

Current Concepts

Motion Loss after Ligament Injuries to the Knee

Part I: Causes

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ABSTRACT

Motion loss continues to be a difficult complication after ligament injury and surgery to the knee. A better understanding of the pathoanatomic causes of motion loss can lead to improved prevention and treatment strategies. When motion loss does occur, early recognition and appropriate treatment can be expected to restore motion and improve function in most patients. Treatment options, although varied, should improve outcome when implemented appropriately. This article is composed of two parts. The first part reviews the current concepts on definitions, incidence, and causes of motion loss. In the second part, to be published later, current strategies on prevention and treatment of motion loss after ligament injury to the knee are reviewed. Emphasis is placed on risk factors and prevention as well as on diagnosis and treatment. The article summarizes the latest information from the basic sciences as well as clinical studies on the problem of motion loss of the knee and attempts to provide a rational approach to these difficult clinical problems.

The purpose of this review is to summarize the current concepts on motion loss of the knee, placing particular emphasis on the causes, prevention, and treatment. The article is in two parts. This first part will discuss causes of motion loss after ligament injury or surgery. The goals of the first article are to discuss normal and abnormal knee

kinematics and to define terminology for motion problems of the knee. Known risk factors, causes, and pathoanatomy will be discussed in detail, summarizing the current and pertinent studies on this difficult clinical problem.

Loss of motion after ligament injuries to the knee is a common problem that may involve loss of flexion, extension, or both. When patients have loss of both flexion and extension, the loss of flexion is usually better tolerated and is also easier to treat.^{27,28}

While normal knee range of motion varies from person to person, most people display some degree of hyperextension, with an average of 5° of recurvatum in men and 6° in women.^{10,61} Hyperextension of the knee is necessary for two reasons: first, it allows the normal “screw home” mechanism to occur, and second, it allows the knee to be “locked-out” during the stance phase, allowing the quadriceps muscle to relax.

Normal knee flexion is approximately 140° in men and 143° in women.¹⁰ Small flexion deficits typically do not alter gait; however, most people readily notice an asymmetric loss of flexion (unilateral), particularly those who are involved in running or jumping sports. Therefore, loss of motion in the knee causes significant problems, particularly when it occurs in young, active athletes. Most patients do not tolerate the inability to perform everyday activities such as squatting, running, and bicycling. In severe cases of flexion loss, stair climbing and sitting ability become affected. Early recognition and aggressive intervention can usually prevent significant disability and permanent functional impairment.

Most people find the loss of extension more disabling than the loss of flexion, as relatively small extension deficits place undue strain on the quadriceps muscle and patellofemoral joint.⁴ Without full extension, patients are unable to stand on the affected leg with the quadriceps

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muscle relaxed. In the classic cadaveric study by Perry et al.,⁴⁸ the forces in the quadriceps muscle, patella, and tibia during flexed-knee stance were measured. The quadriceps muscle force required to stabilize the knee was 75% of the load on the femoral head at 15° of knee flexion, 210% at 30°, and increased to 410% at 60°. Stresses at the tibiofemoral and patellofemoral joint surfaces increased in a similar fashion. Furthermore, the authors found that at 15° and 30° of flexion the quadriceps muscle force was equivalent to 20% and 50%, respectively, of the average maximum quadriceps muscle strength. This study demonstrates how loss of extension leads to significant increases in joint contact pressures, quadriceps muscle activity, and fatigue.

TERMINOLOGY

The literature is replete with various descriptions of motion problems around the knee. Terms such as “arthrofibrosis,” “flexion contracture,” and “ankylosis” may be confusing, misleading, or misunderstood. The generic terms “extension loss” and “flexion loss” more precisely describe any deviation in knee motion from the normal (contralateral) side. These terms, while precise and descriptive, do not imply cause and therefore may be used to describe motion problems that result from a variety of causes.⁴⁹

The term arthrofibrosis has been used to mean flexion loss, extension loss, or both. For the purposes of discussion in this article, the term will only be used when the specific cause (fibrosis and abnormal scarring of the joint) is implied. Arthrofibrosis is a specific process in which scar

tissue or fibrous adhesions form diffusely within a joint.⁴⁹ A thickened, fibrotic capsule, which in its most severe forms can completely prohibit joint motion, is characteristic.

The term flexion contracture is defined as a loss of extension due to a relative shortening of the posterior soft tissue structures of the knee (either capsular or muscular). It is a specific cause of extension loss. Because of this shortening, the soft tissues cannot attain their normal passive length and a loss of extension results. In many instances, the term flexion contracture has been used incorrectly and generically to describe any loss of extension. To eliminate confusion, the term flexion contracture should only be used when the specific cause (shortening of the posterior soft tissues) is implied.

Ankylosis is a nonspecific term that has been used to describe stiffness of joints. It has been defined as abnormal stiffness, immobility, or consolidation of a joint that can result from bony, cartilaginous, or fibrous tissue overgrowth. Loss of extension, flexion, or both can be implied by the term ankylosis; moreover, a specific diagnosis or cause is usually not implied.

For the purposes of discussion in this article, the term “motion loss” will be used to describe deviations from normal knee kinematics, and the more specific terms, extension loss, flexion loss, or both, will be used to specify which particular knee motion has been affected.

INCIDENCE

The true incidence of motion problems after ligament injury to the knee is unknown and varies according to the

TABLE 1
Incidence of Motion Problems after Knee Injury

Study	Year published	Patients	Treatment ^a	Incidence (%)	How defined
Johnson et al. ²⁹	1984	87	ACL reconstruction (B-PT-B autograft)	Extension 67.9 Flexion 76.5	Compared with contralateral
Fried et al. ¹⁷	1985	40	ACL reconstruction (PT with ITB transfer)	Extension 25 Flexion 22.5	>5° loss of extension >5° loss of flexion
Sisto and Warren ⁶⁴	1985	20	Multiple ligaments	65	Not stated
Zarins and Rowe ⁷²	1986	100	ACL reconstruction (ST/G and ITB)	10	>5° loss of extension
Kornblatt et al. ³⁰	1988	38	ACL reconstruction (Quadriceps tendon)	74	>5° loss of extension
Jackson and Schaefer ²⁸	1990	230	ACL reconstruction (B-PT-B autograft)	(none > 15° total motion) 6	>10° loss of flexion >10° loss of extension
Harner et al. ²⁴	1992	240	ACL reconstruction (mixed grafts)	11.3	>10° loss of extension <125° of flexion
Noyes et al. ⁴³	1992	207	ACL reconstruction (B-PT-B allograft)	9	<0°–135°
Fisher and Shelbourne ¹⁵	1993	959	Open ACL reconstruction (B-PT-B autograft)	4	Any loss of extension with pain, stiffness, inability to return to activity
Cosgarea et al. ⁸	1995	188	ACL reconstruction (B-PT-B autograft)	12	>10° loss of extension <125° of flexion
Noyes and Barber-Westin ⁴²	1997	11	Multiple ligaments	45	Deviation from contralateral
Shelbourne and Gray ⁵⁸	1997	1057	ACL reconstruction (B-PT-B autograft)	7 in 1987 1 in 1993	Symptomatic lack of extension compared with the other side
Plancher et al. ⁵⁰	1998	72 (75 knees)	ACL reconstruction (B-PT-B autograft)	4	>10° loss of extension

^a B-PT-B, bone-patellar tendon-bone; ITB, iliotibial band; ST/G, semitendinosus and gracilis graft.

specific injury, treatment modality, and how motion loss is defined (Table 1). Shelbourne and Rask,⁶¹ in an attempt to precisely define loss of motion, stressed the importance of using the contralateral knee for comparison. Any deviation from that of the contralateral normal limb should be considered abnormal. Many studies define motion loss as a deviation of 5° from full extension; others define it as a deviation from the contralateral side. Unfortunately, most studies only report motion in the involved limb. As such, it is possible that the true incidence of motion problems about the knee may be underreported.

The incidence of motion loss after ACL reconstruction has decreased with a better understanding of risk factors, with proper surgical technique, and with early, aggressive rehabilitation.^{8,17,24,29,30,50,63,72} With isolated ACL tears, most problems with motion can be avoided. Harner et al.,²⁴ in a retrospective review of 240 patients with minimum follow-up of 1 year, found motion problems (defined as a loss of extension of 10° or greater, or flexion less than 125°) in 27 patients (11%) who underwent ACL reconstruction. Jackson and Schaefer²⁸ reported a 6% incidence (13 of 230) after intraarticular ACL reconstruction with autogenous bone-patellar tendon-bone. More recently, a study has reported incidences as low as 2% with careful attention to these factors.⁶⁰

Motion problems remain prevalent, however, in knees with multiple ligament injuries and in knees with dislocations.^{42,56,64} Sisto and Warren⁶⁴ noted motion problems in 6 of 20 patients (30%) with traumatic knee dislocations. In another study, four of seven patients (57%) who underwent combined ACL and PCL reconstructions for knee dislocations required an additional procedure (either manipulation or surgery) to restore motion.⁵⁶

RISK FACTORS

A variety of risk factors have been shown to be associated with loss of knee motion (Table 2). These can be conveniently divided into factors associated with the injury itself, the treatment, or the rehabilitation.

It is not known why the knees of certain patients form abnormal scars and others do not. Although as yet unproven, it seems likely that certain patients have a genetic predisposition that places them at a higher risk for motion problems.

Mechanism and Associated Injuries

Certain soft tissue injuries have a very high association with motion loss; patients with multiple injured ligaments

and knee dislocations are perhaps among the most at risk.^{5,13,42,56,64} In the knee with multiple injured ligaments or in the dislocated knee, the magnitude of the soft tissue injuries both at the time of injury and again at the time of surgical reconstruction places them at significant risk.³⁶ The associated soft tissue injury to the medial side may be the injury that causes stiffness. Knee dislocations and multiple injured ligaments typically occur from high-energy forces and in the setting of polytrauma, both of which can dictate surgical timing, technique, and postoperative rehabilitation.

Timing of Surgery

For acute, isolated ACL injuries, the timing of ligament reconstruction and its role in motion loss are among the most controversial subjects related to reconstructive knee surgery. Unfortunately, the lack of uniformity among patient selection, surgical technique, and follow-up data makes it difficult to interpret the results from different case series. The definitions of full extension and of motion loss vary among studies. Nevertheless, many authors have reported an association between early surgery and the development of arthrofibrosis (Table 3)^{24,59,63,68}; others, however, have found no relationship between the timing of surgery and the development of motion problems.^{2,26,32,33,67}

Shelbourne et al.⁶³ retrospectively studied the association between the timing of surgery for ACL reconstruction and the development of motion loss in a group of 169 patients. Patients whose ligaments were reconstructed within the 1st week of injury had a statistically significant increase in the incidence of motion loss when compared with those who waited at least 3 weeks. Wasilewski et al.⁷⁰ retrospectively reviewed the effects of surgical timing on recovery and on associated injuries. A correlation was noted between both slower recovery and the incidence of motion loss in knees that were reconstructed acutely (5 to 10 days after injury). In their study, a delay in surgery (up to 6 months) did not adversely affect outcome. Other studies, however, suggest that the outcomes after acute ACL reconstruction in appropriately selected patients may be equivalent to those from patients who have waited 3 to 6 weeks for surgery.^{26,32} Hunter et al.²⁶ reported 185 patients who were studied prospectively and found timing of surgery to be independent of motion problems.

Currently, it appears that delaying surgery when there is persistent loss of motion, inflammation, or swelling decreases the risk of motion problems. Advantages of waiting include mental and physical preparation by the

TABLE 2
Risk Factors for Motion Loss

Patient	Injury	Surgical	Postoperative
Genetic predisposition (?)	Multiple ligaments	Timing of surgery	Prolonged immobilization
	Dislocations	Preoperative motion	Poor rehabilitation
	Infection	Malpositioned grafts	Reflex sympathetic dystrophy
	Synovitis	Excessive graft tension (if nonanatomic)	Infection
		Associated extraarticular procedures	Synovitis
		Associated meniscal repairs	

TABLE 3
Effect of Timing of Surgery on Incidence of Motion Problems

Study	No. of patients	Technique ^a	Timing	Definition of motion loss	Early surgery associated with motion loss?
Strum et al., 1990 ⁶⁸	156	ACL repair or reconstruction, immobilized postop	I, <3 weeks; II, >3 weeks	Loss of extension from 0° and flexion from 135°	Yes
Shelbourne et al., 1991 ⁶³	169	ACL reconstruction, open, B-PT-B	I, less than 1 week; II, 1–3 weeks; III, >3 weeks	>5° loss of extension or flexion as compared with contralateral side	Yes
Harner et al., 1992 ²⁴	240	ACL reconstruction (mixed grafts)	Less than 4 weeks	>10° loss of extension and <125° of flexion	Yes
Wasilewski et al., 1993 ⁷⁰	87	ACL reconstruction, doubled semitendinosus	Acute, less than 1 month; Subacute, 1–6 months; Chronic, >6 months	Any loss of extension or flexion as compared with contralateral side	Yes
Shelbourne and Johnson, 1994 ⁶⁹	9	ACL reconstruction, 8 B-PT-B 1 Semitendinosus with MCL advancement	<2 weeks	>15° loss of extension	Yes
Bach et al., 1994 ²	62	ACL reconstruction, B-PT-B	Acute, <3 weeks; chronic, >3 weeks	Prone heel-height differences	No
Marcacci et al., 1995 ³³	82	ACL reconstruction, B-PT-B or, fascia lata and LAD	I, <15 days; II, >3 months	Compared with contralateral	No
Steadman et al., 1995 ⁶⁷	160	ACL reconstruction, B-PT-B	I, <1 week; II, 1–3 weeks; III, >3 weeks	Less than 10°–120°	No
Hunter et al., 1996 ²⁶ Prospective study	185	ACL reconstruction, B-PT-B	I, <3 days; II, 3–7 days; III, 7–21 days; IV, >21 days	Loss of full extension or flexion (0°–135°)	No
Majors and Woodfin, 1996 ³²	119	ACL reconstruction, B-PT-B (118), synthetic (1)	I, <2 weeks; II, 2–4 weeks; III, >4 weeks	Loss of full extension (0°)	No

^a B-PT-B, bone-patellar tendon-bone; MCL, medial collateral ligament; LAD, ligament augmentation device.

patient. Before surgery, full motion, strength, and appearance of the knee can also be achieved. The main advantage of intervening acutely is less time lost by the patient before returning to work and athletic activity,⁷⁰ although the authors in one study have found that a delay in surgery may result in faster return to activity.⁵⁷

Some authors have called this so-called “golden time period” into question.² Hutton and colleagues (unpublished data, 1995) reported their experience with early surgical reconstruction of the ACL and found no difference in patients operated on early (less than 1 week) versus those operated on late (1 to 3 weeks and greater than 3 weeks). Bach et al.² were unable to detect a difference in the incidence of motion loss after arthroscopically assisted ACL reconstruction when comparing acute versus chronic reconstructions. This controversy led the AOSSM to publish a position statement on the topic, stating that “if a knee injury such as an ACL sprain or tear occurs, there is no minimum time period in which these injuries need to be repaired. It is often possible to return home before further treatment (or surgery) is needed.” We believe that, in certain more complex knee injuries, early surgery may be necessary. Early surgery may facilitate surgical dissection, repair, or reconstruction because the knee is less distorted by the fibrous healing response.⁶⁸

Preoperative motion is an important preoperative predictor of ultimate motion and may even be the key clinical factor to help in decisions regarding timing of ACL reconstruction.⁸ Cosgarea et al.,⁸ in their retrospective analysis of 191 consecutive patients, found a statistically significant increase in the incidence of motion loss in those

patients who lacked 10° or more of extension preoperatively. Nine of 38 patients (24%) with preoperative extension deficits of 10° or greater developed motion loss, compared with 13 of 150 patients (8.7%) with preoperative extension deficits of less than 10°.

A normal inflammatory healing response begins after injury.⁶⁷ However, in some acutely injured knees, this response may be excessive, leading to disordered healing and motion problems. It has been our experience that this response is variable from patient to patient: some have a minimal inflammatory response with little swelling and normal knee motion. In these patients, ligament reconstruction can safely be performed early. Conversely, some patients display a heightened response with significant inflammation. Most experts would agree that, in such patients, reconstructive surgery should be delayed until the swelling resolves and full motion has been restored.^{60,67} Most patients fall somewhere between these categories and, unless there are multiple ligament injuries that necessitate early surgery, we prefer to delay surgical reconstruction until swelling subsides, pain is alleviated, and normal motion is achieved.

Technical Factors

Many technical factors have also been associated with postoperative motion loss.^{28,65,66} Proper graft positioning is essential to prevent graft impingement (Fig. 1). A malpositioned or nonisometric graft is a common cause of motion deficit after ACL reconstruction.^{19,40} Grafts that are too tight or placed in the wrong position can obviously

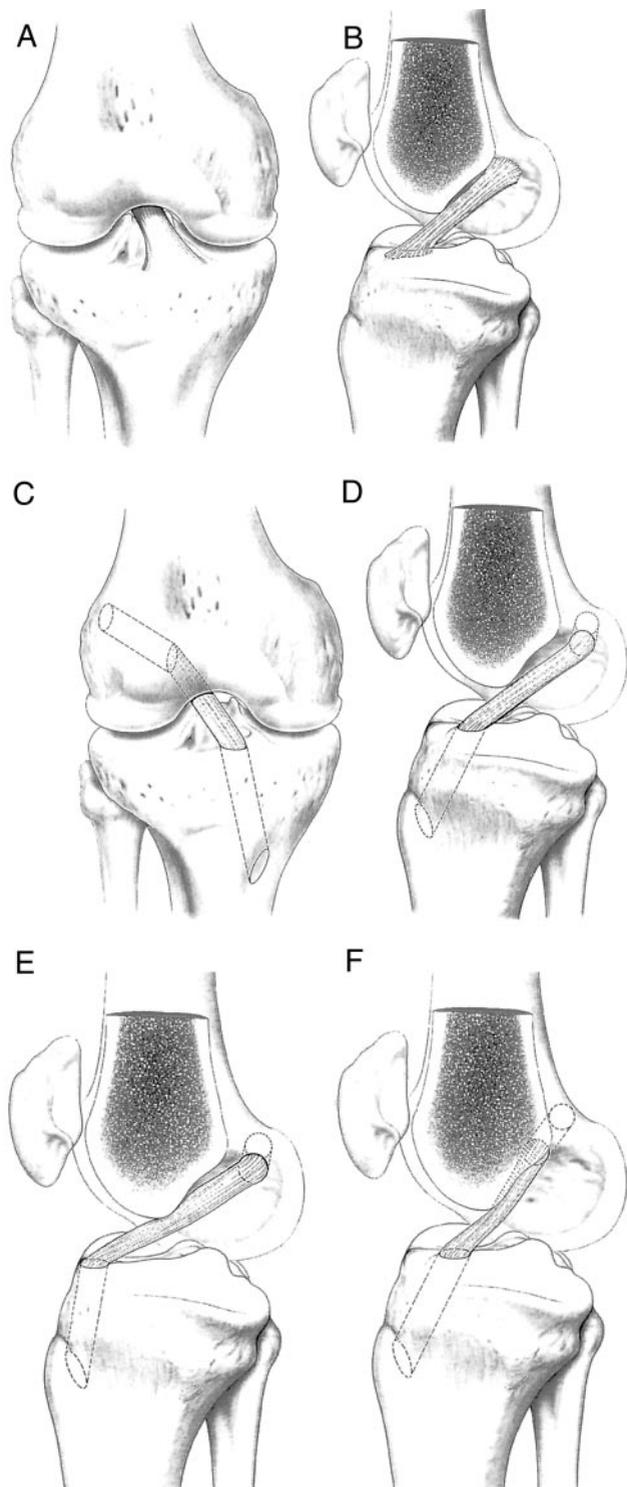


Figure 1. Proper tunnel placement is essential to prevent graft impingement and motion loss. A and B, normal ACL anatomy; C and D, proper ACL graft position; E, the effects of anterior tibial tunnel placement. Notice the impingement of the graft in the intercondylar notch during terminal extension. F, the effects of anterior femoral tunnel placement. Notice the impingement of the graft in the intercondylar notch with extension. This results in loss of extension or graft failure.

interfere with normal knee kinematics.⁴⁹ Graft position must be accurate in all planes and at both the tibial and femoral tunnels.

Extraarticular procedures also increase the risk for the development of motion loss.^{24,46} Harner et al.²⁴ found that procedures that involved the medial capsule, particularly medial collateral ligament repair and posterior oblique ligament reefing, were associated with a higher incidence of motion loss (Fig. 2). They postulated that the normal tissue planes may be disrupted and subsequently interfere with motion. Alternatively, if the medial collateral ligament is fixed too close to the tibia or femur, excursion is lost and motion is blocked. Furthermore, the trauma of surgery can accelerate the inflammatory healing response of the knee, leading to excess scar tissue and, in severe cases, calcification of the medial collateral ligament. Quadriceps muscle inhibition from extensive dissection also leads to decreased motion and stiffness.

Postoperative/Rehabilitation Factors

Prolonged immobilization has detrimental effects on periarticular cartilage, bone, and soft tissues and can lead to motion loss.^{14,46,64} Modern rehabilitation programs have stressed early motion and weightbearing, resulting in fewer motion problems and better outcomes.^{42,43,58,63} Several researchers have shown that patients who participated in early motion and weightbearing had a decreased incidence of motion loss and regained extension more quickly.^{8,58,63} Failure to achieve terminal hyperextension will result in poor results, as reported by Shelbourne et al.⁶⁰ Obviously, poor rehabilitation, whether because of lack of motivation or lack of instruction, will adversely affect the ultimate outcome.

When bracing is needed to maintain extension, the knee



Figure 2. The AP (A) and lateral (B) radiographs of a patient who underwent a combined ACL/MCL (medial collateral ligament) reconstruction. The patient developed a motion problem postoperatively because of arthrofibrosis. The addition of an extraarticular procedure increased the risk of motion problems.

should be splinted in full extension and motion should be started as soon as possible.^{8,40} Terminal hyperextension should be the goal. In one retrospective analysis of consecutive patients who underwent ACL reconstruction using the central third of the patellar tendon, 22 of 188 patients (12%) developed arthrofibrosis.⁸ These authors reported a 23% incidence of motion problems when patients were braced in 45° of flexion and waited 1 week to start passive extension, versus a 3% incidence when the knee was braced in full extension with motion starting within 24 hours.

Reflex sympathetic dystrophy is a regional pain syndrome that has been associated with loss of motion in the knee.^{23,45} Reflex sympathetic dystrophy may cause motion problems because of swelling, which is usually extraarticular, or because of increased sensitivity to pain.⁴⁵ In patients with reflex sympathetic dystrophy, it is essential to establish a precise diagnosis before contemplating any rehabilitative or surgical intervention, as the insult may exacerbate symptoms. Continuous epidural analgesia is often helpful in breaking the cycle of pain and in improving motion.⁷ When reflex sympathetic dystrophy is noted preoperatively, it is often necessary to delay surgery and take a multispecialty approach to treatment that includes orthopaedic surgeons, anesthesiologists, and pain management specialists.^{6,45}

Infection may also contribute to motion loss. Joint infections stimulate an aggressive inflammatory response that results in synovitis, enzymatic cartilage degradation, and fibrotic scar deposition. The pain that infection causes further inhibits motion and leads to joint stiffness. Prompt recognition and early treatment of the septic knee are necessary for prevention of these sequelae.

PATHOANATOMY

Loss of motion in the knee is a complex, multifactorial process with a variety of risk factors. The pathoanatomy of motion loss similarly spans a broad spectrum. While most motion problems are due to simple mechanical blocks to extension or flexion,⁹ it is important to understand the myriad causes of motion loss so that appropriate preventive, diagnostic, and treatment strategies can be implemented.

Arthrofibrosis

Arthrofibrosis occurs when diffuse scar tissue or fibrous adhesions form within a joint.⁴⁹ The periarticular scarring restricts both flexion and extension and may occur as a localized or a global process. Cytokines stimulate a cellular response that results in fibrosis of the suprapatellar, medial, and lateral gutters. Fibrosis occurs in the anterior compartment and frequently in the posteromedial and posterolateral capsule as well.

Sprague et al.,⁵⁶ who were among the first to describe the phenomenon of arthrofibrosis in 1982, published a classification scheme with three groups based on the pathoanatomic findings. Group one describes patients with discrete bands or a single sheet of adhesions travers-

ing the suprapatellar pouch, while group two describes patients with complete obliteration of both the suprapatellar pouch and the peripatellar gutters with masses of adhesions. Group three describes the most severe form, in which there is complete obliteration of the suprapatellar pouch and peripatellar gutters combined with extracapsular involvement with bands of tissue extending from the proximal patella to the anterior femur. Patients with severe involvement (group three) were unable to regain flexion even after arthroscopic debridement. Millett et al.⁴⁰ reported a group of patients with profound motion loss of the knee secondary to severe arthrofibrosis. The eight patients in their series had abundant scar tissue, both intraarticular and extraarticular, which blocked both flexion and extension.

ACL Nodule

An ACL nodule is a fibroproliferative scar nodule that occurs after ACL reconstruction with bone-patellar tendon-bone autograft (Fig. 3). It is most commonly located anterolateral to the tibial tunnel.^{21,28,52} Jackson and Schaefer²⁸ were among the first authors to describe the ACL nodule, which they called a "cyclops lesion." The nodules, which occur in 2% to 4% of ACL reconstructions, are typically attached to the graft as well as to the soft tissue overlying the tibiae. Microscopically, they are composed of dense fibrous tissue with a central area of granulation tissue. With extension, impingement occurs between the ACL nodule and the intercondylar notch, blocking terminal extension. Hypertrophy of the graft or other soft tissue scarring in the notch can cause a similar block to extension.^{15,37}

Marzo et al.³⁷ reported a group of 21 patients who developed restricted knee extension after ACL reconstruction using either the central third of the patellar ligament or the hamstring tendons as an autogenous graft. The patients were seen at an average of 4 months postoperatively with a clinical syndrome of loss of extension associated with pain at terminal extension, crepitus, and grinding with extension. The consistent finding at arthroscopy was a fibrous ACL nodule occupying the intercondylar notch, varying in size from 1 by 1 cm to 2 by 3 cm. The ACL nodule acted as a mechanical block to full extension. The authors hypothesized that anterior placement of the graft, particularly on the tibia, resulted in injury to the graft and subsequent nodule formation. Histologic studies of the nodule revealed disorganized, dense fibroconnective tissue that, with time, underwent modulation to fibrocartilage. The authors postulated that this occurs in response to compressive loading of the nodule.

In another study on the pathogenesis of ACL nodules, Delincé et al.¹² performed second-look arthroscopic evaluations of patients with ACL nodules. The authors believed that the cause of the nodules was most likely multifactorial with a natural, fibroproliferative tissue response that originated from either debris from the tibial tunnel, remnants of the ACL stump, or, more rarely, from broken graft fibers.¹⁻³ Delcogliano et al.¹¹ performed a light and electron microscopic analysis of ACL nodules

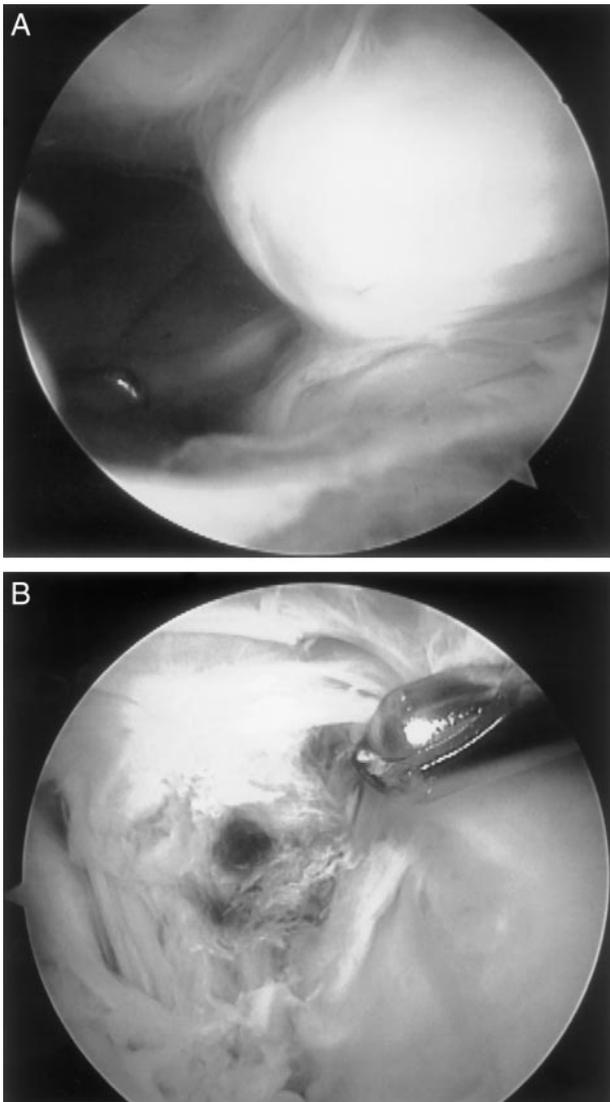


Figure 3. A, intraarticular ACL nodule. This arthroscopic view demonstrates a typical ACL nodule that occurs after ACL reconstruction. The nodule caused a loss of extension that was symptomatic and required surgical debridement. B, the view after debridement of the nodule. Full extension of the knee was achieved postoperatively.

from four patients. They noted neovascularization with vessels consisting of hyperplastic and hypertrophic cells that were surrounded by bundles of disorganized fibrous tissue. Inflammatory cells, bone, and cartilaginous tissues were not seen. In both of these studies, repeated micro-trauma that damaged and exposed the collagen graft fibers was postulated as the cause for nodule formation. Shelbourne and Trumper⁶² have proposed that anterior knee pain and ACL nodules can be prevented if terminal extension is achieved during rehabilitation because the ACL graft in the intercondylar notch may block nodule formation.

We have also seen bony nodules form at the tibial insertion of the ACL (Fig. 4). These also act as a mechanical

block to extension. Whether they result from bony overgrowth of the tibial tunnel or from repetitive trauma remains unclear. In 1994, Puddu and coworkers⁵¹ reported 20 patients (22 knees) treated by arthroscopic removal of an anterior tibial osteophyte. The patients in this series had loss of extension (ranging from 5° to 20°) that was associated with pain and discomfort in recreational sporting activities and in activities of daily living. Radiographs demonstrated an anterior tibial osteophyte just anterior and medial to the anteromedial tibial spine of the intercondylar eminence. The patients were treated arthroscopically with excision of the osteophyte and a moderate notchplasty with good relief of symptoms and restoration of motion. Another study has reported extension loss due to notch hypertrophy and scarring after nonoperative treatment of a type III tibial avulsion fracture.¹⁶

Infrapatellar Contracture Syndrome

Paulos et al.⁴⁶ were among the first researchers to describe the infrapatellar contracture syndrome as an unrecognized cause of posttraumatic knee stiffness with patella entrapment and patella infera (baja). Patients who develop infrapatellar contracture syndrome have a combination of restricted knee extension and flexion associated with patellar entrapment. The cause is an exaggerated pathologic fibrous hyperplasia of the anterior fat pad.^{46, 47} Prolonged immobility and lack of extension, particularly after intraarticular ACL reconstruction, may also be contributing factors. The fat pad becomes densely adherent to the underlying tibia, resulting in diminished excursion of the patella and loss of motion.

Other authors have reported similar findings.^{44, 53} Noyes et al.⁴⁴ reported a group of five patients in whom contracture of peripatellar tissues, fat pad tissues, and quadriceps muscle weakness developed. The process eventually progressed to patella infera and patellofemoral arthrosis and was termed “patella infera syndrome.” All patients required arthroscopic treatment.

In 1994, Paulos et al.⁴⁷ reported the long-term outcome of infrapatellar contracture syndrome in 75 patients (76 knees) evaluated at an average of 53 months after the initial procedure or injury. Poorer results were correlated with acute surgery, use of patellar tendon autograft for ACL reconstruction, nonisometric graft placement, multiple surgical procedures, use of closed manipulation, and the development of patella infera. In their study, 80% of patients (60) demonstrated patellofemoral arthrosis and 16% (12) demonstrated patella infera. The authors concluded that prevention or early detection and aggressive treatment are the only ways of avoiding complications; they cautioned that the natural history of an ACL-deficient knee appeared to be more benign than the natural history of a knee that develops infrapatellar contracture syndrome.

Malpositioned Graft

Motion loss may also be the result of a technical error such as a malpositioned graft that impinges or an insufficient



Figure 4. The AP (A) and lateral (B) radiographs and an arthroscopic view (C) of an intercondylar bony nodule (osteophyte) that caused loss of terminal extension. Simple arthroscopic debridement restored the patient's knee motion.

notchplasty that blocks full extension.^{1,20,40} In our experience, a tibial tunnel that is placed too far anteriorly results in limited flexion and graft impingement when the knee moves into extension. This can contribute to excessive scarring on the graft, development of an ACL nodule, or graft failure.

In a cadaveric model, Yaru et al.⁷¹ performed ACL reconstructions using seven different tibial attachment sites. The tibial attachment site affected both range of motion and impingement in the intercondylar notch. Grafts placed anterior and lateral to the insertion of the anterior fibers of the ACL consistently produced impingement in the intercondylar notch. Based on their data, the authors concluded that optimum placement of the

tibial tunnel should be at the insertion of the anteromedial fibers of the ACL. They also recommended 3 mm of graft clearance during passive range of motion to prevent impingement in the intercondylar notch during active extension.

In 1993, Romano et al.⁵⁴ reviewed the radiographs of 111 patients who had ACL reconstructions; postoperative radiographic measurements of the tibial tunnels were correlated with the final range of motion achieved. In the 25 patients with extension deficits of 10° or more, placement of the tibial tunnel was more anterior than in the remaining 86 patients with extension deficits of less than 10°. Using regression analysis, they found that the more anterior the placement of the tibial tunnel, the greater the loss

of both flexion and extension. In the 21 patients with full extension but flexion less than 130°, placement of the tibial tunnel tended to be more medial than in the 65 patients without flexion deficit. Based on their data, they concluded that placement of the tibial tunnel in the “eccentric,” anteromedial position may contribute to the development of flexion and extension deficits after ACL reconstruction.

The proper tibial tunnel position is located posteromedially in the ACL “footprint,”^{39,49} although some authors have argued that the tibial tunnel should be customized according to the notch roof angle.²⁵ Miller and Olszewski³⁹ discussed proper tunnel positions in their cadaveric study of ideal graft length and position. They found that the average intraarticular graft length for the ACL is 23.6 mm and for the PCL it is 30.7 mm. The average patellar tendon graft intertendinous distance (between patella and tubercle) is 43.3 mm. Therefore, using standard tibial and femoral tunnel positions, the patellar tendon graft is of adequate length to be used for reconstruction of these ligaments.

Poor placement of the femoral tunnel can lead to non-isometric graft placement and loss of motion. Placement of the femoral graft in the over-the-top position leads to increased tension in extension and may contribute to loss of extension.¹⁸ The ideal femoral tunnel is placed in the posterior wall of the femoral notch, leaving 1 to 2 mm of posterior wall remaining.⁴⁹ An excessively anterior femoral tunnel can lead to loss of motion or graft failure, or both (Fig. 5).

While it is well recognized that an insufficient notchplasty may contribute to graft impingement, there are also data to suggest that an overly aggressive notchplasty may contribute to cartilage degeneration.³¹ Graft impingement from regrowth of the notch is another clinically relevant phenomenon that may contribute to late graft failure or extension loss.³ Goss et al.²² have shown in an experimental cadaveric model that quadriceps muscle load can aggravate active impingement of ACL grafts against the intercondylar roof. In their experimental model, use of roofplasty enabled them to mitigate this impingement.

Excessive Graft Tension

Whether excessive tension in the ACL graft leads to loss of motion postoperatively is debatable. We believe that excessive graft tension in a properly positioned graft will not, in and of itself, cause motion loss because of the viscoelastic properties of the ligament when the knee is moved through a full range of motion. It may be possible to overtension a graft when it is positioned in a nonanatomic position, such as the over-the-top position.^{18,38}

Nevertheless, the question often arises as to what is the best position for tensioning the graft? Some biomechanical studies have shown that tensioning the graft in 30° of flexion (the Lachman position) may lead to excessive graft tension. In a cadaveric model in which the graft was fixed in the over-the-top position, Melby et al.³⁸ demonstrated that excessive tension in the graft could lead to restricted



Figure 5. The patient developed a symptomatic loss of extension due to a malpositioned femoral tunnel. The anterior tunnel placement led to graft impingement in terminal extension.

motion. In a prospective study of 57 patients who underwent ACL reconstruction, Nabors et al.⁴¹ concluded that graft tensioning in full extension provided a low incidence of flexion deformity while maintaining excellent functional results and satisfactory biomechanics. The best initial tension and the position at the time of tensioning remains controversial.

In a series of studies, Markolf and coauthors^{34,35} studied the biomechanical consequences of replacement of the ACL with a patellar tendon allograft. Overtensioning of the graft resulted in decreased anteroposterior laxity and increased graft tension at all angles of flexion. Although they were unable to demonstrate any loss of extension with overtensioning of the graft, the normal screw-home mechanism was eliminated. We prefer to tension our ACL reconstructions in approximately 30° of flexion, with the tibiofemoral joint centered. Tensioning the graft when there is anterior subluxation of the tibia leads to increased laxity; conversely, overtensioning with the tibia sublux-

ated posteriorly leads to increased graft tension at all angles of flexion. Both of these scenarios—too loose or too tight—can predispose the graft to failure. Despite these considerations, graft position remains the most important determinant of ultimate stability and motion.

Soft Tissue Calcifications

Another less common cause of loss of motion about the knee is soft tissue calcification that can occur in the joint capsule, the ligaments, or both (Fig. 6). Calcification of the medial collateral ligament usually results in loss of extension and flexion due to decreased excursion of the ligament. Myositis ossificans, the most severe type of calcification, can occur in the quadriceps muscle after contusions or overaggressive manipulation.⁵⁵ The decreased compliance of calcified soft tissues will lead to motion loss and joint stiffness.

Flexion Contracture

Flexion contracture usually results from scarring in the posterior capsule with resultant shortening and contracture of the posterior soft tissues. The cause is usually prolonged immobilization or prolonged loss of extension from another cause. We have also seen iatrogenic flexion contractures after meniscal repair, when sutures placed across the posterior capsular recess effectively shortened the posterior capsule and resulted in loss of extension.

SUMMARY

Motion loss remains a difficult problem after ligament injury and surgery to the knee. Better description, classification, and understanding of the problem can lead to improved prevention and treatment strategies. When motion loss does occur, early recognition and appropriate treatment can be expected to restore motion and improve

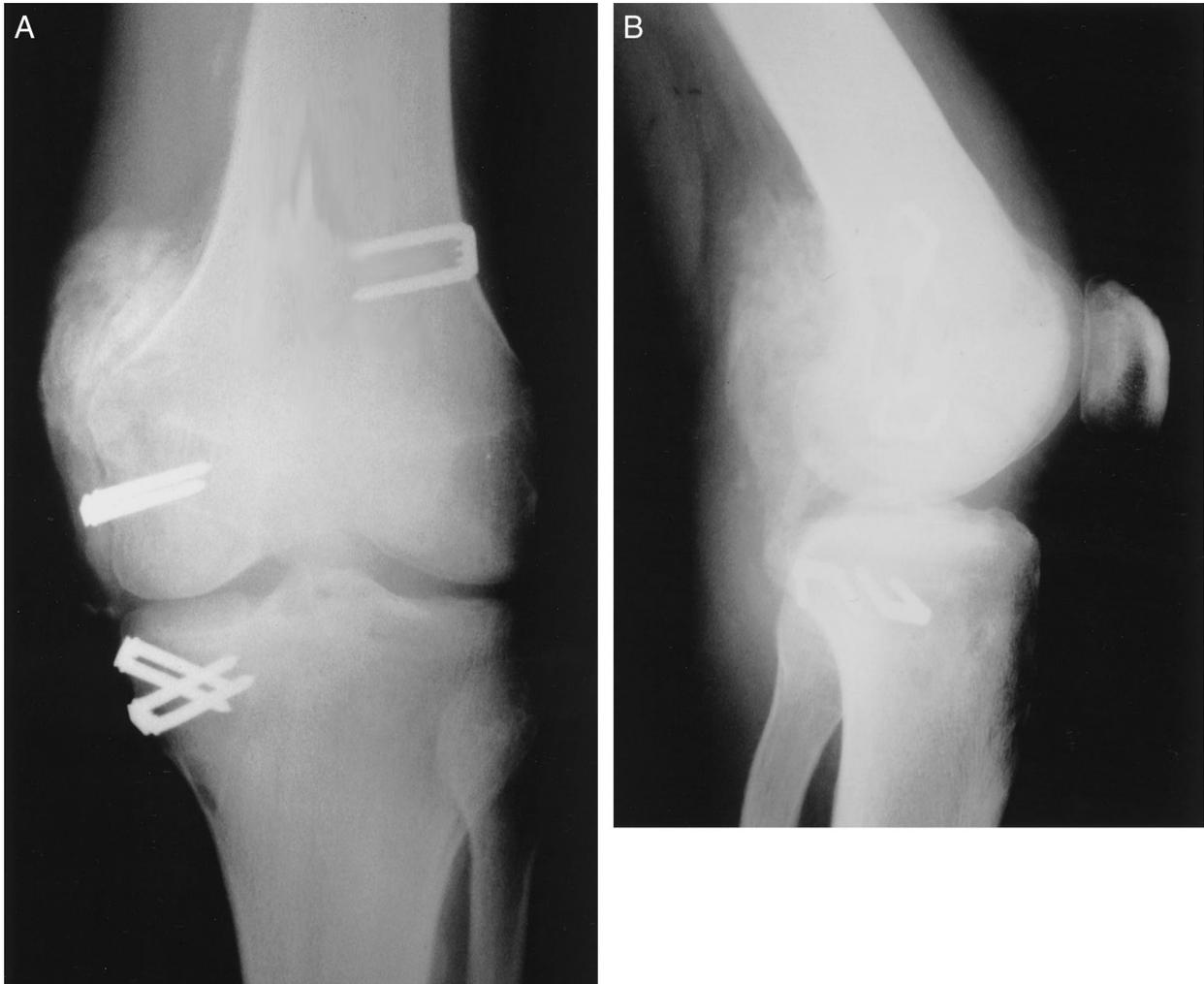


Figure 6. This patient, who had multiple ligament injuries and underwent ACL and MCL reconstruction, developed severe soft tissue calcifications in the capsule and posterior soft tissue structures that prohibited normal knee motion and necessitated open debridement and release. A, AP radiographic view; B, lateral radiographic view.

function. This article has reviewed current concepts on the definitions, incidence, and causes of motion loss. The article summarizes the latest information from basic science and clinical studies on the problem of motion loss of the knee and attempts to provide a rational approach to these difficult clinical problems. To optimize outcomes, it is important for the clinician to identify risk factors and to understand pathoanatomy so that motion problems can be recognized and treated appropriately or prevented altogether. The second article in this series, which will be published in the November/December 2001 issue, will review current strategies on prevention and treatment of motion loss after ligament injury or surgery.

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