and donor site morbidity, there are concerns that early unrestricted activities, such as running, and sports-specific activities put the immature ACL at risk and may result in a higher long-term graft failure rate.[132]

The incidence of traumatic reruptures has been reported to range from 2.2% to 2.7%.[34, 130] The traumatic event is usually similar to the initial ACL injury, often with a "pop," immediate hemarthrosis, and a increase in anteroposterior laxity.

PREOPERATIVE EVALUATION

Preoperative evaluation is one of the most important aspects of revision ACL surgery. First and foremost, it must be determined if the previous surgery has truly failed. Because of the different categories of failure and overlap between them, determining whether the patient's residual complaints are primarily caused by graft failure can at times be very difficult. Current indications for revision ACL surgery include instability with activities of daily living or athletic activities, and the presence of pathologic anterior laxity on clinical examination that reproduces the patient's sensation of giving-way. Both criteria must be met before proceeding with surgery.

History

A thorough evaluation first involves a detailed patient history. The sequence of events leading up to the patient's presentation should be examined. Such information may shed light on the possible mechanism of failure. Patients who present with a gradual onset of instability after initially having a stable knee may have graft failure secondary to gradual stretching of the ligament secondary to nonanatomic graft placement, or attrition of the ligament secondary to impingement. Patients who state that the knee was never stable following the primary surgery should be suspected of having a failure of graft fixation, or more commonly a failure to address associated ligamentous laxity. The history leading up to the previous types of failures is quite distinct from the patient who has returned to their preinjury level of activity and has sustained graft failure secondary to the onset of new trauma.

The surgeon must clearly differentiate the patient's chief complaint: pain versus instability. Patients with either complaint may demonstrate increased laxity on physical examination; however, each complaint has a different prognosis and treatment. Instability can often be improved by revision ligament surgery; however, pain, which is usually associated with injury or damage to articular cartilage, must be addressed separately or simultaneously by microfracture, osteochondral grafting, chondrocyte transplantation, meniscal allograft, or osteotomy.

Review of the patient's previous medical record is especially important. Relevant information that should be obtained by reviewing the patient's medical records includes the previous examination under anesthesia (associated ligamentous laxity); surgical technique (endoscopic versus two-incision); graft source; associated pathology; type, size, and manufacturer of the hardware used for graft fixation; and possible intraoperative complications that may have occurred. As part of the chart review, the postoperative period and rehabilitation protocol should be examined critically. Potential factors in the postoperative period that may have contributed to failure of the primary surgery include an overly aggressive rehabilitation program, returning to sporting activities before neuromuscular control of the lower extremity was established, problems regaining full range of motion, development of a reflex sympathetic dystrophy, and infection.

Physical Examination

A comprehensive physical examination including an evaluation of the entire lower extremity must be performed. The overall alignment of the lower extremity and gait pattern (varus thrust) should be examined. Varus alignment can lead to excessive loads on the reconstructed ACL, leading eventually to graft failure. In these cases, a tibial osteotomy should be performed before or in conjunction with the revision ligament surgery. The patient' range of motion should be assessed. The heel height difference and heel-to-buttock distance are useful ways to measure the loss of extension and flexion.[122] A staged procedure should be considered if there is a loss of extension sufficient enough to result in anterior knee pain, quadriceps muscle weakness, or a bent-leg gait pattern.

The ACL status of the primary reconstruction should be assessed using the Lachman test, anterior drawer test, pivot-shift test, and KT-1000 arthrometer testing. The secondary restraints must also be closely evaluated. The medial secondary restraints to anterior tibial translation include the superficial MCL, the POL, and the posterior horn of the medial meniscus. The medial structures are evaluated by comparing valgus rotation at 0 and 30 degrees of flexion, and external tibial rotation at 30 degrees and 90 degrees of flexion to the opposite normal knee.[114] Similarly, the lateral and posterolateral structures are evaluated by comparing varus rotation at 0 degrees and 30 degrees of flexion, and external tibial rotation at 30 degrees of flexion to the opposite normal knee.[46, 50, 107] Failure to recognize and treat associated ligamentous laxities may result in failure of the revision ACL reconstruction.

The location of previous skin incisions should be examined carefully. The primary graft may have been harvested through a vertical, oblique, or transverse incision. Based on the length and orientation of the original harvest incision, the surgeon must decide whether it is possible to harvest the revision graft through the original incision or whether a new incision is required.

Radiographic Examination

Radiographic assessment should include a complete knee series: standing anteroposterior, 45-degree posteroanterior flexion weight bearing, lateral with the knee in maximum hyperextension, notch, and Merchant views. These radiographs help assess the overall status of the knee: limb alignment, degenerative joint changes, bone quality, type and placement of hardware, and tunnel placement and enlargement.

Other imaging modalities that may be utilized in selected cases include bone scintigraphy, CT scanning, and MR imaging. Bone scintigraphy can be used to determine osseous homeostasis and may be of help detecting infection and early radiographically silent arthritis.[40] CT is helpful in defining the extent of tunnel enlargement and osteolysis. Tunnel enlargement has been most commonly associated with synthetic grafts and allografts.[49, 140] Information about the size of the bone tunnels is useful in terms of planning graft selection, and whether to bone graft the enlarged tunnels concurrently or as part of a staged procedure (Fig. 11). MR imaging with sagittal images through the plane of the ACL graft can be helpful in assessing integrity of the ACL graft. In most cases, however, clinical examination and plain radiographs are sufficient to determine the status of the primary ACL graft, and MR imaging is necessary in only selected cases.

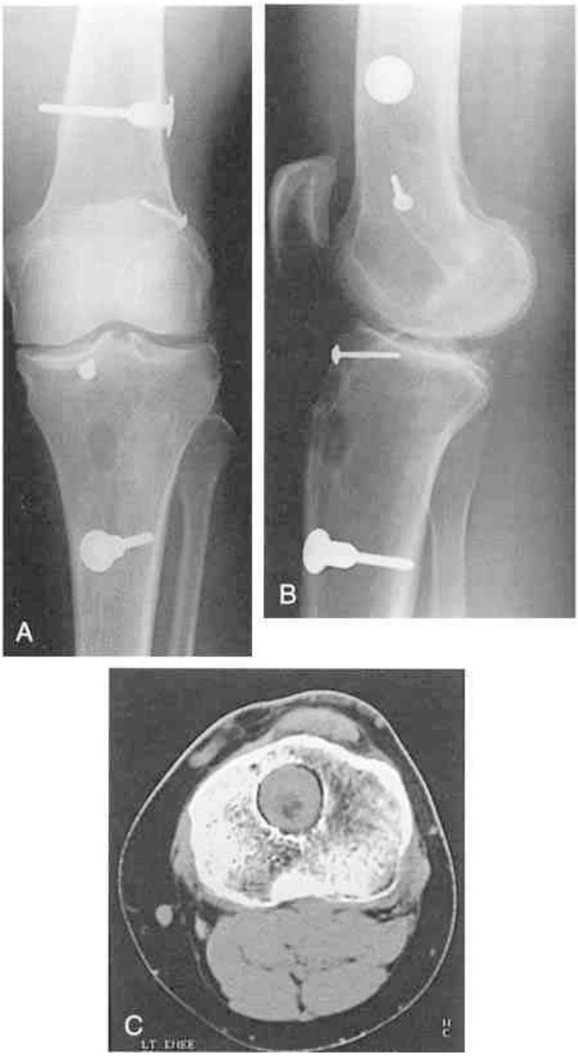


Figure 11. *A* and *B*, Failed synthetic ACL reconstruction. The first procedure was an autogenous patellar tendon reconstruction that failed. (Note the anterior placement of the tibial and femoral tunnels.) A revision procedure was performed using an artificial ligament. Note the marked enlargement of the tibial tunnel on the AP radiograph. *C*, CT scan demonstrates marked enlargement of the tibial tunnel. This case requires a two-stage approach. Stage one is removal of the artificial ligament and bone grafting of the bony defects. Following consolidation of the bone graft, residual instability is addressed by ligament reconstruction.

PREOPERATIVE PLANNING

Once the preoperative evaluation has been completed, the surgeon should have determined the origins of the primary failure and determined whether the patient is a candidate for revision ligament surgery. Patient compliance and motivation are important factors that are critical to the success of revision ACL surgery. If revision ACL surgery is recommended, the patient must be given a realistic expectation of the expected outcome, and not be promised too much. In general, the results of revision ACL surgery have been favorable with regards to improving stability; however, the results are not equivalent to those of primary ACL surgery, particularly when evaluating return to preinjury levels of activity.[71, 72, 103, 117, 126, 151, 157, 158] Given its complexity, revision ACL surgery should be considered salvage surgery. False expectation of the revision surgery may lead to a subjective failure by the patient despite a technically successful procedure.

The success of revision ACL surgery is influenced by the etiology of the primary failure, the preoperative laxity of the knee, the status of the menisci, articular cartilage, and secondary restraints. The primary goals of revision ACL surgery are to stabilize the knee, prevent further damage to the menisci and articular cartilage, and maximize the functional level of the patient.

Important factors to determine from the preoperative assessment include the range of motion of the knee, the placement of previous incisions, the type of graft used in the primary reconstruction, the type and location of graft fixation hardware, the size and location of the bone tunnels, and the presence of any associated ligamentous patholaxities. A staged procedure is recommended if there is a loss of more than 5 degrees of extension or 20 degrees of flexion. Loss of motion should be addressed with physical therapy, arthroscopic or open releases, and manipulation with the goal of obtaining a painless functional range of motion prior to consideration of revision ACL surgery. If tunnel enlargement has resulted in large bony tunnels that interfere with the placement of new tunnels or fixation of the revision ACL graft, then bone grafting of the defects should be performed as the first procedure and revision ligament surgery delayed until the bone graft has incorporated.

Graft Selection

Graft selection for the revision procedure depends on the type of graft used for the primary reconstruction, the placement of incisions used to harvest the primary graft, the presence of enlarged bone tunnels, and the presence of associated ligamentous laxity. Synthetic ligaments are not currently recommended for primary or revision ACL surgery because of the high complication and failure rates reported with their use.[49, 140] At the present time, graft selection options for revision ACL surgery consist of autograft or allograft tissue.

Autogenous graft options include patellar tendon, quadriceps tendon, hamstring tendons, and reharvest of the ipsilateral patellar tendon. The patellar tendon and Achilles tendon are the most commonly used allografts (Fig. 12). The advantages of autograft tissues include elimination of the risk of disease transmission, no addition cost, elimination of possible immune reactions, and superior biologic incorporation. The major disadvantages of autograft tissue are donor site morbidity, limitation of the size and number of grafts available, and the increased surgical dissection required to harvest the graft tissue. The advantages of allograft tissue are the lack of donor site morbidity, the availability of variable graft shapes and sizes, the ability to customize bone blocks to accommodate enlarged bone tunnels, and the need for smaller incisions and less surgical dissection.

Concerns about allograft tissues have focused primarily on the issues of disease transmission, the effects of secondary sterilization on the initial mechanical properties of the graft tissue, and the possibility of an immune response. The risk of disease transmission is probably the major deterrent to the routine use of allograft tissue. Viruses that transmit disease are not killed by freezing the allograft tissue. In an attempt to eradicate viruses, allograft tissue is commonly secondarily sterilized with radiation (1.5 to 2.5 Mrad). This dose of radiation, however, has been shown to alter the collagen structure and reduce the tensile strength of the allograft tissue.[118]

Although animal studies have demonstrated that autograft and allograft tissues undergo the same healing process, the time course for allografts appears to be prolonged, and the biologic response is less robust than seen with autograft tissue.[66] Clinical results of primary ACL reconstructions performed with irradiated allograft tissue, and chronic ACL reconstructions performed with nonirradiated allograft tissues have in general been favorable.[70–72, 99] The results of irradiated allografts used

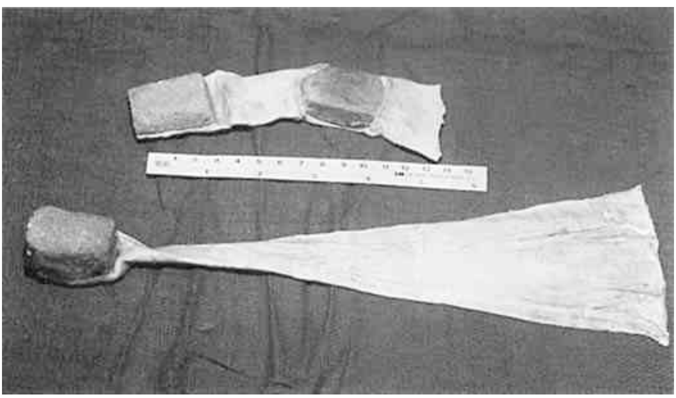


Figure 12. Patellar tendon and Achilles tendon allografts. Both grafts come with large bone blocks that can be fashioned to fill enlarged bone tunnels.

in chronic ACL-deficient knees, however, appear to be inferior to those of autografts used in acute or chronic knees, or nonirradiated allografts.[70–72, 98, 158] Because of delayed biologic incorporation, allografts are probably contraindicated in revision cases where failure of the graft to incorporate was the etiology of the primary failure.

Because the patellar tendon is the most commonly used autograft for primary ACL reconstruction, this type of reconstruction is also the most common type of ACL reconstruction requiring revision surgery. Autograft options for revision of a failed patellar tendon reconstruction include (1) ipsilateral hamstring tendons, (2) ipsilateral quadriceps tendon, (3) reharvest of the ipsilateral patellar tendon, and (4) harvest of the contralateral patellar tendon.

Although concerns have been raised about the tensile properties of hamstring tendon grafts, recent studies have demonstrated that hamstring tendon grafts are stronger and stiffer than 10-mm patellar tendon and 10-mm quadriceps tendon grafts.[23, 54] The tensile properties of hamstring tendon grafts in the age range (23 to 43 years) of patients who typically undergo ACL reconstruction have recently been investigated by Hecker et al.[54] This study reported a mean failure load of 3560 ± 742 N, and a stiffness of 855 ± 156 N/m for quadrupled semitendinosus grafts, and a mean failure load of 4140 ± 969 N, and stiffness of 807 ± 164 N/mm for combined doubled semitendinosus and gracilis grafts, values significantly higher than those of 10-mm patellar tendon grafts. The donor site morbidity of hamstring tendons has also been reported to be slight (Fig. 13).[84, 161]

Potential disadvantages of hamstring tendon grafts include a smaller diameter compared with patellar tendon grafts and the lack of bone blocks at the ends of the graft. The typical tunnel size for patellar tendon reconstruction is between 9 and 11 mm, compared with 7 to 9 mm for four-stranded hamstring tendon grafts. Because of the smaller diameter of hamstring tendon graft bone tunnels, it is not possible to overdrill the existing patellar tendon bone tunnel into virgin bone.

If the surgeon desires to use a hamstring tendon graft in a knee in which the placement of the existing patellar tendon bone tunnels is satisfactory, either the extra-articular position of the new bone tunnels must be diverged from the path of the pre-existing bone tunnels, or the pre-existing bone tunnels overdrilled, bone grafted, and the reconstruction performed as a two-stage procedure. By converting to a two-incision technique in cases where the primary ACL reconstruction was performed using an endoscopic technique, or converting to an endoscopic technique when the primary ACL reconstruction was performed using a two-incision technique, it is usually possible to diverge the femoral tunnels (Fig. 14).[124] The tibial tunnel can either be diverged by changing the medial-lateral position, or the inclination angle of the tibial aiming device. Intraoperative radiographs or fluoroscopy can be used to confirm guidepin placement prior to drilling the new tunnels.

A second potential disadvantage of hamstring tendon grafts is their inability to fill bony defects. In cases of femoral tunnel enlargement, one

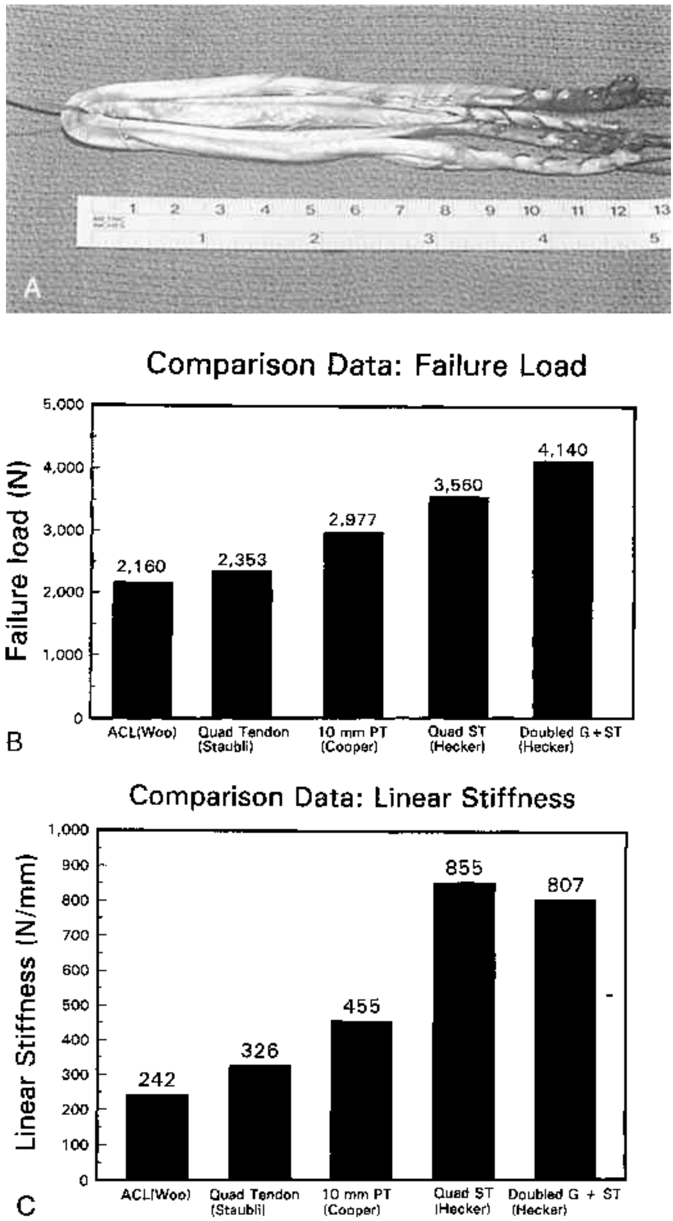


Figure 13. *A*, Doubled gracilis and semitendinosus graft. The combined grafts are typically 7–9 mm in diameter. In our biomechanical studies, the cross-sectional area of doubled gracilis and semitendinosus grafts varies from 40–53 mm^2.[54] *B* and *C*, Mean failure loads and linear stiffness reported in young patients for commonly used ACL replacement grafts.

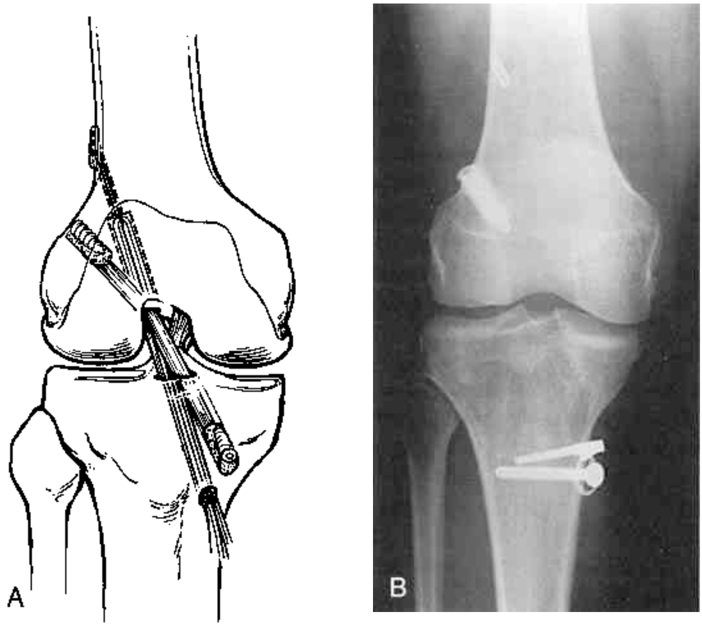


Figure 14. *A*, Because a rear-entry femoral tunnel has a more horizontal orientation, it is usually possible to drill a new divergent femoral tunnel using a transtibial approach. This can often eliminate the need to remove the original femoral fixation device. Similarly, it is possible to diverge a well-placed femoral tunnel that was drilled using a transtibial approach by converting to a two-incision or rear-entry technique. In the case of a well-positioned tibial tunnel, a new divergent tibial tunnel can be drilled by changing the external starting position. *B*, Original procedure was performed with an autogenous patellar tendon graft using a rear-entry technique. The revision procedure was performed using an 8-mm doubled-loop gracilis and semitendinosus autograft. The revision femoral tunnel was drilled using a transtibial approach. The femoral side of the hamstring tendon graft was fixed with an EndoButton (Smith & Nephew Endoscopy, Andover, MA). Because the new tunnel diverged from the original rear-entry femoral tunnel, it was not necessary to remove the original femoral interference screw.

has the option of using the "over-the-top" position, thereby avoiding the need to drill a bone tunnel. In cases where the tibial tunnel is enlarged, however, bone grafting and a staged reconstruction are usually required.

A third potential disadvantage of hamstring tendon grafts is the longer elongation to failure and lower stiffness of many hamstring graft fixation techniques.[24, 149] Our recent biomechanical testing has demonstrated, however, that the stiffness and elongation to failure of doubled gracilis and semitendinosus grafts fixed in the femur with bioabsorbable interference screws, the Bone Mulch Screw (Arthrotek, Ontario, CA), TransFix (Arthrex, Naples, FL), LinX-HT (Innovasive Devices, Marlborough, MA), and EndoButton with continuous polyester loop (Smith & Nephew Endoscopy, Andover, MA) to be similar to those previously reported for patellar tendon grafts fixed with interference screws.[80, 144] Although the effects of initial graft fixation stiffness on the outcome of

ACL reconstructions remain unknown, good long-term results have been reported in patellar tendon reconstructions utilizing a graft fixation technique that has been demonstrated to have stiffness values similar to those reported for some hamstring tendon reconstructions.[24, 32, 130–132, 149] It is our feeling that lower-stiffness hamstring fixation techniques require a longer period of cyclical loading prior to graft fixation, higher initial graft tension, and fixation at 20 to 30 degrees of flexion. At the present time there have been no published clinical results on the use of hamstring tendons for revision ACL surgery.

Stäubli[146–148] has recently refocused attention on the use of the quadriceps tendon as an alternative graft source for primary and revision cruciate ligament surgery. Biomechanical testing has shown that the quadriceps tendon is about the same strength as the patellar tendon but has lower initial stiffness.[148] The cross-sectional area of the quadriceps tendon, however, is significantly greater than that of the patellar tendon (quadriceps tendon = 65 mm^2; patellar tendon = 36.8 mm^2). the large cross-sectional area of this graft source and the presence of a bone block at one end make it possible to fill enlarged bone tunnels, and also allows for the possible correction of graft misplacement by eccentric positioning of the bone block in an existing bone tunnel. The donor site morbidity is said to be less than that of the patellar tendon; however, there are no published reports that document this. One area of potential concern about this graft source is that harvest of a second bone block from the superior pole of the patella may place the patella at risk for fracture in revision cases. Purnell and Lamoreaux[117] have reported good results in 14 patients with an average follow-up of 3.5 years using a 9- to 11-mm width quadriceps tendon graft to revise failed primary ACL reconstructions. All knees were reported to have less than 2-mm side-to-side difference using the KT-1000 arthrometer, the hop test averaged 94%, and the isokinetic strength testing at 240 degrees per second and 60 degrees per second averaged 82%. Complications included one nondisplaced patellar fracture 1 year after surgery (Fig. 15).

Reharvest of the patellar tendon from the ipsilateral knee is another potential option for revision of a failed primary patellar tendon reconstruction. Proctor et al[116] using a goat model have demonstrated that the patellar tendon donor site fills with scar repair tissue, and the tensile properties of this new tissue are significantly reduced compared with the normal contralateral patellar tendon at 21 months postoperative. Similar findings have been reported in a dog model by LaPrade et al.[82] In this study it was reported that the failure load and energy to cause failure of the reharvested patellar tendons at both 6 and 12 months was significantly less than that of the control contralateral tendon. As a result of these findings, both authors have recommended that alternative grafts be used for revision ACL surgery.

Kartus et al[76] reported the clinical results using the reharvested patellar tendon to perform revision ACL surgery in 20 patients. The ipsilateral patellar tendon was reharvested and used as the revision ACL graft source in 10 patients, and the contralateral patellar tendon was used in another 10 as a control. The Lysholm score, IKDC rating, and

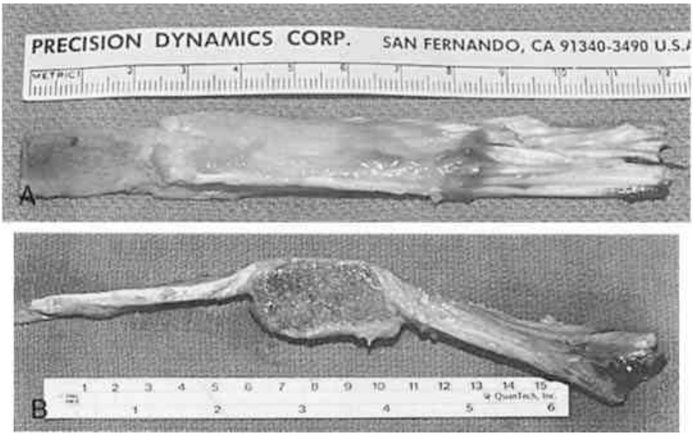


Figure 15. *A*, Eleven-millimeter quadriceps tendon graft. *B*, Human cadaveric cross section of the extensor mechanism. Note the increased thickness of the quadriceps tendon compared to the patellar tendon. According to Staubli et al, the mean cross-sectional area of a 10-mm–wide quadriceps tendon is 65-mm^2 versus 36.8-mm^2 for a 10-mm patellar tendon graft.[146]

Tegner activity levels were reported to be significantly lower in the patients with reharvest of the ipsilateral patellar tendon. One patellar fracture and one patellar tendon rupture were reported to have occurred in the reharvest group. Because of the significantly lower functional scores and 20% incidence of major donor site complications in the reharvest group, the authors do not recommend the use of the reharvested ipsilateral patellar tendon for revision ACL surgery.

Use of the contralateral patellar tendon provides the same advantages as that of the primary patellar tendon. Use of the contralateral patellar tendon, however, carries the risk of creating a problem in a knee that was previously normal. Rubinstein et al[121] reported on the use of the contralateral patellar tendon as a graft source in 26 patients undergoing cruciate ligament surgery and found that all patients had regained full range of motion by 3 weeks, and that quadriceps strength had returned to 93% at 1 year and 95% at 2 years postoperatively in the donor knee. No patient complained of patellofemoral pain in the donor knee; however, patellar tendinitis occurred in 55% of the patients during the first year, but was said to be rarely restricting and to resolve after the first year. Based on their experience, the authors felt that the donor site morbidity from harvesting the contralateral patellar tendon was of short duration and largely reversible.

Autograft options following a failed hamstring primary ACL reconstruction include (1) ipsilateral patellar tendon, (2) ipsilateral quadriceps tendon, and (3) contralateral hamstring tendons.

In most cases the graft of choice is the ipsilateral patellar tendon. In cases with tunnel enlargement, however, the larger cross-sectional area of the quadriceps tendon may be advantageous.

TECHNICAL CONSIDERATIONS OF REVISION ACL SURGERY

Skin Incisions

Careful planning of skin incisions around the knee is needed to avoid wound healing problems. Meticulous surgical technique and handling of the soft tissues is critical. In general, previous incisions should be used or extended if they allow simultaneous hardware removal, graft harvest, and proper placement and fixation of the new graft.

Old vertical incisions can be extended proximally or distally to harvest either a patellar tendon or hamstring tendon graft for the revision procedure (Fig. 16). Extension of the old vertical incision also allows for removal of the tibial fixation hardware, drilling of the tibial tunnel, and tibial fixation. Short transverse incisions can be crossed with a vertical incision (Fig. 17).

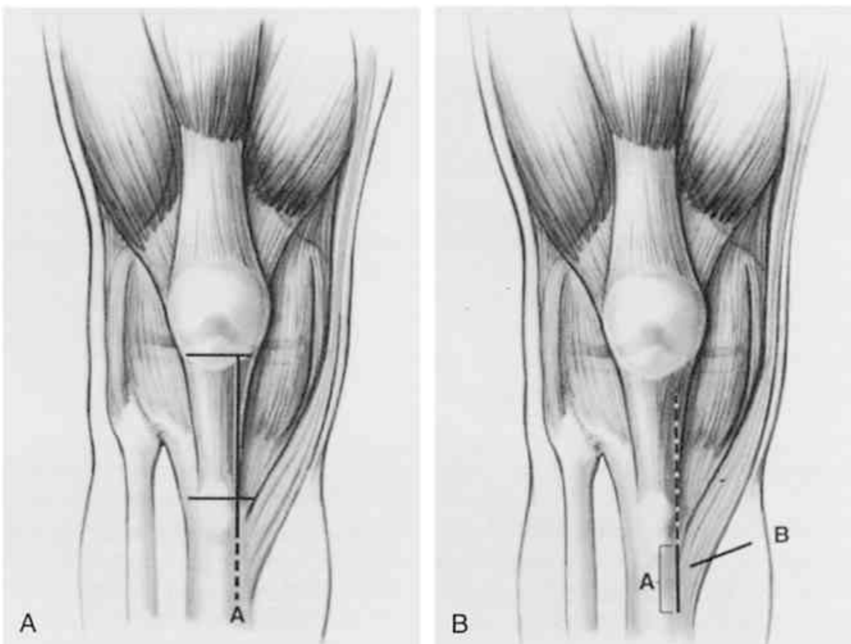


Figure 16. *A*, Hamstring tendon grafts can be harvested, and tibial fixation hardware removed, by extending the lower portion of the vertical patellar tendon harvest incision distally over the pes anserinus, *dashed line (A)*. A short transverse patellar tendon harvest incision can be crossed by a vertical incision to harvest the hamstring tendons and remove tibial fixation hardware. *B*, Patellar tendon graft can be harvested by extending original vertical hamstring harvest incision *(A)* proximally *(dotted line)*. If the hamstring tendons were harvested through a transverse incision *(B)*, a new vertical incision *(dotted line)* can be used to harvest the patellar tendon graft.

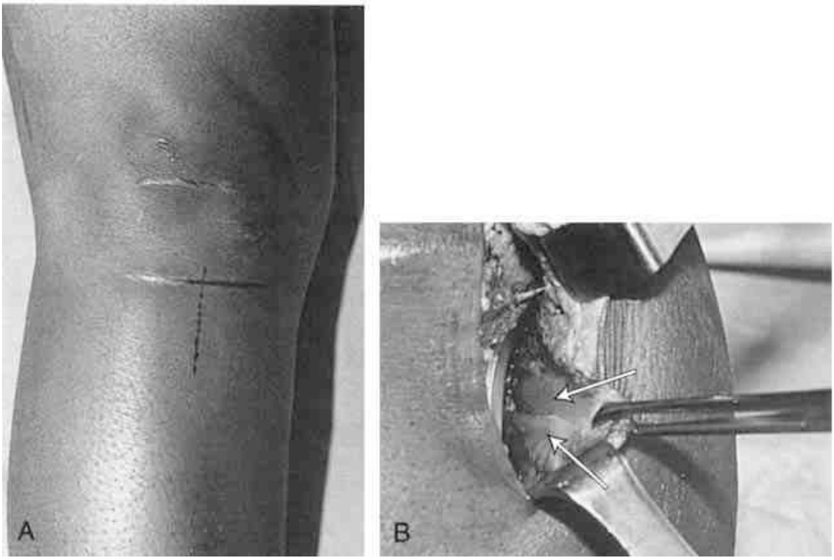


Figure 17. *A*, Patellar tendon graft for the primary reconstruction was harvested through two small transverse incisions. The inferior transverse incision *(solid line)* can be crossed with a short vertical incision *(dashed line)* to harvest the hamstring tendons, and remove the tibial fixation hardware. *B*, Exposure of the gracilis (upper tendon), and semitendinosus tendon (lower tendon) *(arrows)*.

In cases where multiple skin incisions were used to perform the primary procedure, or in situations where the skin viability is in doubt, the use of allografts can minimize subcutaneous dissection and the creation of large skin flaps. When harvesting hamstring tendons layer 1, the sartorius fascia, should be preserved and closed over the tibial fixation hardware.

Hardware Removal

During the preoperative planning phase the surgeon should review the surgeon's dictated operative note as well as the nursing notes to determine the type, size, and manufacturer of the fixation devices used during the primary reconstruction so that appropriate instrumentation to remove the hardware is available at the time of the revision procedure.

The surgeon should be aware that special extraction devices may be required to remove hardware in reconstructions performed outside of the United States. Special equipment to remove a stripped or buried screw should also be available at the time of the revision procedure (ACL ReDux Instrumentation, Smith & Nephew Endoscopy, Andover, MA). To seat the extraction device properly, complete removal of all soft tissue and bone from around the implant is required. Failure to do so

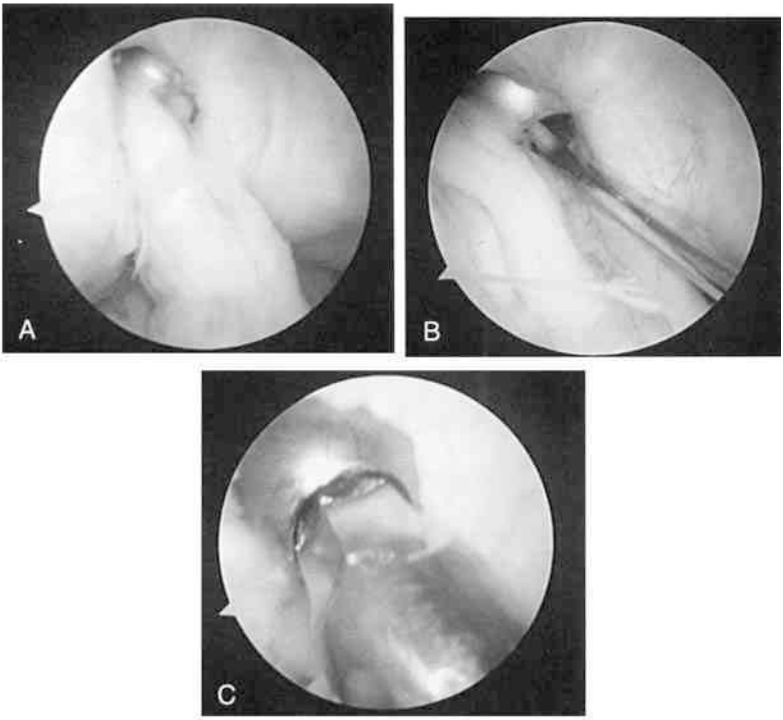


Figure 18. *A*, Failed ACL graft, secondary to anterior placement of the femoral tunnel (error in surgical technique). Note the soft tissue covering the head of the screw. *B*, Bone pick (ACL ReDux instrumentation, Smith & Nephew Endoscopy, Andover, MA) is used to clear soft tissue out of the head of the interference screw. *C*, The corresponding screw driver must be completely engaged in the head of the interference screw to prevent stripping of the screw.

may result in the hardware being stripped, in which case extensive bone removal may be required to remove the implant (Fig. 18). Removal of bent staples can be extremely challenging.

Pre-existing hardware can often be left in place unless it interferes with the revision tunnels, fixation of the graft, or is loose. In general, tibial fixation devices require removal in order to drill a new tibial tunnel and adequately fix the new graft. If the primary reconstruction was performed using a two-incision technique, the femoral fixation device can often be left in place by drilling a divergent tunnel using an endoscopic technique.[93, 124] Anteriorly placed endoscopic screws can often be left in place because the new femoral tunnel can be drilled behind the old one (Fig. 19).[93] Removing these screws may weaken the bone and can result in a large bony defect on the femur. Endoscopically placed screws that are prominent, however, may require removal because they may impinge on the new ligament. If removal of an endoscopically

Figure 19. *A*, Failed patellar tendon ACL reconstruction, secondary to anterior placement of the femoral tunnel (error in surgical technique). Hardware removal is unnecessary because there is sufficient room to drill behind the original femoral interference screw. *B*, Arthroscopic view (*left* knee). The probe is located just anterior to the over-the-top position. There is sufficient room to drill a tunnel behind the original endoscopically inserted femoral interference screw.

placed screw is necessary, it is important to determine from the original operative note through which portal the screw was inserted to ensure parallel placement of the guidewire and screwdriver at the time of removal. Failure to insert the screwdriver parallel to the axis of the screw may result in the screw being stripped.

Prosthetic Ligament Removal

Removal of a prosthetic ligament can present a major challenge. The surgeon must have a thorough understanding of the technique used to implant the various types of prosthetic ligaments. Preoperative CT or MR imaging scans are useful in evaluating tunnel enlargement and osteolysis. If there is a significant bony defect grafting with autogenous iliac crest, bone graft is required following the revision ACL reconstruction once the bone graft has incorporated.

The removal of Gore-Tex, Dacron, and carbon fiber synthetic ligaments presents a special set of problems.[49] These grafts have been associated with significant inflammatory reactions secondary to the creation of synthetic fiber particles. These particles have been shown to stimulate an inflammatory response that can destroy bone and cartilage. An attempt should be made to remove the ligament en bloc without the use of drills or shaver blades, which may create tiny particles that can lead to further synovitis and osteolysis. Bone gouges or trephines can be used to loosen the ligament attachments from the walls of the bone tunnels on both the tibial and femoral side. The extra-articular portion of the ligament is then grasped and removed by wrapping the end of the ligament around a Kocher clamp in a circular fashion like a sardine can opener (Fig. 20).

Ligament augmentation devices, which should be fixed to bone at one end only if properly used, can also be a challenge to remove. Review of the operative note should indicate which end of the device is fixed to bone. The end of the ligament augmentation device that is fixed to bone should be freed from the walls of the bone tunnel with a bone gouge or trephine. The device can then be removed by wrapping the fixed end around a Kocher clamp in the manner of a sardine can opener (Fig. 21).

Revision Notchplasty

Revision notchplasty is necessary in almost all revision ACL reconstructions because some element of notch regrowth occurs after most ACL reconstructions. Revision notchplasty is required to visualize the previous femoral tunnel and the "over-the-top" position, and to prevent impingement of the new graft against the roof of the notch and the medial wall of the lateral femoral condyle. Four-stranded hamstring tendon grafts and quadriceps tendon grafts have a significantly larger cross-sectional area than the normal ACL, and a larger notch is required to accommodate these grafts.[23, 54, 148] Careful review of the flexion weight bearing or tunnel-view radiographs gives information on the notch architecture and helps determine the amount of bone to be removed.

Excessive bone removal should be avoided because it can lead to compromise of the patellofemoral and tibiofemoral articular surfaces.[83] Excessive bone removal from the medial wall of the lateral femoral condyle can also result in the femoral tunnel being lateralized, thus changing the axis of the new ligament. An impingement rod as described by Howell et al[59, 60] can be used at the time of surgery to determine if an adequate notchplasty has been performed.

Bone Tunnels

The most important and technically demanding aspect of revision ACL surgery involves the placement of new bone tunnels. Once the revision notchplasty has been performed and the over-the-top position visualized, the borders of the old tunnels can be assessed regarding the ideal anatomic placement. Anterior placement of the tibial and femoral tunnels is the most common situation confronting the surgeon.

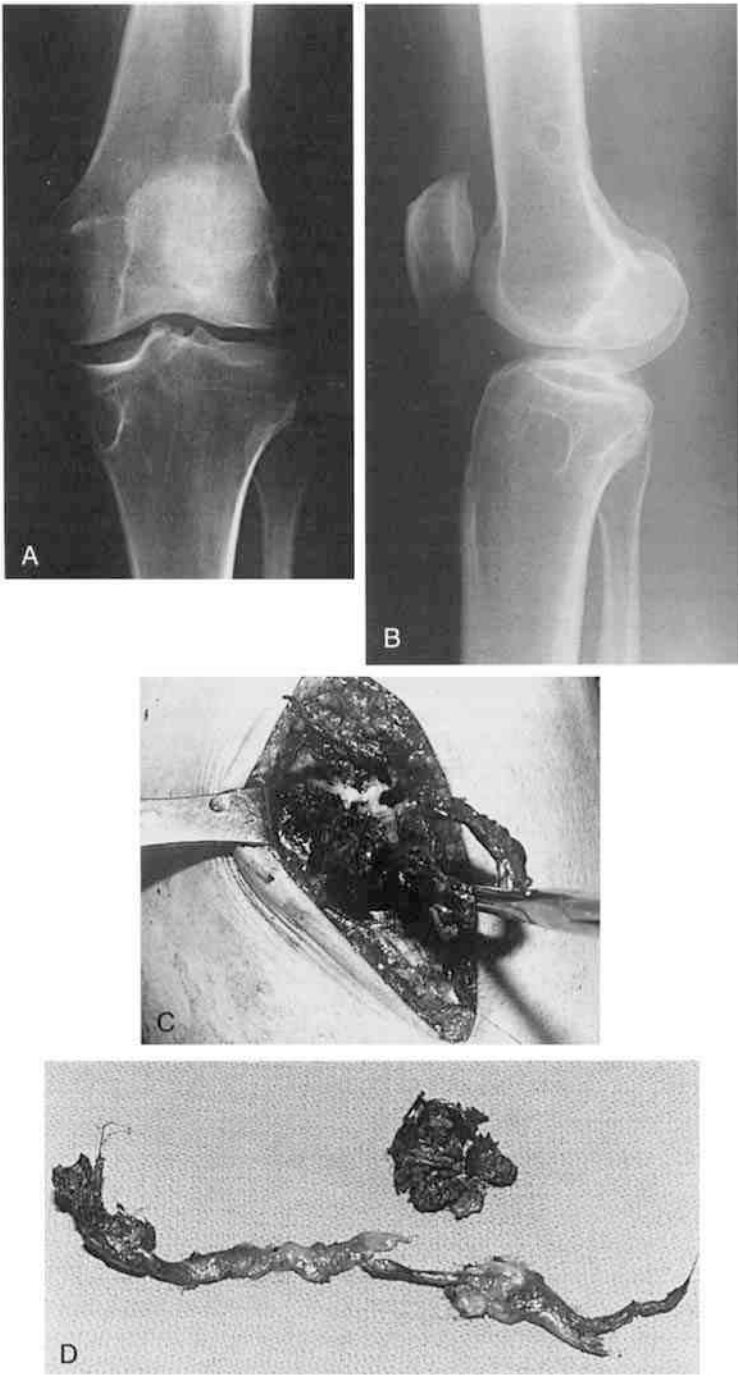


Figure 20. *See legend on opposite page*

Figure 20. *A* and *B*, Patient underwent ACL reconstruction with carbon fiber synthetic ligament 15 years ago. AP and lateral radiographs demonstrate lytic changes in the proximal tibia and distal femur. *C*, Removal of carbon fiber synthetic ligament from the tibia. *D*, Removed carbon fiber ligament. *E*, Autogenous iliac crest bone graft was required to fill the resulting bony defect. *F*, Revision with a doubled gracilis and semitendinosus graft was performed 3 months later, following consolidation of the bone graft.

If the original femoral tunnel has been positioned more than one tunnel diameter anterior to its optimal position, it is possible to drill a new tunnel in the optimal position without tunnel overlap (Fig. 22). Intraoperative radiographs or fluoroscopy can be used to confirm proper placement of the femoral guide pin prior to the drilling of the new tunnel.

If the position of the original femoral tunnel is less than one tunnel diameter anterior to its optimal position, however, then the potential for tunnel overlap exists. Because the diameter of a four-stranded hamstring tendon graft is typically in the range of 7.5 to 9 mm, it is usually possible

Figure 21. En-bloc removal of ligament augmentation device (LAD).

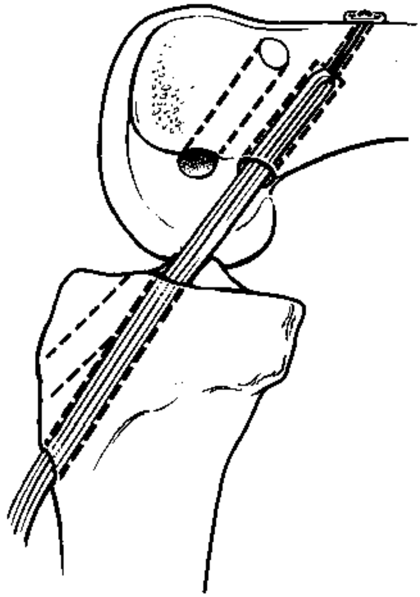


Figure 22. If the original femoral tunnel is more than 1 tunnel diameter anterior to its optimal placement, a new tunnel can be drilled at the optimal location without tunnel overlap.

to drill the revision hamstring femoral tunnel behind the original tunnel while maintaining a bony bridge between the two tunnels. This far posterior tunnel position can be achieved by using a 3.5-mm femoral offset guide to position the guide pin for the new femoral tunnel. A 4.5-mm femoral tunnel is drilled into the lateral femoral condyle using an EndoButton drill bit (Smith & Nephew Endoscopy, Andover, MA). Smooth 0.5-mm tunnel dilators (Arthrex, Naples, FL) can then be used to expand the new femoral tunnel up to the measured size of the hamstring tendon graft (Fig. 23).

Figure 23. *A*, If the original femoral tunnel is less than 1 tunnel diameter anterior to its optimal placement, the possibility of tunnel overlap exists. Because the diameter of hamstring tendon grafts is typically in the range of 7–9 mm, it is usually possible to drill the new tunnel just anterior to the posterior cortex, and then use tunnel dilators to expand the tunnel up to the measured size of the hamstring tendon graft. EndoButton (Smith & Nephew, Endoscopy, Andover, MA) femoral fixation gives one the option of "blowing out" the posterior cortex to avoid tunnel overlap. *B*, Correctly placed femoral tunnel. Original patellar tendon reconstruction failed because of a traumatic reinjury. *C*, New femoral tunnel was drilled behind the original tunnel using a 4.5-mm EndoButton drill bit (Smith & Nephew Endoscopy, Andover, MA) and progressively dilated with 0.5-mm smooth dilators (Arthrex, Naples, FL) up to the measured size of the graft. *D*, Note the thin bony bridge between the old and new divergent femoral tunnel. *E*, New tunnel has been placed into virgin cancellous bone. *F*, Final arthroscopic appearance of four stranded hamstring tendon ACL graft. Original femoral tunnel was bone grafted with bone harvested using a coring reamer during drilling of the tibial tunnel.

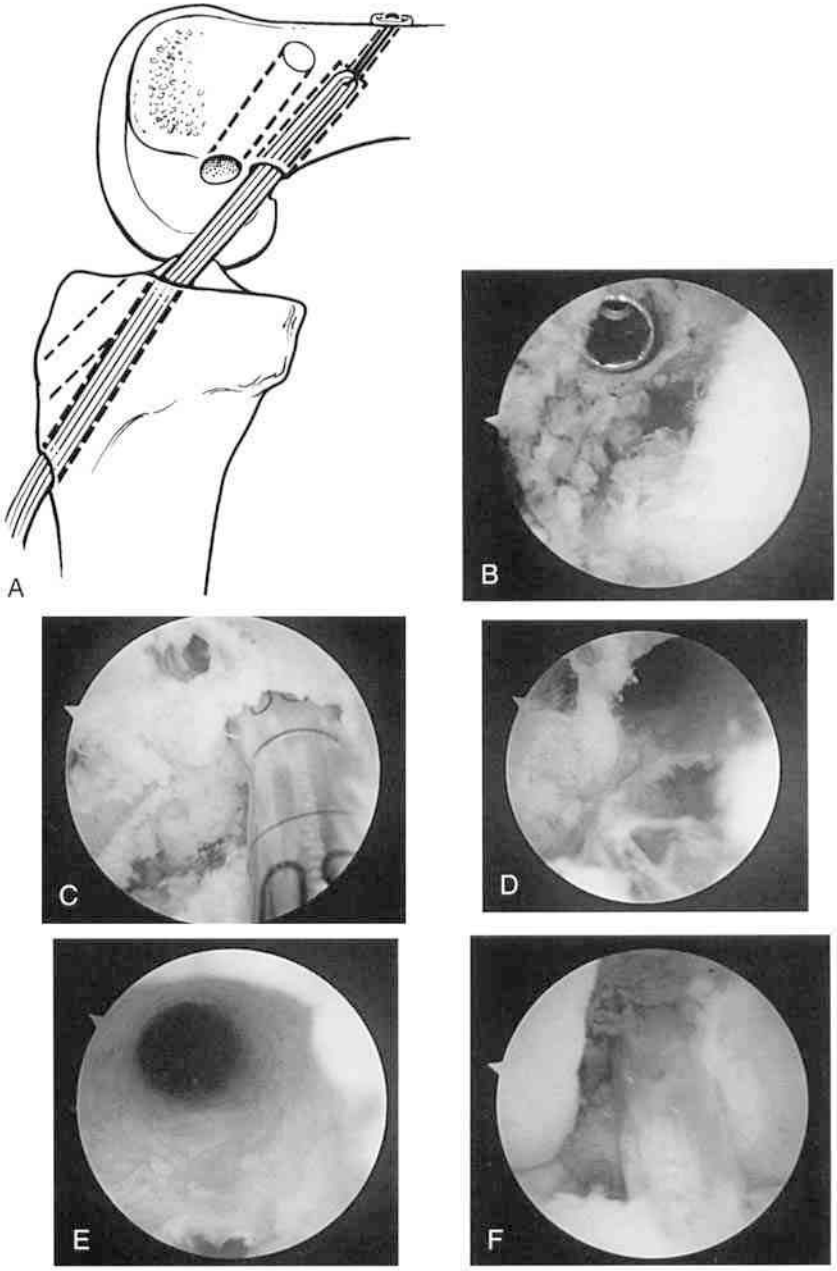


Figure 23. *See legend on opposite page*

Similarly, if the original ACL graft was positioned too posteriorly and a posterior cortical wall blowout occurred at the time of the original reconstruction, the guidewire for the revision hamstring tendon graft bone tunnel is positioned at the optimal site for the bone tunnel and a EndoButton used to fix the femoral end of the graft (Fig. 24). If the original femoral tunnel is in optimal position, the intra-articular position of the original tunnel can be maintained, and a divergent new tunnel drilled using a different surgical technique (*see* Fig. 14*A*).

Three options exist for dealing with enlarged femoral bone tunnels. The first option is to avoid drilling a femoral bone tunnel by placing the hamstring tendon graft in the over-the-top position (Fig. 25). The study of Karlson et al[74] demonstrated no significant difference in outcome for hamstring tendon grafts placed through a femoral drill hole versus grafts positioned in the over-the-top position. The second option consist of removing the old ligament and bone grafting the resulting defect with iliac crest bone graft as the first stage, followed by revision ACL reconstruction after consolidation of the bone graft (Fig. 26). The third option is to use an allograft with large bone block to fill the enlarged bone tunnel (Fig. 27).

Similar options exist for dealing with the malpositioned tibial tunnel. If the tibial tunnel is more than one tunnel diameter too anterior to its optimal position, then a new tunnel can be drilled in the optimal position without tunnel overlap occurring (Fig. 28). The slightly anterior

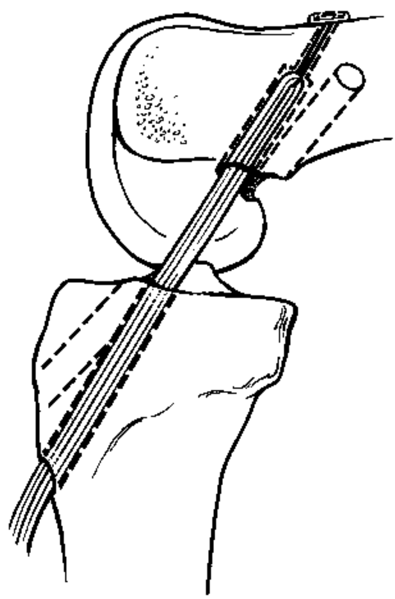


Figure 24. If the original femoral tunnel violated the posterior cortex, a new tunnel can be drilled in the optimal position and the graft fixed to the femoral cortex with an EndoButton.

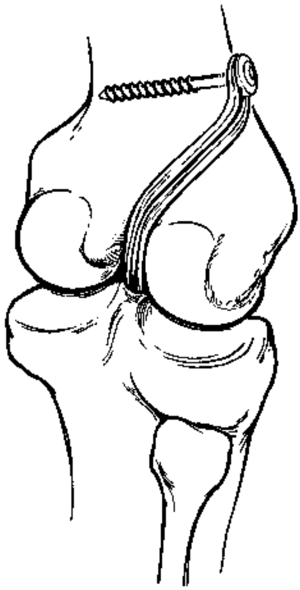


Figure 25. An enlarged femoral tunnel can be bypassed by using the over-the-top position.

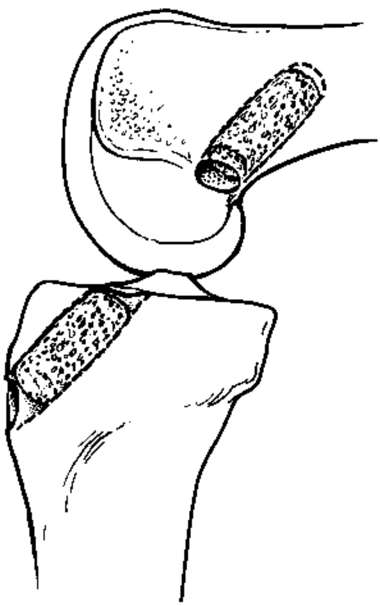


Figure 26. Enlarged bone tunnels can be grafted with iliac crest bone graft.

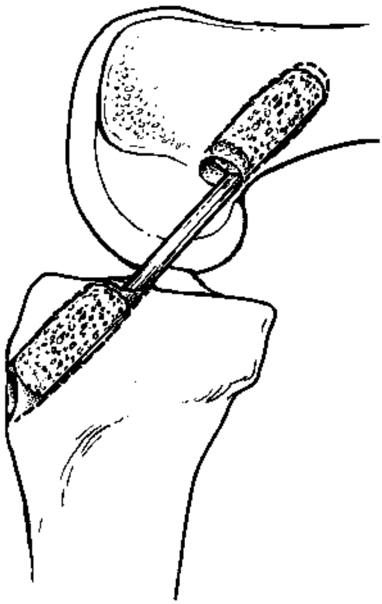


Figure 27. A patellar tendon allograft with large bone blocks can be used to fill enlarged bone tunnels.

tibial tunnel (less than one tunnel diameter anterior to its ideal position) presents a slightly more challenging problem. Options include drilling a new tunnel or expanding the existing tunnel until it is located in the optimal position. The resulting gap between the anterior wall of the old tunnel and the revision graft can be filled by using an allograft with a large bone block, or the tunnel bone grafted with autogenous iliac crest bone graft concurrently.

Another option consists of positioning the intra-articular position of the new tunnel at the optimal location and changing the external starting position, thus diverging the two tunnels. If the original tibial tunnel was optimally placed, then a divergent tibial tunnel can be drilled by changing the external starting position (Fig. 29).

A tibial tunnel placed more than one tunnel diameter posterior to its optimal position can be handled by drilling the new tunnel in optimal position (Fig. 30). A tibial tunnel placed slightly posteriorly (less than one tunnel diameter from its optimal position) can be very challenging. Because of the potential for tunnel overlap and the new graft falling posteriorly into the old tunnel, this situation is best handled by removing the old ligament and tibial fixation hardware and bone grafting the old tibial tunnel and staging the revision ligament reconstruction.

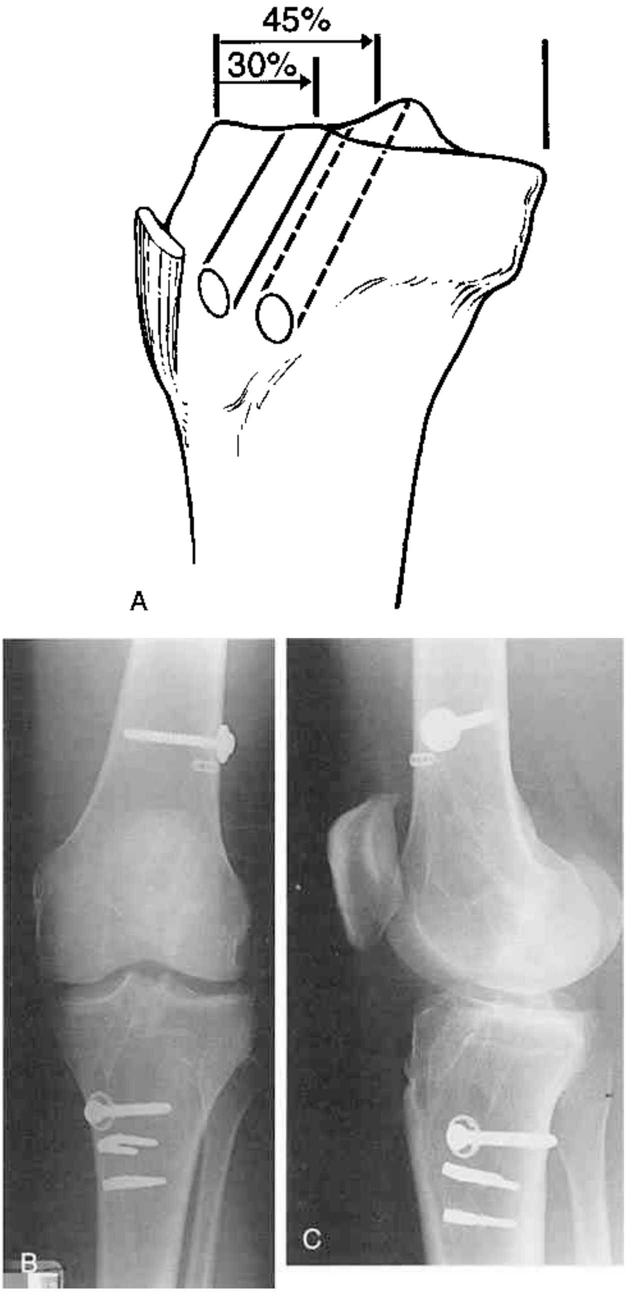


Figure 28. *A,* If the tibial tunnel is located more than 1 tunnel diameter anterior to its optimal position *(solid lines)*, a new tunnel can be drilled in the optimal tunnel without the potential for tunnel overlap *(dashed lines)*. *B* and *C,* AP and lateral radiographs at 2-year follow-up. Original tunnel was located more than 1 tunnel diameter anterior to its optimal position. New tibial tunnel was drilled in the optimal position. It was possible to leave the original femoral fixation hardware.

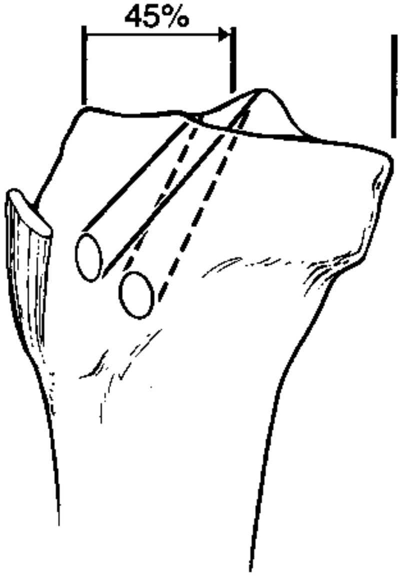


Figure 29. The optimally positioned tibial tunnel can be handled by changing the external starting position of the new tunnel in the sagittal and frontal planes.

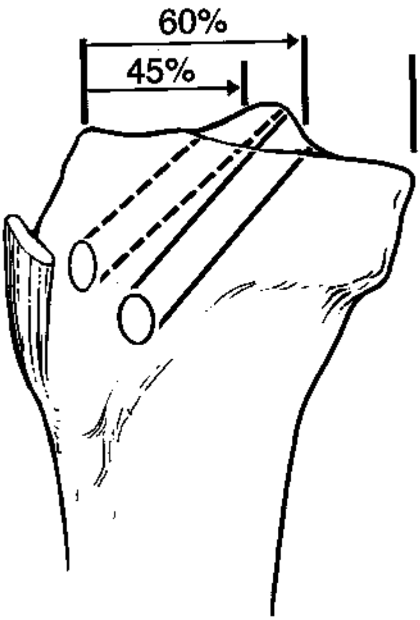


Figure 30. If the original tibial tunnel is located more than 1 tunnel diameter posterior to its optimal position, a new tunnel can be drilled in the optimal position without tunnel overlap.

Graft Fixation

During the early postoperative period, graft fixation is the weak link in the ACL reconstruction.[28] Because of the variations in the type of replacement graft, tunnel placement, bone quality, and surgical techniques used during revision ACL reconstruction, the surgeon must be knowledgeable and proficient with all methods of ACL graft fixation.

Interference screw fixation has been demonstrated to be the strongest and stiffest fixation technique for bone-tendon-bone grafts.[80, 149] Interference screw fixation strength is dependent on the local bone quality, however, and in revision ACL surgery bone stock and bone quality may be compromised by the existing or the new tunnels making interference screw fixation inadequate.

Under these circumstances, alternative fixation methods, such as tying the bone block sutures around a screw and post, should be considered. If the posterior femoral cortex was violated during the primary reconstruction, use of an endoscopically inserted interference screw is also not possible. In this situation, the surgeon has the option of using a two-incision approach and fixing the femoral bone block with an outside-in interference screw or tying the bone block sutures around a screw and post. Alternatively, the EndoButton (Smith & Nephew Endoscopy, Mansfield, MA) can be used in this situation because this implant relies on the integrity of the lateral femoral cortex and does not depend on fixation within the bone tunnel.

Although direct fixation of hamstring tendon grafts with metal or bioabsorbable interference screws has become an increasingly popular method of graft fixation, and may provide adequate fixation strength when used in primary ACL reconstructions, potential tunnel overlap and poor bone quality may make this an unreliable method of fixation for revision ACL surgery.[129, 145]

Brown et al[24] have demonstrated that hamstring tendons fixed on tibia with spiked ligament washers and on the femur with the Bone Mulch Screw (Arthrotek, Ontario, CA). EndoButton (Smith & Newphew Endoscopy, Mansfield, MA) using EndoButton Tape or three number 5 sutures, and the TransFix (Arthrex, Naples, FL) implants have adequate fixation strength even in osteopenic bone. Recent biomechanical testing at the Orthopaedic Biomechanics Laboratory (CHB) using human cadaveric knees (< 50 years) has demonstrated that the EndoButton with continuous polyester loop is the strongest femoral fixation device currently available (mean failure load $= 1430 \pm 124$, stiffness $= 180 \pm 33$). Tibial fixation options include tandem barbed staples, ligament washers, sutures tied around a post, and the WasherLoc (Arthrotek, Warsaw, IN).

Addressing Associated Ligamentous Laxity

Failure to address the secondary restraints may result in abnormal loads being applied to the revised ACL reconstruction and eventual failure of the reconstruction. The secondary restraints to anterior translation on the medial side of the knee that may need to be addressed at the time of the revision procedure include the superficial MCL, the POL, and the medial meniscus.[114]

Chronic laxity of the MCL can be addressed by recession of the femoral attachment site or advancement of the tibial insertion of the lax ligament.[114] In cases where the existing ligamentous tissue is inadequate an autogenous semitendinosus graft, Achilles tendon, or patellar tendon allograft may be used to reconstruct the MCL. The POL can be tightened by advancing the femoral side of the ligament as described by Hughston and Eilers,[63] and Paulos et al.[114] If this tissue is inadequate, then the POL can be reconstructed with a strip of semimembranous as described by Muller.[94]

At the present time, although the indications and role of meniscal transplantation are controversial, medial meniscal transplantation may have a place in revision ACL surgery because it may allow restoration of the important brake stop mechanism in a knee that has previously undergone total or subtotal medial meniscectomy.[94, 107, 114]

Failure to address posterolateral instability may result in continued complaints of the "knee giving-way backward" in the face of an otherwise successful ACL reconstruction.[97, 102] The lateral and posterolateral structures that must be addressed are the lateral collateral ligament (LCL), the popliteal tendon, and the popliteofibular ligament.[46, 50, 102, 153, 154] In chronic cases where a definitive LCL is present and the popliteal attachments to the fibula and tibia are intact, a proximal advancement of these structures as described by Noyes and Barber-Westin[102] can be used to tighten these structures at the time of revision ACL surgery.

In cases where the LCL is thin or deficient, we prefer to reconstruct the LCL with one half of the biceps femoris tendon and suture the remaining LCL tissue to the autogenous graft.[102, 153] The biceps tendon is tubularized with a baseball stitch using a no. 5 nonabsorbable suture and passed through a bone tunnel positioned at the lateral femoral epicondyle and drilled to the medial side of the distal femur (Fig. 31). To avoid drilling the tunnel for the LCL biceps tendon graft across the femoral end of the ACL graft, the tunnel for the LCL reconstruction should be drilled prior to passing the ACL graft. A fully fluted reamer or smooth tunnel dilator is placed into the ACL femoral tunnel while the bone tunnel for the biceps tendon graft is drilled, thereby avoiding drilling through the ACL graft. The biceps tendon is passed into the bone tunnel and the no. 5 sutures tied to a button on the medial side of the femur. If the biceps tendon has previously been injured or is felt to be inadequate, the LCL can be reconstructed with doubled autogenous semitendinosus or gracilis tendons as described by Aglietti and Buzzi (Fig. 32).[1]

We prefer to address chronic laxity of the popliteal tendon complex with an doubled autogenous semitendinosus graft. The graft is fixed on the medial side of the distal femur with an EndoButton and on the tibia and fibula head with sutures tied over buttons (Fig. 33). In cases where autogenous grafts are not available, then the LCL and popliteal complex

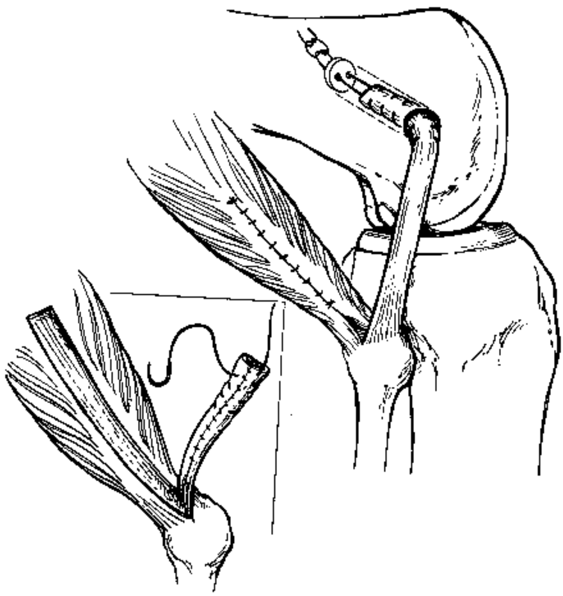


Figure 31. The lateral collateral ligament can be reconstructed using a biceps tendon graft.

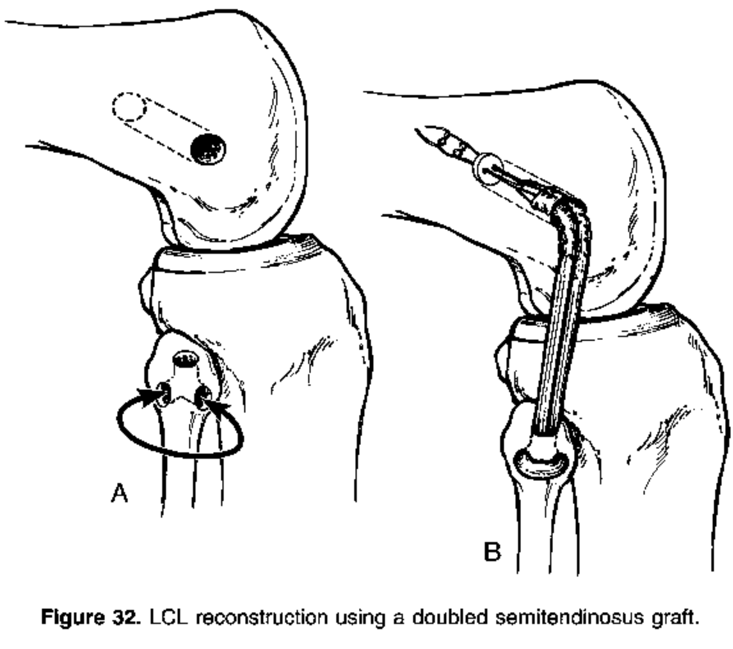


Figure 32. LCL reconstruction using a doubled semitendinosus graft.

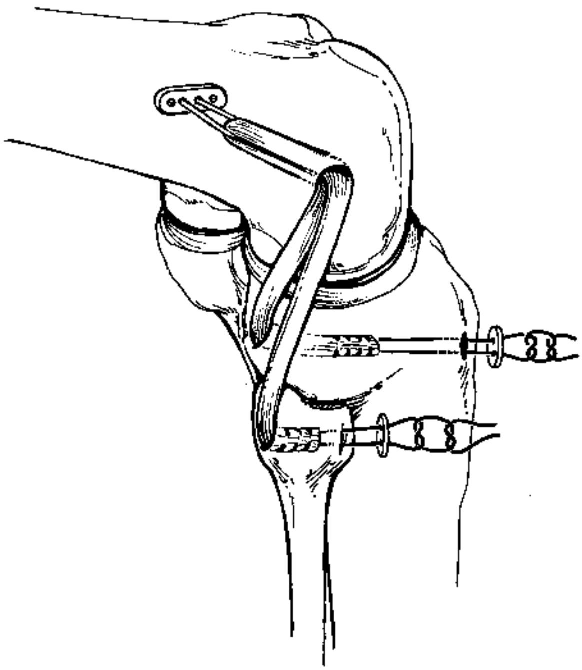


Figure 33. Reconstruction of the popliteus tendon and popliteofibular ligament, using a semitendinosus graft and EndoButton fixation.

is reconstructed with an Achilles tendon or patellar tendon allografts as described by Veltri and Warren[153, 154] and Noyes and Barber-Westin.[102]

Rehabilitation Following Revision ACL Surgery

The rehabilitation program after revision ACL surgery is influenced by both surgical and patient variables. Patient variables include the presence of generalized ligamentous laxity, bone quality, the preoperative laxity of the knee, patient size, limb alignment, and patient motivation and compliance. Surgical variables include the type of ACL replacement graft used, the type of graft fixation, graft placement, and the need for concomitant extra-articular surgery. Because of the many patient and surgical variables, a "cookbook" type of rehabilitation program should not be used; rather, a customized protocol taking these variables into account should be developed.

In general, revision ACL surgery should be considered salvage surgery, and a less aggressive rehabilitation program used in most cases. Weaker initial graft fixation, laxity of secondary restraints, the potential need to address associated ligamentous injuries, and the presence of more significant articular cartilage changes make the use of an accelerated rehabilitation program inappropriate in most revision cases.

The major changes in the rehabilitation program following revision ACL surgery consist of a slower progression of weight bearing and functional exercises.

Full passive extension to 0 degrees (avoidance of hyperextension); active-assisted exercises using the opposite leg, heel drags, wall slides, quadriceps isometrics, straight leg raises (quad lag less than 10 degrees), ankle pumps, and patellar mobilization are all allowed immediately after surgery.

In most cases full range of motion should be re-established by 6 to 8 weeks postoperatively. A straight leg orthosis is used until the patient demonstrates good muscular control of the leg. Assuming that there has been no associated ligamentous surgery performed, the weight-bearing status of the patient is increased a maximum of 25% body weight per week.

Patients are weaned off crutches no earlier than the end of week 4, and then only if they demonstrate good neuromuscular control of the leg and a normal or near normal gait pattern. The stationary bicycle is begun between weeks 4 to 6; however, weight-bearing closed-chain exercises, such as minisquats, lateral step-ups, toe raises, and the stair-climber, are delayed until the beginning of the sixth postoperative week. Jogging and running are delayed until 16 to 20 weeks after surgery. Turning, twisting, and pivoting drills are started at 24 weeks postoperatively. In general, most patients are advised against returning to twisting, pivoting sports before 9 months.

SUMMARY

An increasing number of revision ACL reconstructions are being performed each year. Revision ACL surgery is challenging and cannot be approached in the same manner as primary ACL surgery. Successful revision ACL surgery requires a detailed history, a comprehensive physical examination, appropriate radiologic studies, and careful preoperative planning. The results of revision ACL surgery do not equal the results of primary ACL surgery, and this should be explained to the patient prior to surgery.

In order to avoid repeating errors that led to failure of the primary reconstruction, the etiology of the primary failure must be clearly understood before proceeding with the revision procedure. Although graft failure is the most common reason for failure of the original reconstruction and revision surgery, other non–graft-related problems, such as loss of motion, extensor mechanism dysfunction, and degenerative arthritis, can also result in an unsatisfactory outcome and residual complaints.

Errors in surgical technique, specifically nonanatomic graft placement and failure to address associated ligamentous injuries at the time of the original procedure, are responsible for graft failures in most reported series. Preoperative planning must address the issues of graft selection, skin incisions, hardware removal, tunnel placement, graft fixation, and associated ligamentous injuries. Loss of motion and in some cases enlarged bone tunnels may require a staged approach. Because of the weaker initial graft fixation, laxity of secondary restraints, the potential need to address associated ligamentous injuries, and the presence of more significant articular cartilage changes, an accelerated rehabilitation program is inappropriate in most revision cases.

Successful revision ACL surgery requires a motivated and compliant patient, a well thought out plan, and an experienced surgeon who is knowledgeable and proficient with a variety of different surgical techniques, graft sources, and graft fixation techniques.

Case Study

Category of failure: Graft failure

Origin of failure: Error in surgical technique (anterior placement of the tibial tunnel)

Original ACL reconstruction was performed with an autogenous patellar tendon graft utilizing a rear-entry technique. Postoperative radiographs (Fig. 34) shows what appears to be satisfactory tunnel placement. The patient resumed full athletic activities and was symptomfree for 2 years at which time they sustained a giving-way episode playing soccer. Examination at that time revealed a +2 Lachman test, +1 anterior drawer test, and a +3 pivot-shift test.

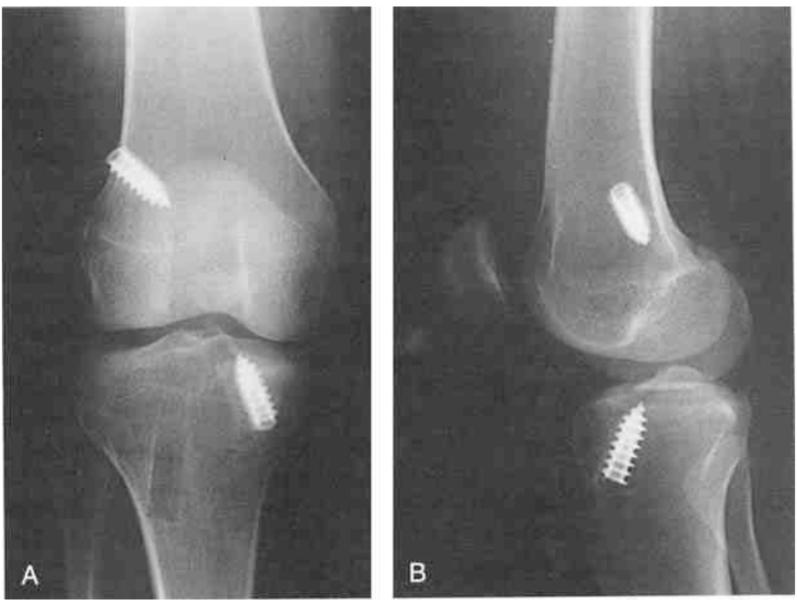
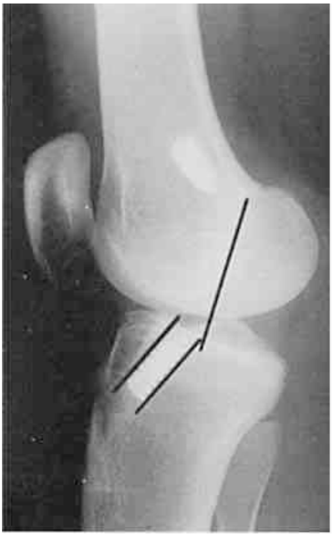


Figure 34. *A* and *B*, Postoperative radiographs taken after the initial ACL reconstruction.



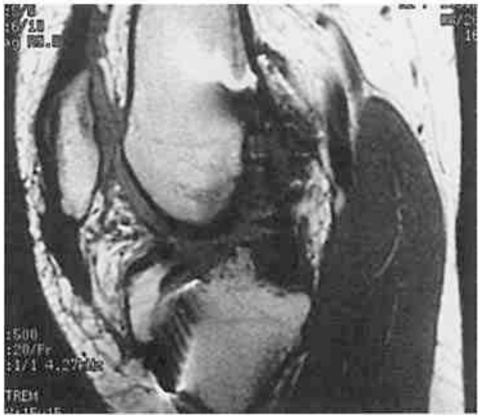


Figure 35. Lateral radiograph in maximum hyperextension demonstrating that the entire tibial tunnel lies anterior to the Blumensaat's line (severe roof impingement).

Figure 36. Postoperative MR imaging 2 years after the onset of "giving-way" symptoms. The MR image demonstrates severe graft impingement by the roof of the intercondylar notch.

A lateral radiograph with the knee in maximum hyperextension (Fig. 35) demonstrated a vertically oriented Blumensaat's line and recurvatum. The tibial tunnel was noted to be positioned anterior to Blumensaat's line. An MR imaging scan (Fig. 36) demonstrated impingement of the ACL graft by the roof of the intercondylar notch. Revision ACL reconstruction was performed using a doubled gracilis and semitendinosus graft. The distal portion of the original patellar tendon harvest incision was extended distally to harvest the hamstring tendons and remove the tibial fixation hardware (Fig. 37). An 8-mm diameter graft was obtained (Fig. 38).

At surgery the notch was noted to have regrown and the ACL graft had been guillotined by the roof of the notch with only a remnant remaining (Fig. 39). The guide pin for the new tibial tunnel was positioned posterior and parallel to the roof of the intercondylar notch with the knee in maximum extension. Correct guide pin placement was verified by an intraoperative radiograph (Fig. 40). Arthroscopic appearance of the revision hamstring tendon graft (Fig. 41). An MRI scan (Fig. 42) and arthroscopic second look at 2 years (Fig. 43) demonstrated a healthy appearing ACL graft.

At the 2 year follow-up, the patient had returned to their preinjury activity level, had a full range of motion, and no swelling. Ligamentous examination revealed a negative Lachman test, negative pivot shift test, and a side-to-side manual maximum difference of 3 mm. Radiographs (Fig. 44) taken at the 2 year follow-up demonstrated that the new tibial tunnel was positioned parallel and posterior to the roof of the intercondylar notch with the knee in maximum hyperextension (impingement-free tibial tunnel).

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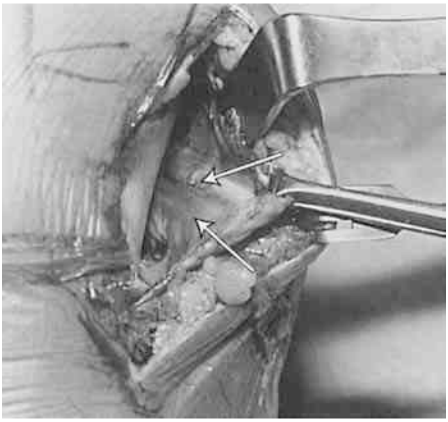

Figure 37. Exposure of the gracilis (upper tendon) and the semitendinosus (lower tendon) *(arrows)*.

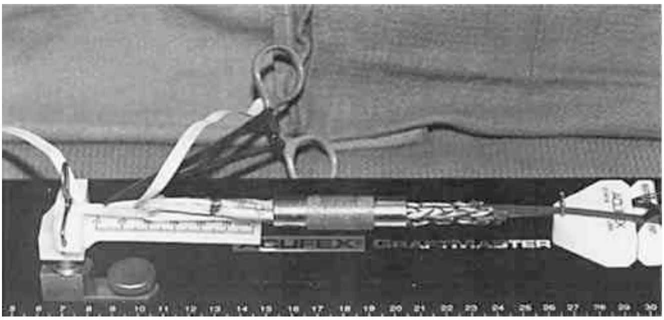

Figure 38. Pretensioning of the doubled gracilis and semitendinosus graft. The graft measured 8-mm in diameter.

Figure 39. Arthroscopic appearance of the notch at the time of revision ACL surgery. Note the notch regrowth and destruction of the ACL graft.

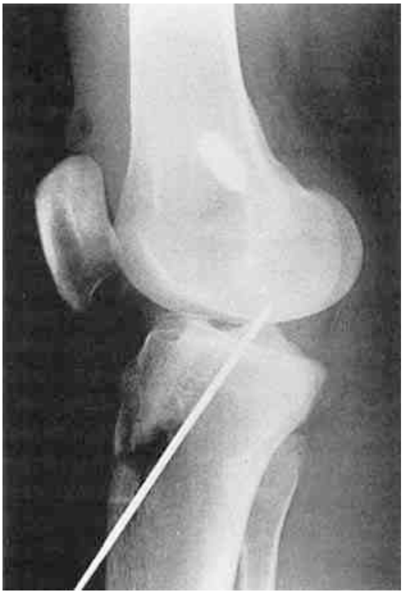

Figure 40. Intraoperative lateral radiograph checking tibial guide pin placement. The guide pin lies posterior and parallel to the roof of the intercondylar notch.

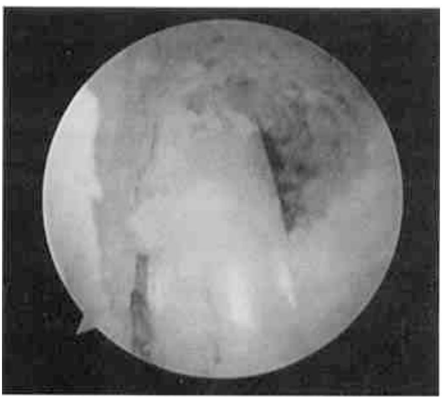

Figure 41. Arthroscopic appearance of the revision hamstring tendon graft.

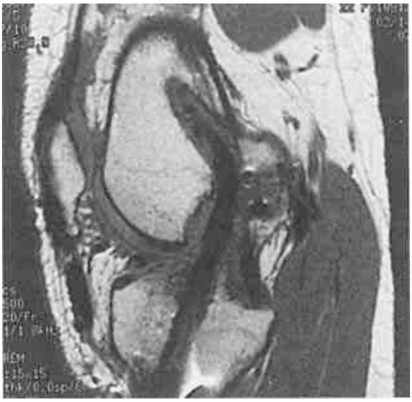

Figure 42. MR imaging of the revision ACL graft, demonstrating a healthy appearance at 2 years.

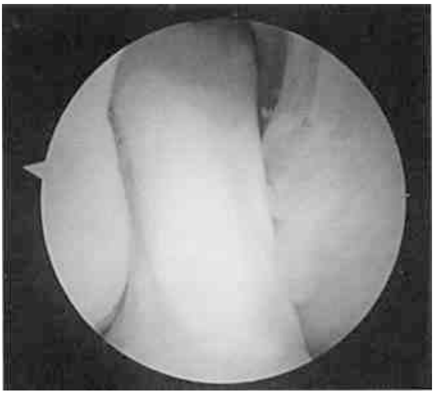

Figure 43. Arthroscopic second look of the revision ACL graft at 2 years.

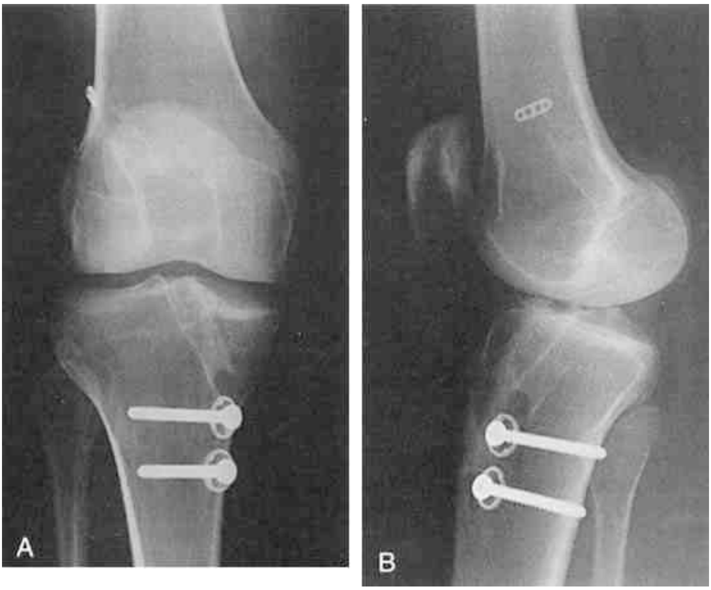

Figure 44. *A* and *B*, Two-year postoperative radiographs. The new tibial tunnel can be seen to lie posterior to the original tunnel and is parallel and posterior to the roof of the intercondylar notch.

References

1. Aglietti P, Buzzi R: Chronic anterior cruciate ligament injuries. *In* Insall JN, Windsor RE, Scott NW, et al (eds): Surgery of the Knee, vol 1, ed 2. New York, Churchill Livingstone, 1993, pp 425–504
2. Aglietti P, Buzzi R, D'Andria S, et al: Arthroscopic anterior cruciate ligament reconstruction with patellar tendon. Arthroscopy 8:510–516, 1992
3. Aglietti P, Buzzi R, D'Andria S, et al: Patellofemoral problems after intraarticular anterior cruciate ligament reconstruction. Clin Orthop 288:195–204, 1993
4. Aglietti P, Buzzi R, Giron F, et al: Arthroscopic-assisted anterior cruciate ligament reconstruction with the central third patellar tendon. Knee Surg Sports Traumatol Arthrosc 5:138–144, 1997
5. Aglietti P, Buzzi R, Zaccherotti G, et al: Patellar tendon versus doubled semitendinosus and gracilis tendons for anterior cruciate ligament reconstruction. Am J Sports Med 22:211–217, 1994
6. Amiel D, Kleiner JB, Akeson WH: The natural history of the anterior cruciate ligament autograft of patellar tendon origin. Am J Sports Med 14:449–462, 1986
7. Amiel D, Kleiner JB, Roux RD, et al: The phenomenon of ligamentization anterior cruciate ligament reconstruction with autogenous patellar tendon. J Orthop Res 4:162–172, 1986
8. Andersen HN, Amis AA: Review on tension in the natural and reconstructed anterior cruciate ligament. Knee Surg Sports Traumatol Arthrosc 2:192–202, 1994
9. Anderson C, Odensten M, Good L, et al: Surgical or nonsurgical treatment of acute rupture of the anterior cruciate ligament: A randomized study with long-term follow-up. J Bone Joint Surg Am 71:965–974, 1989
10. Arnoczky SP, Warren RF, Ashlock MA: Replacement of the anterior cruciate ligament using a patellar tendon allograft: An experimental study. J Bone Joint Surg Am 68:376–385, 1986
11. Bach BR: Potential pitfalls of Kurosaka screw interference fixation for ACL surgery. Am J Knee Surg 2:76–82, 1989
12. Bach BR, Jones GT, Sweet FA, et al: Arthroscopy-assisted anterior cruciate ligament reconstruction using patellar tendon substitution: Two- to four-year follow-up results. Am J Sports Med 22:758–767, 1994
13. Bach BR, Levy ME, Bojchuk J, et al: Single-incision endoscopic anterior cruciate ligament reconstruction using patellar tendon autograft: Minimum two-year follow-up evaluation. Am J Sports Med 26:30–39, 1998
14. Bach BR, Tradonsky S, Bojchuk J, et al: Arthroscopically assisted anterior cruciate ligament reconstruction using patellar tendon autograft: Five- to nine-year follow-up evaluation. Am J Sports Med 26:20–29, 1998
15. Bach BR, Wojtys EM, Lindenfeld TN: Reflex sympathetic dystrophy, patellar infera contracture syndrome, and loss of motion following anterior cruciate ligament surgery. Instr Course Lect 46:241–260, 1996
16. Ballmer PM, Ballmer FT, Jakob RP: Reconstruction of the anterior cruciate ligament alone in the treatment of a combined instability with complete rupture of the MCL:Medial collateral ligament: A prospective study. Arch Orthop Trauma Surg 110:139–141, 1991
17. Bear BJ, Cohen S, Bowen M, et al: Patellar fracture after anterior cruciate ligament reconstruction using bone patellar bone autogenous grafts. Orthop Tarns 20:9, 1996
18. Berg EE: Management of patellar fractures associated with central third bone-patellar tendon-bone autograft ACL reconstructions: Technical note. Arthroscopy 12:756–759, 1996
19. Bern GS, Howell SM: Roofplasty requirements in vitro for different tibial hole placements in anterior cruciate ligament reconstructions. Am J Sports Med 21:292–298, 1993
20. Bonamo J, Krinick RM, Sporn AA: Rupture of the patellar ligament after use of its central-third for anterior cruciate reconstruction. J Bone Joint Surg Am 66:1294–1297, 1984
21. Bonatus TJ, Alexander AH: Patellar fracture and avulsion of the patellar ligament

complicating arthroscopic anterior cruciate ligament reconstruction. Orthop Rev 20:770–774, 1991
22. Breitfuss H, Fröhlich R, Povacz P, et al: The tendon defect after anterior cruciate ligament reconstruction using the mid third patellar tendon—a problem for the patellofemoral joint? Knee Surg Sports Traumatol Arthrosc 3:194–198, 1996
23. Brown CH, Hamner D, Hecker AT, et al: Biomechanics of semitendinosus and gracilis tendon grafts. *In* Book of Abstracts Sports Medicine 2000, Stockholm, 1995, pp 39–41
24. Brown CH, Sklar JH, Hecker AT, et al: Endoscopic anterior cruciate ligament graft fixation. *In* Book of Abstracts 10th Combined Orthopaedic Association Meeting, New Zealand, 1998
25. Brownstein B, Bronner S: Patella fractures associated with accelerated ACL rehabilitation in patients with autogenous patellar tendon reconstructions. J Orthop Sports Phys Ther 26:168–172, 1997
26. Burks RT, Leland R: Determination of graft tension before fixation in anterior cruciate ligament reconstruction. Arthroscopy 4:260–266, 1988
27. Buss DD, Warren RF, Wickiewicz TJ, et al: Arthroscopically assisted reconstruction of the anterior cruciate ligament with use of autogenous patellar-ligament grafts: Results after twenty-four to forty-two months. J Bone Joint Surg Am 75:1346–1355, 1993
28. Butler DL: Evaluation of fixation methods in cruciate ligament replacement. Instr Course Lect 23:173–183, 1987
29. Bylski-Austrow DI, Grood ES, Hefzy MS, et al: Anterior cruciate ligament replacements: A mechanical study of femoral attachment location, flexion angle at tensioning, and initial tension. J Orthop Res 8:522–531, 1990
30. Carangelo RJ, Langeland RH, Caputo AE, et al: Postoperative patellar fractures after autogenous patellar tendon harvest for anterior cruciate ligament reconstruction. *In* Final Program 62nd Annual Meeting American Academy of Orthopaedic Surgeons, Orlando, 1995, p 318
31. Christen B, Jakob RP: Fractures associated with patellar ligament grafts in cruciate ligament surgery. J Bone Joint Surg Br 74:617–619, 1992
32. Clancy WG, Nelson DA, Reider B, et al: Anterior cruciate ligament reconstruction using one-third of the patellar ligament augmented by extra-articular tendon transfers. J Bone Joint Surg Am 64:352–359, 1982
33. Cooper DE, Wilson TW: Clinical failure of tibial interference screw fixation after anterior cruciate ligament reconstruction. Am J Sports Med 24:693–697, 1996
34. Corry IS, Webb JM, Clingeleffer AJ, et al: Endoscopic reconstruction of the anterior cruciate ligament—a comparison of patellar tendon with four strand hamstring autograft. Poster 10th Combined Orthopaedic Associations Meeting, New Zealand, 1998
35. Corsetti JR, Jackson DW: Failure of anterior cruciate ligament reconstruction: The biologic basis. Clin Orthop 323:42–49, 1996
36. Crosby LA, Kamins P: Fracture of the patella during graft harvest for cruciate ligament reconstruction. Complications in Orthopaedics 6:104–107, 1991
37. Daluga D, Johnson C, Bach BR: Primary bone grafting following graft procurement for anterior cruciate ligament insufficiency. Arthroscopy 6:205–208, 1990
38. Daniel DM, Stone ML, Dobson BE, et al: Fate of the ACL-injured patient: A prospective outcome study. Am J Sports Med 22:632–644, 1994
39. DeLee JC, Craviotto DF: Rupture of the quadriceps tendon after a central third patellar tendon anterior cruciate ligament reconstruction. Am J Sports Med 19:415–416, 1991
40. Dye SF, Chew MH: Restoration of osseous homeostasis after anterior cruciate ligament reconstruction. Am J Sports Med 21:748–750, 1993
41. Feagin JA, Curl WW: Isolated tear of the anterior cruciate ligament. Am J Sports Med 4:95–100, 1976
42. Fetto JF, Marshall JL: The natural history and diagnosis of anterior cruciate ligament insufficiency. Clin Orthop 147:29–38, 1980
43. Foreman SK, Jackson DW: Cyclops lesions. *In* Jackson DW, Arnoczky SP, Frank CB, et al (eds): The Anterior Cruciate Ligament. New York, Raven Press, 1993, pp 365–372
44. Fuss FK: Optimal replacement of the cruciate ligament from the functional-anatomical point of view. Acta Anat 140:260–268, 1991

45. Gersoff WK, Clancy WG: Diagnosis of acute and chronic anterior cruciate ligament tears. Clin Sports Med 7:727–738, 1988
46. Gollehan DL, Torzilli PA, Warren RF: The role of the posterolateral and cruciate ligaments in the stability of the human knee. J Bone Joint Surg Am 69:233–242, 1987
47. Graf B, Simon T, Jackson DW: Isometric placement of substitutes for the anterior cruciate ligament. *In* Jackson DW, Drez D Jr (eds): The Anterior Cruciate Deficient Knee. St Louis, CV Mosby, 1987, pp 102–113
48. Graf B, Uhr F: Complications of intra-articular cruciate reconstruction. Clin Sports Med 7:835–848, 1988
49. Greis PE, Steadman JR: Revision of failed prosthetic anterior cruciate ligament reconstruction. Clin Orthop 323:78–90, 1996
50. Grood ES, Stowers SF, Noyes FR: Limits of movement in the human knee. Effects of sectioning the posterior cruciate ligament and posterolateral structures. J Bone Joint Surg Am 70:88–97, 1988
51. Hardin GT, Bach BR: Distal rupture of the infrapatellar tendon after use of its central third for anterior cruciate ligament reconstruction: Case report. Am J Knee Surg 5:140–143, 1992
52. Harner CD, Irrgang JJ, Paul JJ, et al: Loss of motion after anterior cruciate ligament reconstruction. Am J Sports Med 20:499–506, 1992
53. Harter RA, Osternig LR, Singer KM, et al: Long-term evaluation of knee stability and function following surgical reconstruction for anterior cruciate ligament insufficiency. Am J Sports Med 16:434–443, 1988
54. Hecker AT, Brown CH, Deffner KT, et al: Tensile properties of young multiple stranded hamstring tendon grafts. *In* Book of Abstracts and Outline Specialty Day American Orthopaedic Society for Sport Medicine, California, 1997, p 8
55. Hefzy MS, Grood ES, Noyes FR: Factors affecting the region of the most isometric femoral attachment. Part II: The anterior cruciate ligament. Am J Sports Med 17:208–216, 1989
56. Holmes PF, James SL, Larson RL, et al: Retrospective direct comparison of three intraarticular anterior cruciate ligament reconstructions. Am J Sports Med 19:596–600, 1991
57. Howe JG, Johnson RJ, Kaplan MS, et al: Anterior cruciate ligament reconstruction using quadriceps patellar tendon graft: Part I. Long-term follow-up. Am J Sports Med 19:447–457, 1991
58. Howell SM, Barad SJ: Knee extension and its relationship to the intercondylar roof. Am J Sports Med 23:288–294, 1995
59. Howell SM, Clark JA: Tibial tunnel placement in anterior cruciate ligament reconstructions and graft impingement. Clin Orthop 283:187–195, 1992
60. Howell SM, Clark JA, Farley TE: A rationale for predicting anterior cruciate graft impingement by the intercondylar roof: An MRI study. Am J Sports Med 19:276–281, 1991
61. Howell SM, Taylor MA: Brace-free rehabilitation with early return to activity for knees reconstructed with a double-loop semitendinosus and gracilis graft. J Bone Joint Surg Am 78:814–825, 1996
62. Howell SM, Taylor MA: Failure of reconstruction of the ACL due to impingement by the intercondylar roof. J Bone Joint Surg Am 75:1044–1055, 1993
63. Hughston JC, Eilers AF: The role of the posterior oblique ligament in repairs of acute medial (collateral) ligament tears of the knee. J Bone Joint Surg Am 55:923–940, 1973
64. Irrgang JJ, Harner CD: Loss of motion following knee ligament reconstruction. Sports Med 19:150–159, 1995
65. Jackson DW, Gasser SI: Tibial tunnel placement in ACL reconstruction. Arthroscopy 10:124–131, 1994
66. Jackson DW, Grood ES, Goldstein J, et al: A comparison of patellar tendon autograft and allograft used for anterior cruciate ligament reconstruction in the goat model. Am J Sports Med 21:176–185, 1993
67. Jackson DW, Schaefer RK: Cyclops syndrome: Loss of extension following intra-articular cruciate ligament reconstruction. Arthroscopy 6:171–178, 1990
68. Jaureguito JW, Paulos LE: Why grafts fail? Clin Orthop 323:25–41, 1996

69. Johnson DL, Fu FH: Anterior cruciate ligament reconstruction: Why do failures occur? Instr Course Lect 44:391–406, 1995
70. Johnson DL, Fu FH: Revision ACL surgery: Etiology, indications, techniques, and results. Am J Knee Surg 8:155–167, 1995
71. Johnson DL, Harner CD, Swenson TM, et al: Revision anterior cruciate ligament reconstruction using fresh-frozen allograft tissue. Scientific exhibit at the 61st Annual American Academy of Orthopaedic Surgeons Meeting, New Orleans, 1994
72. Johnson DL, Swenson TM, Irrgang JL, et al: Revision anterior cruciate ligament surgery: Experience from Pittsburgh. Clin Orthop 323:100–109, 1996
73. Kaplan N, Wickiewicz TL, Warren RF: Primary surgical treatment of anterior cruciate ligament ruptures: A long-term follow-up study. Am J Sports Med 18:354–358, 1990
74. Karlson JA, Steiner ME, Brown CH, et al: Anterior cruciate ligament reconstruction using gracilis and semitendinosus tendons: Comparison of through the condyle and over the top graft placements. Am J Sports Med 22:659–666, 1994
75. Kartus J, Stener S, Lindahl S, et al: Factors affecting donor-site morbidity after anterior cruciate ligament reconstruction using bone-patellar tendon-bone autografts. Knee Surg Sports Traumatol Arthrosc 5:222–228, 1997
76. Kartus J, Stener S, Lindahl S, et al: Ipsi- or contralateral patellar tendon in anterior cruciate ligament revision surgery: A comparison of two methods. Am J Sports Med 26:499–504, 1998
77. Kleipool AE, Van Loon T, Marti RK: Pain after use of the central third of the patellar tendon for cruciate ligament reconstruction: 33 patients followed 2–3 years. Acta Orthop Scand 65:62–66, 1994
78. Kohn D, Saunder-Beuermann A: Donor-site morbidity after harvest of a bone-tendon-bone patellar tendon autograft. Knee Surg Sports Traumatol Arthrosc 2:219–223, 1994
79. Kornblatt I, Warren RF, Wickiewicz TL: Long-term follow-up of anterior cruciate ligament reconstruction using the quadriceps tendon substitution for chronic anterior cruciate ligament insufficiency. Am J Sports Med 16:444–448, 1988
80. Kurosaka M, Yoshiya S, Andrish JT: A biomechanical comparison of different surgical techniques of graft fixation in anterior cruciate ligament reconstruction. Am J Sports Med 15:225–229, 1987
81. Langan P, Fontanetta AD: Rupture of the patellar tendon after use of its central third. Orthop Rev 16:317–321, 1987
82. LaPrade RF, Hamilton CD, Montgomery RD, et al: The reharvested central third of the patellar tendon: A histologic and biomechanical analysis. Am J Sports Med 25:779–785, 1997
83. LaPrade RF, Terry GC, Montgomery RD, et al: The effects of aggressive notchplasty on the normal knee in dogs. Am J Sports Med 26:193–200, 1998
84. Larson RV, Erickson D: Complications in the use of hamstring tendons for anterior cruciate ligament reconstruction. Sports Medicine and Arthroscopy Review 5:83–90, 1997
85. Maeda A, Shino K, Horibe S, et al: Anterior cruciate ligament reconstruction with multistranded autogenous semitendinosus tendon. Am J Sports Med 24:504–509, 1996
86. Martin RP, Galloway MT, Daigneault JP, et al: Patellofemoral pain following ACL reconstruction: Bone grafting the patellar defect. Ortho Trans 20:9, 1996
87. Marumoto JM, Mitsunaga MM, Richardson AB, et al: Late patellar tendon ruptures after removal of the central-third for anterior cruciate ligament reconstruction: A report of two cases. Am J Sports Med 24:698–701, 1996
88. Marzo JM, Bowen MK, Warren RF, et al: Intra-articular fibrous nodule as a cause of loss of extension following anterior cruciate ligament reconstruction. Arthroscopy 8:10–18, 1992
89. Matthews LS, Soffer SR: Pitfalls in the use of interference screws for anterior cruciate ligament reconstruction. Arthroscopy 5:225–226, 1989
90. McCarroll JR: Fracture of the patella during a golf swing following reconstruction of the anterior cruciate ligament: A case report. Am J Sports Med 11:26–27, 1983
91. McDaniel WJ, Dameron TB: Untreated ruptures of the anterior cruciate ligament: A follow-up study. J Bone Joint Surg Am 62:696–705, 1993

92. Meade TD, Dickson TB: Technical pitfalls of single incision arthroscopy-assisted ACL ligament reconstruction. Am J Arthroscopy 2:15–19, 1992
93. Miller MD: Revision cruciate ligament surgery with retention of femoral interference screws. Arthroscopy 14:111–114, 1997
94. Muller W: Kinematics. *In* The Knee: Form, Function, and Ligament Reconstruction. New York, Springer-Verlag, 1983, p 8
95. Muller W: Secondary repair and plastic reconstruction of the ligaments in old injuries. *In* The Knee: Form, Function, and Ligament Reconstruction. New York, Springer-Verlag, 1983, p 243
96. Muneta T, Yammoto H, Ishibashi T, et al: The effects of tibial tunnel placement and roofplasty on reconstructed anterior cruciate ligament knee. Arthroscopy 11:5762, 1995
97. Noyes FR: An unresolved dilemma: Treatment of injuries to the posterolateral knee structures. Am J Knee Surg 6:95, 1993
98. Noyes FR, Barber SD: The effect of a ligament-augmentation device on allograft reconstruction for chronic ruptures of the anterior cruciate ligament. J Bone Joint Surg Am 74:960–973, 1992
99. Noyes FR, Barber SD, Mangine RE: Bone-patellar ligament-bone and fascia lata allograft for reconstruction of the anterior cruciate ligament. J Bone Joint Surg Am 72:1125–1136, 1990
100. Noyes FR, Barber-Westin SD: A comparison of results in acute and chronic anterior cruciate ligament ruptures of arthroscopically assisted autogenous patellar tendon. Am J Sports Med 25:460–471, 1997
101. Noyes FR, Barber-Westin SD: Revision anterior cruciate ligament surgery: Experience from Cincinnati. Clin Orthop 323:116–129, 1996
102. Noyes FR, Barber-Westin SD: Treatment of complex injuries involving the posterior cruciate and posterolateral ligaments of the knee. Am J Sports Med 9:200–214, 1996
103. Noyes FR, Barber-Westin SD, Roberts CS: Use of allografts after failed treatment of the anterior cruciate ligament. J Bone Joint Surg Am 76:1019–1031, 1994
104. Noyes FR, Butler DL, Grood ES, et al: Biomechanical analysis of human ligament grafts used in knee ligament repairs and reconstructions. J Bone Joint Surg Am 66:344–352, 1984
105. Noyes FR, Mangine RE, Barber SD: The early treatment of motion complications after reconstruction of the anterior cruciate ligament. Clin Orthop 227:217–228, 1992
106. Noyes FR, Mooar PA, Matthews DA: The symptomatic anterior cruciate ligament knee: Part I. The long-term functional disability in athletically active individuals. J Bone Joint Surg Am 65:154–162, 1983
107. Noyes FR, Stowers SF, Grood ES, et al: Posterior subluxations of the medial and lateral tibiofemoral compartments: An in vitro ligament sectioning study in cadaveric knees. Am J Sports Med 21:407–414, 1993
108. Noyes FR, Wojtys EM, Marshall MT: The early diagnosis and treatment of developmental patella infera syndrome. Clin Orthop 265:241–252, 1991
109. O'Brien SJ, Warren RF, Wickiewicz TL, et al: Reconstruction of the chronically insufficient anterior cruciate ligament using the central-third of the patellar ligament. J Bone Joint Surg Am 73:278–286, 1991
110. Odensten M, Lysholm J, Gillquist J: Suture of fresh ruptures of the anterior cruciate ligament: A 5 year follow-up. Acta Orthop Scand 55:270–272, 1984
111. O'Neill DB: Arthroscopically assisted reconstruction of the anterior cruciate ligament: A prospective randomized analysis of three techniques. J Bone Joint Surg Am 78:803–813, 1996
112. Otto D, Pinczewski LA, Clingeleffer A, et al: Five-year results of single-incision arthroscopic anterior cruciate ligament reconstruction with patellar tendon autograft. Am J Sports Med 26:181–188, 1998
113. Paulos LE, Rosenberg TD, Drawbert J, et al: Infrapatellar contracture syndrome: An unrecognized cause of knee stiffness with patellar infera. Am J Sports Med 15:131–141, 1987
114. Paulos LE, Rosenberg TD, Parker RD: The medial knee ligaments: Pathomechanics and surgical repair with emphasis on the external-rotation pivot-shift test. Tech Orthop 2:37–46, 1987

115. Paulos LE, Wnorowski DC, Greenwald AE: Infrapatellar contracture syndrome: Diagnosis, treatment, and long-term follow-up. Am J Sports Med 22:440–449, 1994
116. Proctor CS, Jackson DW, Simon TM: Characterization of the repair tissue after removal of the central one-third of the patellar ligament. J Bone Joint Surg Am 79:997–1006, 1997
117. Purnell ML, Lamoreaux L: Autogenous quadriceps tendon ACL revision reconstruction: Long-term follow-up study. *In* Book of Abstracts Sports Medicine 2000, Stockholm, 1995, p 65
118. Rasmussen TJ, Feder SM, Butler DL, et al: The effects of 4 Mrad of gamma irradiation on the initial mechanical properties of bone-patellar tendon-bone grafts. Arthroscopy 10:188–197, 1994
119. Rosenberg TD, Franklin JL, Baldwin GN, et al: Extensor mechanism function after patellar tendon graft harvest for anterior cruciate ligament reconstruction. Am J Sports Med 20:519–526, 1992
120. Rubinstein RA, Shelbourne KD: Preventing complications and minimizing morbidity after autogenous bone-patellar tendon-bone anterior cruciate ligament reconstruction. Oper Tech Sports Med 1:72–78, 1993
121. Rubinstein RA, Shelbourne KD, VanMeter CD, et al: Isolated autogenous bone-patellar tendon-bone graft site morbidity. Am J Sports Med 22:324–327, 1994
122. Sachs RA, Daniel DM, Stone ML, et al: Patellofemoral problems after anterior cruciate ligament reconstruction. Am J Sports Med 17:760–765, 1989
123. Safran MR: Graft selection in knee surgery. Am J Knee Surg 8:168–180, 1995
124. Safran MR, Harner CD: Technical considerations of revision anterior cruciate ligament surgery. Clin Orthop 323:50–64, 1996
125. Schabus R, Fuchs M, Kwasny O: The effect of ACL graft pre-load on the static pressure distribution in the knee-joint. Orthop Trans 14:431–432, 1990
126. Schepsis A, Getelman MH, Zimmer J: Revision ACL reconstruction: Overall results and a comparison between autograft and allograft. *In* Book of Abstracts and Instructional Courses of the 14th Annual Meeting of the Arthroscopy Association of North America, San Francisco, 1995, p 49
127. Scherl J, Hershman E: Patellar fracture in anterior cruciate ligament patellar tendon autograft. Poster presented at 2nd World Congress on Sports Trauma/AOSSm 22nd Annual Meeting, Florida, 1996
128. Schrock KB, Jackson DW: Allograft reconstruction of the anterior cruciate ligament: Basic science. Oper Tech Sports Med 3:139–147, 1995
129. Scranton PE, Pinczewski L, Auld K: Outpatient endoscopic quadrupled hamstring anterior cruciate ligament reconstruction. Oper Tech Orthop 6:177–180, 1996
130. Shelbourne KD, Gray T: Anterior cruciate ligament reconstruction with autogenous patellar tendon graft followed by accelerated rehabilitation: A two- to nine-year follow-up. Am J Sports Med 25:786–795, 1997
131. Shelbourne KD, Klootwyk TE, Wilckens JH, et al: Ligament stability two to six years after anterior cruciate ligament reconstruction with autogenous patellar tendon graft and participation in accelerated rehabilitation program. Am J Sports Med 23:575–579, 1995
132. Shelbourne KD, Nitz P: Accelerated rehabilitation after anterior cruciate ligament reconstruction. Am J Sports Med 18:292–299, 1990
133. Shelbourne KD, Patel DV: Management of combined injuries of the anterior cruciate and medial collateral ligaments. Instr Course Lect 45:275–280, 1996
134. Shelbourne KD, Patel DV: Timing of surgery in anterior cruciate ligament-injured knees. Knee Surg Sports Traumatol Arthrosc 3:148–156, 1995
135. Shelbourne KD, Porter DA: Anterior cruciate ligament-medial collateral ligament injury: Nonoperative management of MCL tears with anterior cruciate ligament reconstruction. A preliminary report. Am J Sports Med 20:283–286, 1992
136. Shelbourne KD, Trumper RV: Preventing anterior knee pain after anterior cruciate ligament reconstruction. Am J Sports Med 25:41–47, 1997
137. Shelbourne KD, Wilckens JH, Mollabashy A, et al: Arthrofibrosis in acute anterior cruciate ligament reconstruction: The effect of timing of reconstruction and rehabilitation. Am J Sports Med 19:332–336, 1991

138. Siddles JA, Larson RV, Garbini JL, et al: Ligament length relationship in the moving knee. J Orthop Res 6:593–610, 1988
139. Simonian PT, Mann FA, Mandt PR: Indirect forces and patellar fracture after anterior cruciate ligament reconstruction with the patellar ligament: Case report. Am J Knee Surg 8:60–65, 1995
140. Sledge SL, Steadman JR, Silliman JF, et al: Five-year results with the Gore-Tex anterior cruciate ligament prosthesis. Am J Knee Surg 5:65–70, 1992
141. Sommerlath K, Lysholm J, Gillquist J: The long-term course after treatment of acute anterior cruciate ligament ruptures: A 9 to 16 year follow-up. Am J Sport Med 19:156–162, 1991
142. Souryal TO, Freedman TR: Intercondylar notch size and anterior cruciate ligament injuries in athletes: A prospective study. Am J Sports Med 21:535–539, 1993
143. Speer KP, Spritzer CE, Bassett FH: Osseous injury associated with acute tears of the anterior cruciate ligament. Am J Sports Med 20:382–389, 1992
144. Spindler KP, Schils JP, Bergfeld JA, et al: Prospective study of osseous, articular, and meniscal injuries in recent anterior cruciate ligament tears by magnetic resonance imaging and arthroscopy. Am J Sports Med 21:551–557, 1993
145. Stähelin AC, Weiler A: All-inside anterior cruciate ligament reconstruction using semitendinosus tendon and soft threaded biodegradable interference screw fixation. Arthroscopy 13:773–779, 1997
146. Stäubli H-U: Arthroscopically assisted ACL reconstruction using autologous quadriceps tendon. *In* Jakob RP, Stäubli H-U (eds): The Knee and the Cruciate Ligaments. Berlin, Springer-Verlag, 1992, pp 443–451
147. Stäubli H-U: The quadriceps tendon-patellar bone construct for ACL reconstruction. Sports Medicine Arthroscopy Review 5:59–67, 1997
148. Stäubli H-U, Schatzmann L, Brunner L: Quadriceps tendon and patellar ligament: Cryosectional anatomy and structural properties in young adults. Knee Surg Sports Traumatol Arthrosc 4:100–110, 1996
149. Steiner ME, Hecker AT, Brown CH, et lal: Anterior cruciate ligament graft fixation: Comparison of hamstring and patellar tendon grafts. Am J Sports Med 22:240–246, 1994
150. Tanzer M, Lenczner E: The relationship of intercondylar notch size and content in notchplasty requirement in anterior cruciate ligament surgery. Arthroscopy 6:89–93, 1990
151. Uribe JW, Hechtman KS, Zvijac JE, et al: Revision anterior cruciate ligament surgery: Experience from Miami. Clin Orthop 323:91–99, 1996
152. Vail TP, Malone TR, Bassett FH III: Long-term functional results in patients with anterolateral rotatory instability treated by iliotibial band transfer. Am J Sports Med 20:274–282, 1992
153. Veltri DM, Warren RF: Operative treatment of posterolateral instability of the knee. Clin Sports Med 13:615–627, 1994
154. Veltri DM, Warren RF: Posterolateral instability of the knee. J Bone Joint Surg Am 76:460–472, 1994
155. Vergis A, Gillquist J: Current concepts: Graft failure in intra-articular anterior cruciate ligament reconstructions: A review of the literature. Arthroscopy 11:312–321, 1995
156. Weiss RA, Re LP, Rintz KG, et al: Incidence of anterior knee pain after treatment for anterior cruciate ligament rupture. Arthroscopy 9:366, 1993
157. Wetzler MJ, Bartolozzi AR, Gillespie MJ, et al: Revision anterior cruciate ligament reconstruction. Oper Tech Orthop 6:181–189, 1996
158. Wirth CJ, Kohn D: Revision anterior cruciate ligament surgery: Experience from Germany. Clin Orthop 323:110–115, 1996
159. Woo SL-Y, Hollis JM, Adams DJ, et al: Tensile Properties of the human femur-anterior cruciate ligament-tibia complex: The effect of specimen age and orientation. Am J Sports Med 19:217–225, 1991
160. Yaru NC, Daniel DM, Penner D: The effect of tibial attachment site on graft impingement in an anterior cruciate ligament reconstruction. Am J Sports Med 20:217–220, 1992

161. Yasuda K, Ohkoshi Y, Tanabe Y, et al: Graft site morbidity with autogenous semitendinosus and gracilis tendons. Am J Sports Med 23:706–716, 1994
162. Yasuda K, Tsujino J, Tanabe Y, et al: Effects of initial graft tension on clinical outcome after anterior cruciate ligament reconstruction: Autogenous doubled hamstring tendons connected in series with polyester tapes. Am J Sports Med 25:99–106, 1997
163. Yoshiya S, Andrish JT, Manley MT, et al: Graft tension in anterior cruciate ligament reconstruction: An in vivo study in dogs. Am J Sports Med 15:464–470, 1987

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