

Loss of Extension After Reconstruction of the Anterior Cruciate Ligament

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Abstract

The most common complication of anterior cruciate ligament (ACL) reconstruction is loss of extension, which is often functionally worse for patients than their preoperative instability. Many preventable surgical and nonsurgical etiologic factors have been identified. Accurate placement of the tibial tunnel, adequate notchplasty, and the routing of the femoral side of the graft are all critical factors. Several studies report that early range-of-motion therapy emphasizing immediate postoperative "hyperextension" and avoiding immobilization in flexion reduces the rate of loss of extension. Initial studies investigating the effect of acute versus chronic ACL reconstruction suggested that acute reconstruction is associated with a higher rate of loss of extension. However, the authors of two recent studies in which modern techniques were used have disputed this conclusion. It is likely that the loss of extension historically seen with acute ACL reconstructions was related to tibial tunnel placement and postoperative immobilization. It is possible that the timing of acute ACL reconstruction has less of an effect than originally postulated. On the basis of the results of several biomechanical studies, it appears that ACL reconstruction may be performed with the knee in full extension during graft placement with excellent results and a very low rate of loss of extension. Use of the descriptive term "loss of extension" is preferred to the often misleading terms "arthrofibrosis" and "flexion contracture."

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Anterior cruciate ligament (ACL) reconstruction is a commonly performed orthopaedic procedure that generally results in good to excellent functional outcomes. Loss of extension has been reported by many authors to be the most commonly encountered complication after ACL reconstruction, with an incidence as high as 59%¹⁻¹⁰ (Table 1). Loss of flexion, although common after posterior cruciate ligament reconstruction, is rare after ACL reconstruction.^{3,11}

The clinical experience of many authors indicates that a small loss of extension is functionally signifi-

cant to athletically active individuals. Loss of extension is often more detrimental to the patient's functional capability than preoperative instability.^{3,6,12} In 1989, Sachs et al⁶ reported that the three most common complications after ACL reconstruction were flexion contracture, patellofemoral pain, and quadriceps weakness. They maintained that a loss of 5 degrees of extension or more directly causes an abnormal gait, leading to patellofemoral pain and quadriceps weakness. Since then, other authors have agreed with this conclusion.^{3,5,6,12,13}

Many factors have been associated with a high rate of loss of extension, and most of them are preventable. With the use of the modern operative and postoperative techniques reviewed in this article, the incidence and severity of loss of extension after ACL reconstruction should be dramatically reduced.

Etiology of Loss of Extension

Impingement

The etiology of loss of extension after ACL reconstruction is multifactorial. Anterior-intercondylar notch scar tissue, which prevents full extension by mechanically impinging on the roof of the notch (Fig. 1), is the most commonly reported cause of loss of extension.^{1,4,9,13-15} Jackson and Schaefer¹

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Table 1
Loss of Extension After ACL Reconstruction*

Study	Date	Treatment	Incidence of Loss of Extension >5 degrees, %
Sachs et al ⁸	1989	Mixed techniques	25
Strum et al ¹⁰	1990	Surgery within 21 days after injury	35
		Surgery 21 days or more after injury	12
Jackson and Schaefer ⁴	1990	BPTB repair	5.7
Shelbourne et al ⁹	1991	Surgery within 1 week of injury	17
		Surgery 2 to 3 weeks after injury	11
		Surgery 21 days after injury	0
Fisher and Shelbourne ²	1993	BPTB repair	4.4
Dandy and Edwards ⁵	1994	BPTB repair, immobilization in cast	59
Nabors et al ⁶	1995	BPTB repair, tensioned in extension	1.8

* Over the years, the incidence of loss of extension has tended to diminish with the institution of early mobilization, delay of surgery, and graft-tensioning techniques. (The apparent exception is the results of Dandy and Edwards,⁵ but in that study patients were immobilized in a cast.) BPTB = bone-patellar tendon-bone.

referred to this tissue as a "cyclops lesion" in 1990. They reported on a series of 13 patients with loss of extension after intra-articular ACL reconstruction. All 13 underwent arthroscopy, and all were found to have anterior-intercondylar-notch scar tissue arising anterior and lateral to the tibial insertion of the ACL graft. The cyclops nodule was found to act as a mechanical block to extension by impinging on the roof of the notch with terminal extension. Microscopically, the cyclops nodule contained central granulation tissue with peripheral fibrous tissue; in three specimens, cartilaginous tissue was also found.

In 1992, Marzo et al¹⁶ reported on 21 patients with loss of extension after ACL reconstruction with either a bone-patellar tendon-bone

autograft or a hamstring tendon autograft. All 21 patients underwent arthroscopy, and all were found to have a fibrous nodule causing a mechanical block to extension.

In 1993, Fisher and Shelbourne² reported on loss of extension that necessitated reoperation on 42 of 959 consecutive ACL reconstruction patients. Arthroscopy revealed "hypertrophy of the ligament or abundant tissue formation" in the anterior notch.

In 1994, Shelbourne and Johnson¹⁵ reported on 9 patients referred for "arthrofibrosis (loss of more than 15 degrees of extension)" after ACL reconstruction with bone-patellar tendon-bone autograft. At arthroscopy, all patients were found to have anterior-intercondylar-notch scar tissue.

Capsulitis

Capsulitis is inflammation of the capsule, characterized by abnormal periarticular inflammation and edema. Capsulitis may be either a focal or a diffuse process. Focal capsulitis involves an isolated region of the capsule secondary to localized trauma, such as a symptomatic plica, a contusion, or a unilateral ligament injury (e.g., a medial collateral ligament tear). Focal capsulitis may cause pain with motion but rarely leads to a passive loss of flexion and extension.

Diffuse capsulitis is an excessive inflammatory reaction to a stimulus such as surgery, trauma, or infection. Focal capsulitis may progress to total capsular involvement, but the cause of this transition is unclear. Prolonged immobilization may be related. What is clear, however, is that diffuse capsulitis may progress to arthrofibrosis, in which intra-articular scar tissue restricts both flexion and extension.¹² Arthrofibrosis may involve the fat pad, leading to patella infera, or may diffusely involve the entire patellofemoral articulation, leading to patellar entrapment.⁷ These are particularly debilitating problems.



Fig. 1 Inadequate debridement of the old ACL stump or immobilization after reconstruction in flexion can allow the development of scar tissue, which fills the notch and prevents extension.

Although diffuse capsulitis is referred to by some authors as a cause of loss of extension after ACL reconstruction, our review of the literature indicates that diffuse capsulitis or arthrofibrosis is a rare cause of loss of extension. The most common cause is focal anterior-intracondylar-notch scar tissue (a cyclops lesion).^{4,9,11,13,15}

Immobilization in Flexion

In 1994, Dandy and Edwards¹ reported on ACL reconstruction and the causes of loss of extension. In their study, 34 patients underwent reconstruction with bone-patellar tendon-bone autograft, with cast immobilization in flexion postoperatively. In 59% of cases, loss of extension necessitated reoperation. All of these patients underwent arthroscopic surgery, and all were found to have a mechanical block (a nodule of anterior-intercondylar-notch scar tissue) that prevented full extension. The authors concluded that postoperative immobilization in flexion greatly increases loss of extension, and that a cyclops lesion is usually the cause. They also found that flexion contracture and arthrofibrosis were rare.

Other authors have found similarly high rates of loss of extension with postoperative immobilization in flexion. Cosgarea et al¹⁴ reported a decrease in the rate of loss of extension from 23% to 3% when they changed from postoperative bracing in 45 degrees of flexion to bracing in full extension. Of the nine patients referred to Shelbourne and Johnson¹⁵ for loss of extension greater than 15 degrees after ACL reconstruction, all had been immobilized in flexion postoperatively.

Nonanatomic Graft Placement

Current operative techniques used in ACL reconstruction are based on placing the graft in an anatomic location. Extra-articular, nonanatomic reconstructions have

been abandoned by most authors because of their high rate of recurrent instability and late failures. With intra-articular reconstruction, stability has been more successfully achieved; however, nonanatomic placement of the graft with intra-articular reconstruction will often lead to loss of motion, usually extension.¹¹⁻¹³ With placement of the femoral graft in the "over the top" position, the graft is tighter in extension, which may lead to loss of extension.¹² The ideal femoral tunnel is placed in the posterior quartile of the femoral notch, leaving only 1 to 2 mm of posterior wall remaining when the tunnel is drilled (Fig. 2). If the over-the-top position must be used, forming a trough in the condyle is now recommended by most authors.

Graft impingement and loss of extension as a result of anterior placement of the tibial tunnel (Fig. 3) have been observed by a number of authors.¹⁶⁻¹⁸ Marzo et al¹⁶ reported that anterior placement of the tibial tunnel for the graft results in a greater incidence of loss of extension due to formation of a fibrous nodule. They postulated that the anterior graft impinged on the intercondylar roof, injuring the graft and stimulating the formation of the fibrous nodule. Microscopic examination of the nodules revealed findings similar to those reported by Jackson and Schaefer.⁴

In 1991, Howell et al¹⁸ published a study investigating the relationship between tibial tunnel placement and graft impingement. On the basis of an analysis of magnetic resonance (MR) images of 19 knees with normal ACLs, the authors suggested that placing the tibial tunnel in the posterior aspect of the original ACL insertion would require little to no notchplasty to prevent impingement. Placing the tibial graft farther anteriorly increased the amount of bone that would have to be removed during notch-

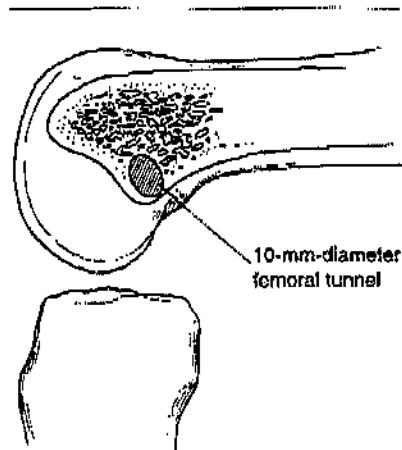


Fig. 2 Poor placement of the femoral tunnel can lead to nonisometric placement of the graft and restricted motion. The ideal placement is at the origin of the ACL on the femur in the posterior quartile of the lateral femoral notch in the 11-o'clock (right knee) or 1-o'clock (left knee) position.

plasty (up to 6 mm) to prevent impingement. The authors recommended notchplasty with more bone resection for all ACL reconstructions performed with an anteriorly placed tibial tunnel. In our opinion, notchplasties may not be necessary if tunnels are appropriately placed, and notchplasties that exceed the space required by the ACL will grow back. Also, the notchplasty may fill in if patients are not allowed to attain immediate full extension to prevent regrowth.

In 1992, Howell and Clark¹⁷ reported on 56 ACL-reconstructed knees that were examined with MR imaging 6 months postoperatively. Thirty demonstrated increased signal in the graft due to impingement; the other 26 did not. Lateral radiographs were taken of all 56 knees to define the location of the tibial tunnel. In the 30 knees with impingement, all the tibial tunnels were placed between 12 and 23 mm from the anterior edge of the tibia. Tunnel placement 22 to 28 mm from the anterior edge of the tibia resulted in

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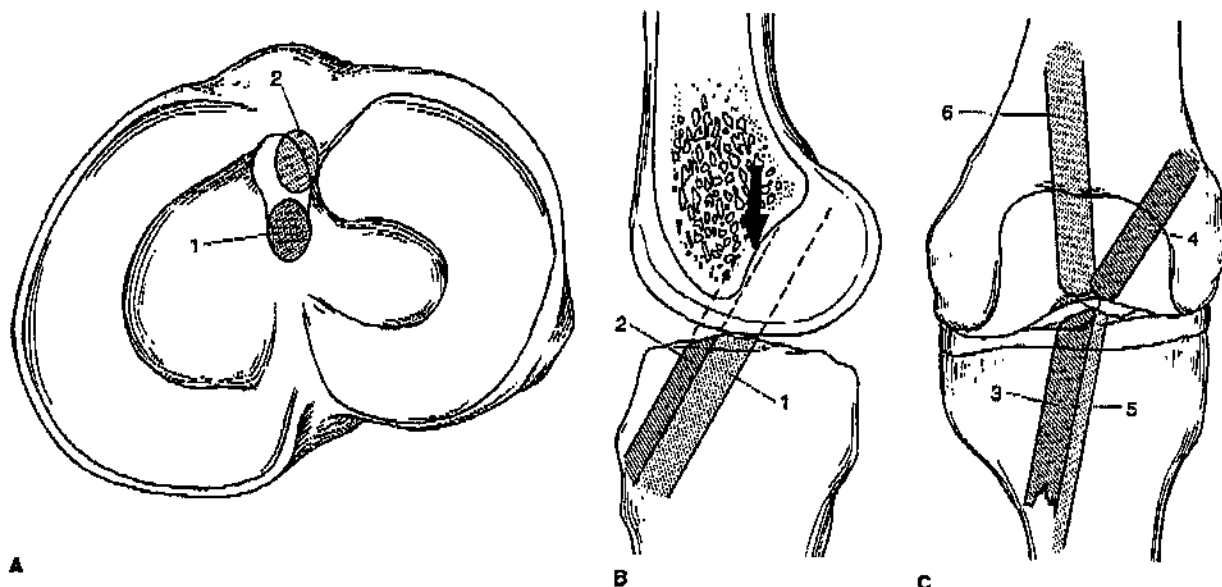


Fig. 3 A, Axial view of the knee demonstrates the normal "footprint" of the tibial insertion of the ACL and the optimal position of the tibial tunnel (1). Anterior tunnel placement (2) leads to anterior impingement on the roof of the intercondylar notch. B, Lateral view demonstrates the ideal tibial tunnel placement (1) in the second quartile of the tibia as measured from anterior to posterior, with the graft lying posterior to the roof of the femoral notch (arrow). Anterior tunnel placement (2) leads to impingement on the roof of the intercondylar notch. C, Anteroposterior view of a left knee demonstrates the ideal placement of the tibial tunnel (3) and the femoral tunnel (4). Lateral tunnel placement (5) can lead to impingement on the lateral condyle. Vertical femoral tunnel placement (6) leads to poor rotational control and recurrent instability.

26 impingement-free knees. In those 26 knees, the tibial tunnels were approximately 3 mm posterior to the center of the original ACL, resulting in improved extension and stability (by KT-1000 arthrometer testing).

In 1993, Romano et al¹⁹ reviewed the radiographs of 111 patients who had undergone ACL reconstruction to determine whether tibial tunnel placement affected final range of motion. Logistic regression analysis showed that loss of extension increased the farther anterior the tibial tunnel was placed. Furthermore, excessive medial tibial tunnel placement was correlated with loss of flexion.

Timing of Surgery

Many articles have evaluated the effect of the time between knee injury and ACL reconstruction on

the ultimate range of motion, with most showing increased loss of motion with early reconstruction. In 1990, Strum et al¹⁰ reported on the rate of loss of motion requiring lysis of adhesions after ACL reconstruction. The incidence was 35% for reconstructions done within 3 weeks of the injury versus 12% for those done after 3 weeks. In 1991, Shelbourne et al⁹ reported on 169 ACL reconstructions. Patients who underwent reconstruction within 1 week of the injury were found to have a higher rate of loss of extension and decreased strength at 13 weeks postoperatively compared with patients who underwent reconstruction 3 weeks or more after injury.

In 1991, Mohtadi et al⁵ reported on loss of motion necessitating manipulation under anesthesia in 37 of 527 patients (7%) following ACL reconstruction. The only variable

associated with a higher rate of knee stiffness was reconstruction within 2 weeks of injury. These results have led many authors to recommend delaying reconstruction until acute edema has resolved and range of motion is at least 0 to 120 degrees.

Despite these recommendations, many authors have continued to perform acute ACL reconstructions with good results. Maraccesi et al²⁰ reported on ACL reconstruction with fascia lata grafts with a ligament augmentation device in 1995. Twenty-three patients were treated within 15 days of injury, and 59 were treated 3 or more months after injury. No difference in the rate of loss of extension was found; however, the early reconstruction group had better results on clinical evaluation and KT-2000 arthrometer laxity testing.

Majors and Woodfin²¹ recently reported a retrospective review of

111 arthroscopic intra-articular ACL reconstructions with bone-patellar tendon-bone grafts. Full extension was obtained in 21 of 21 acute (<2 weeks after injury) reconstructions, 22 of 22 delayed (2 to 4 weeks) reconstructions, and 64 of 64 late (>4 weeks) reconstructions. All 111 were determined to be stable by physical examination and testing with a KT-1000 arthrometer. The authors concluded that the timing of ACL reconstruction does not affect postoperative range of motion, and that a strictly applied program of physical therapy without accelerated rehabilitation is adequate to achieve full range of motion.

Graft Tension

In the eighth edition of *Campbell's Operative Orthopaedics*, 14 authors describe ACL reconstruction techniques.²² Thirteen of the 14 recommend tensioning and securing the graft with the knee in varying degrees of flexion. Most of these authors recommend tensioning the graft in the Lachman position (30 degrees of flexion) while exerting a posterior force on the tibia, despite biomechanical evidence that the ACL is not isometric.

Recent studies have confirmed earlier findings showing that the ACL lengthens 1 to 3 mm in the terminal 30 degrees of extension. In 1990, Bylski-Austrow et al²³ reported on the biomechanics of ACL reconstruction in cadaver knees. Their data showed that knees tensioned in 30 degrees of flexion were overconstrained regardless of the amount of tension at fixation. Reconstructed knees were closest to intact knees when the graft was placed with an initial tension of 44 N while the knee was in full extension during tensioning and fixation.

In 1991, Melby et al²⁴ also reported on the biomechanics of ACL

reconstruction in cadaver knees. They concluded that tensioning at 30 degrees overconstrained the knees. Their data showed that greater initial tension at 30 degrees required greater quadriceps force (up to 26%) to achieve full extension.

Additional studies of anatomic intra-articular ACL reconstructions in cadaver knees have confirmed these results, showing that tensioning at 30 degrees of flexion overconstrains the knee regardless of the amount of force used during tensioning. On the basis of these biomechanical studies, some authors have recommended tensioning and securing the graft with the knee held at full extension.²⁵

Despite the multiple biomechanical studies confirming the 1- to 3-mm lengthening of the ACL in terminal extension, and despite the recommendation by some authors that the graft be tensioned in extension, only one clinical study has been reported in which the ACL was tensioned in full extension. In 1995, Nabors et al⁶ reported on the clinical results obtained with arthroscopically assisted ACL reconstruction with bone-patellar tendon-bone graft. In a prospective study of 57 consecutive patients, the graft was tensioned with maximal one-hand force and secured with the knee in full extension. At the 2-year minimum follow-up, instrumented postoperative laxity testing with a KT-1000 arthrometer revealed an average side-to-side difference of 0.8 mm with a force of 89 N versus 7.5 mm preoperatively. Pivot shift testing was positive in all 57 patients preoperatively. Postoperatively, 51 of 57 (89%) had a negative pivot shift test, 4 (7%) had a pivot glide, and 2 (3.5%) had a true pivot shift. Only 1 patient had loss of extension greater than 3 degrees (specifically, 5 degrees), despite the fact that an accelerated rehabilitation protocol was not used and a

brace with a 10-degree extension block was worn for the first 4 weeks during ambulation. However, the authors did allow immediate active range of motion as tolerated.

Rehabilitation Protocol

A variety of postoperative techniques have been developed to decrease the rate of loss of extension. In 1987, Noyes et al²⁶ reported on early knee motion after ACL reconstruction and concluded that the reconstructed ligament did not stretch out with early motion, and that range of motion was not affected. In 1990, Shelbourne and Nitz²⁷ published their results in 450 patients who underwent accelerated rehabilitation after ACL reconstruction. They encouraged immediate full weight bearing, immediate full extension, early muscle strengthening, and an early return to activity and sports. Only 11 of 247 patients (4%) required reoperation for loss of extension, compared with 16 of 138 patients (12%) in the control group. Long-term evaluation of stability and strength showed no clinically significant differences. In 1993, Fu et al¹¹ reported a reduction in occurrence of loss of extension from 11.1% to 1.7% with aggressive postoperative physical therapy emphasizing early full extension.

A review of the long-term follow-up data on the accelerated rehabilitation protocol disclosed excellent results with regard to preventing anterior knee pain. In 1997, Shelbourne and Trumper²⁸ reviewed the results in 602 patients who underwent ACL reconstructions with bone-patellar tendon-bone autografts between 1987 and 1992. The accelerated rehabilitation protocol was used with emphasis on obtaining immediate postoperative knee hyperextension. The authors examined all 602 patients as well as a control group of 122 patients who had

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no prior knee injury. The results showed no difference in the rate of anterior knee pain in the two groups. The authors concluded that emphasizing immediate postoperative knee hyperextension will prevent anterior knee pain while not compromising long-term knee stability.

Treatment of Loss of Extension

Early diagnosis and treatment of loss of extension may prevent the need for a second operation. In rare instances, capsulitis develops after ACL reconstruction. When this occurs, patients present with diffuse edema, warmth, constant pain, limitation of patellar mobility, and limitation of both extension and flexion.¹³ Late presentation of capsulitis may result in patella infera.⁷ Treatment is usually with nonsteroidal anti-inflammatory agents or a tapered course of methylprednisolone in refractory cases. Gentle physical therapy is indicated, with early efforts directed toward improving extension and quadriceps function. Early manipulation under anesthesia and surgical debridement will only further aggravate the inflammatory process. If loss of extension persists after the inflammation has resolved (which usually takes about 6 months), surgical lysis of adhesions may be considered.¹³

Patients with loss of extension usually have impingement due to anterior-intercondylar-notch scarring. Patients may present asymptotically or complain of anterior knee pain and loss of extension.¹² Physical examination shows that flexion is unaffected. Early treatment is with aggressive physical therapy emphasizing extension and quadriceps-strengthening exercises. The use of an extension drop-out cast at night has been recommended by some authors.¹³ If there is no

improvement after several weeks of conservative treatment, arthroscopic debridement is indicated. Excellent results have been reported with notchplasty enlargement combined with debridement of anterior-intercondylar-notch scar tissue.

Jackson and Schaefer⁴ treated 13 patients with loss of extension. All underwent arthroscopy, all had cyclops lesions, and all improved with arthroscopic debridement and manipulation. Postoperatively, the average loss of extension improved from 16.0 to 3.8 degrees. There were no complications with this treatment. In 1991, Cannon and Vittori²⁹ found a clinically significant benefit with arthroscopic debridement after ACL reconstruction.

In the series of Dandy and Edwards,¹ all 34 cases of loss of extension were due to anterior scar tissue and were relieved with arthroscopic debridement. There were no cases of arthrofibrosis or flexion contracture. The incidence of loss of extension was lowered with notch widening and immediate full extension. The authors concluded that the incidence of loss of extension is increased with immobilization in flexion and is usually due to anterior-intercondylar-notch scar tissue.

In the series reported by Marzo et al,¹⁶ loss of extension due to a fibrous nodule in 21 patients was treated with arthroscopic debridement. The average loss of extension improved from 11 degrees to 3 degrees with surgery and further improved to 0 degrees at 1-year follow-up.

Fisher and Shelbourne² excised the "offending tissue" arthroscopically in 42 ACL-reconstruction patients with loss of extension. The 25 patients available for follow-up at 28 months were all found to have improvement in function and symptoms. Shelbourne and Johnson¹⁵ treated an additional group of 9 patients with arthroscopic anterior scar resection, notchplasty, manipu-

lation, and extension casting; 8 of the 9 achieved near-normal extension. Although these authors refer to the cause of loss of extension as arthrofibrosis, this is misleading because the term "arthrofibrosis" denotes the presence of diffuse scar tissue or fibrous adhesions within the joint, which does not appear consistent with the findings in their studies.

Terminology

A review of the literature shows that failure to regain full extension after ACL reconstruction is the most common complication. Authors have referred to loss of extension by many different terms, but perhaps the two most misleading terms are "arthrofibrosis" and "flexion contracture." The term "arthrofibrosis" is correctly used to describe the formation of diffuse scar tissue or fibrous adhesions within a joint after capsulitis.^{7,13} This usually causes a loss of both extension and flexion. Shelbourne and Johnson¹⁵ have used the term arthrofibrosis to mean loss of more than 15 degrees of extension after ACL reconstruction. We consider this to be misleading because their patients did not have either loss of flexion or diffuse intra-articular fibrosis. We prefer the term "loss of extension," which is a generic descriptive term that neither implies nor excludes any etiologic possibility. "Arthrofibrosis" implies a specific cause and should be used only to describe capsulitis leading to diffuse intra-articular scarring that restricts both flexion and extension.

The term "flexion contracture" has also been used by some authors to describe loss of extension; however, flexion contracture means there is high resistance to lengthening of the flexor muscles or other posterior structures of the knee preventing full extension. In our

review of the literature, neither of these conditions is a common cause of loss of extension after ACL reconstruction; in fact, they occur very rarely. Again, flexion contracture is a specific cause of loss of extension, and it is misleading to use the term generically to refer to loss of extension regardless of cause. Because the terms "arthrofibrosis" and "flexion contracture" imply a specific cause, we believe that the use of these terms has contributed to the failure of many surgeons to recognize that intercondylar-notch scarring is by far the most common cause of loss of extension after ACL reconstruction.

"Cyclops lesion" is the term used by Jackson and Schaefer⁴ to refer to anterior-intercondylar-notch scar tissue that prevents full extension by impinging on the roof of the notch. The expression is easy to remember and emphasizes the singular nature of the commonly found nodule of scar tissue. Unfortunately, the term is not descriptive and has no meaning to a surgeon unfamiliar with it.

Prevention of Loss of Extension

Many of the identified factors associated with loss of extension after ACL reconstruction are easily preventable. Reconstructions performed at least 1 month after injury have been shown by several authors to have a decreased rate of loss of extension. This has led some authors to recommend waiting for acute edema to resolve, for quadriceps function to improve, and for range of motion to be at least 0 to 120 degrees before undertaking surgery. However, there are many confounding variables in these preliminary studies, and two recently published reports dispute those recommendations.^{20,21} A large prospective study with iden-

tical surgical and rehabilitation techniques for both groups is necessary before any clinical recommendations can be made.

Intraoperatively, the key to avoiding loss of extension is careful anatomic placement of the graft tunnels. It has been proved that placement of the tibial tunnel anterior to the center of the original ACL insertion site will cause impingement and loss of extension.¹⁶⁻¹⁹ Furthermore, inadvertent anterior drilling of the tibial tunnel despite accurate placement of the guide wire has been described.¹² Thus, it is imperative that great care be taken during placement of the tibial tunnel, and that adequate notchplasty be performed as needed for all reconstructions.

Techniques to ensure proper tibial tunnel positioning include referencing anatomic landmarks, including the posterior cruciate ligament, the posterior horn of the meniscus, the medial tibial eminence, and the roof of the notch; preoperative x-ray evaluation of the tibia-notch relationship; and intraoperative radiography or other imaging. Testing for impingement before graft insertion and fixation is valuable.¹⁷ A large roof notchplasty may compensate for far-anterior placement of the tibial tunnel; however, this may not be ideal and can be associated with degenerative joint disease. A femoral tunnel is preferable to placing the graft over the top of the condyle because of the tensioning issues discussed previously.¹² Another intraoperative technique associated with very low rates of loss of extension is tensioning the graft with the knee in full extension. Several biomechanical studies and one clinical study strongly support this technique.^{6,10,23,24} One study showed a higher rate of loss of extension with use of autograft versus allograft.³ It was hypothesized that bone-patellar tendon-bone harvest-site pain pre-

vents full early extension; however, this was the only study in which this conclusion was drawn.

There are several postoperative techniques for the prevention of loss of extension. It has been definitively proved that postoperative immobilization in any amount of flexion is deleterious.^{1,7,12,14} Immediate emphasis on obtaining full extension is clearly the most important factor in preventing loss of extension.³⁰ It has been hypothesized that immediate full extension engages the ACL graft in the notch and, by occupying this space, prevents the formation of anterior-intercondylar-notch scar tissue. Postoperative immobilization in extension may prevent fibrin clot from forming in the notch and thus prevent scar tissue formation.

Accelerated rehabilitation has been shown by a large number of authors to decrease the rate of loss of extension. Additionally, longer follow-up of Shelbourne's original group of patients treated with accelerated rehabilitation²⁷ has shown that function and stability are not adversely affected by immediate postoperative full-knee hyperextension.^{28,30} Other authors have applied Shelbourne's accelerated rehabilitation protocol²⁷ to patients undergoing ACL reconstruction with semitendinosus and gracilis tendon grafts. The results have shown similarly decreased rates of loss of extension with no loss of stability.

Continuous-passive-motion machines are used by a number of authors in the early postoperative stage. One study found no benefit from routine use after ACL reconstruction.³¹ Others argue that continuous passive motion may help to improve flexion in patients at risk for loss of flexion but is of little use in improving extension.¹³ In general, as the continuous-passive-motion device reaches full extension, the restricted knee simply remains

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slightly flexed. New machines have been designed with anterior straps or hinges locked to the machine to achieve complete extension, but no study has been performed on patients after ACL reconstruction to confirm their efficacy.

Summary

Loss of extension is the most common complication of ACL recon-

struction. Various intraoperative and postoperative techniques are useful in markedly decreasing the rate of loss of extension: careful anatomic placement of graft tunnels; strict avoidance of anterior placement of the tibial tunnel; avoidance of over-the-top placement of the femoral graft; utilization of a trough in the condyle if over-the-top placement must be employed; use of the intraoperative impingement test before graft tensioning; tensioning

the graft with the knee in full extension; encouragement of immediate postoperative full-knee hyperextension; strict avoidance of immobilization in flexion or restriction of full hyperextension in any way; and early diagnosis and appropriate treatment of loss of extension. It is recommended that, for greater clarity of expression, authors should adopt the term "loss of extension," rather than "arthrofibrosis" or "flexion contracture."

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