Arthrofibrosis of the Knee

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Abstract

Better understanding of surgical timing, improved surgical technique, and advanced rehabilitation protocols has led to decreased incidence of motion loss after anterior cruciate ligament injury and reconstruction. However, motion loss from high-energy, multiligament injuries continues to compromise functional outcome. Prevention, consisting of control of inflammation and early motion, remains the key element in avoiding motion loss. However, certain techniques, such as manipulation under anesthesia in conjunction with arthroscopic lysis of adhesions, are reliable treatment options. Open surgical débridement is rarely necessary and should be considered only as a salvage procedure. A greater understanding of the pathogenesis of arthrofibrosis and related inflammatory mediators may result in novel therapies for treating the patient with motion loss.

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None of the following authors or a member of their immediate families has received anything of value from or owns stock in a commercial company or institution related directly or indirectly to the subject of this article: Dr. Magit, Dr. Wolff, Dr. Sutton, and Dr. Medvecky.

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J Am Acad Orthop Surg 2007;15:682-694

Loss of knee motion is a devastating consequence of both single- and multiligamentous injury and their reconstruction. Increased recognition of this problem in the past two decades has led to better prevention and improved management of these injuries. Despite these advances, however, motion loss remains a problematic consequence of knee ligament injury.

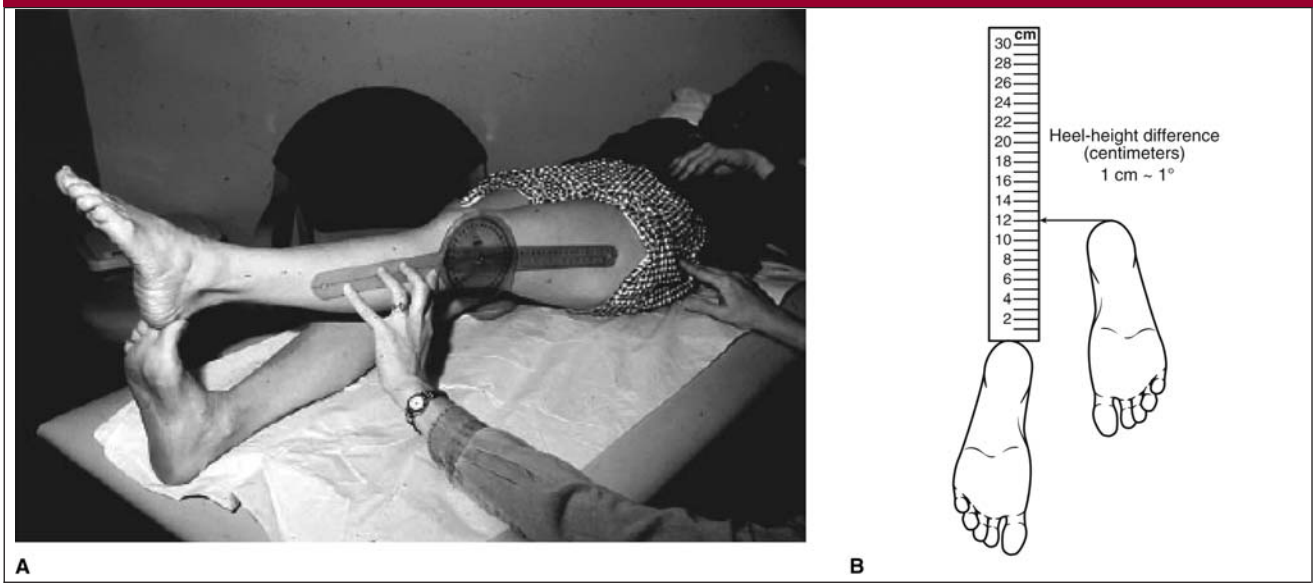
The incidence of motion loss varies according to the degree of injury. Motion loss is less severe after single-ligament, low-energy injury than after high-energy, multiligament injury. The etiology of motion loss is multifactorial, involving a combination of mechanical and biologic factors. Major risk factors include technical errors during intra-articular ligament reconstruction and extra-articular procedures, injury severity, timing of surgery, delayed postoperative physical rehabilitation, heterotopic ossification, prolonged immobilization, infection, and complex regional pain syndrome. Recently, authors have begun to examine possible genetic differences among patients with arthrofibrosis.[1]

Arthrofibrosis represents a wide spectrum of disease, ranging from localized to diffuse involvement of all compartments of the knee and of the extra-articular soft tissues. Prevention of motion loss remains essential to successful outcome. In the patient who experiences motion loss despite preventive measures, treatment options include static or dynamic bracing, manipulation under anesthesia, and arthroscopic or open débridement. In recalcitrant cases, arthrodesis in the older patient or total knee arthroplasty may be required.

Normal Knee Motion

Normal knee motion involves a combination of longitudinal axial, rotation, varus/valgus angulation, and flexion/extension arcs. Flexion and extension can be categorized into three sub-arcs: terminal extension, active function, and passive flexion. The arc of terminal extension, also called "screw home," begins at the limit of passive extension. This arc moves from 10° of flexion to 5° of hy-

Figure 1



Knee motion can be measured by the standard goniometric method **(A)** or by measuring the heel-height difference **(B)**. (Panel A reproduced and panel B adapted from Schlegel TF, Boublik M, Hawkins RJ, Steadman JR: Reliability of heel-height measurement for documenting knee extension deficits. *Am J Sports Med* 2002;30:479-482.)

perextension. The arc of terminal extension is rarely used in normal gait but is thought to allow for quadriceps muscle relaxation during the stance phase. The arc of active function ranges from 10° to approximately 120°, which covers the range needed for most activities of daily living, including sitting and stair climbing. The arc of passive flexion begins at approximately 120° and continues to the passive limit of an applied external force. Typically, passive flexion is 140° in men and 143° in women. However, flexion to 165° is seen in societies in which full kneeling or squatting is common, such as in Japan, India, and the Middle East.[2]

Flexion and Extension Deficits

Flexion Loss

The functional effects of knee motion loss vary depending on patient activity. In general, flexion to 125° is adequate for completing activities of daily living and usually does not adversely affect normal gait. Without flexion beyond 125°, the patient may report an inability to squat. Small flexion deficits in the athlete, however, can produce marked changes in performance. Loss of flexion ≥10° can affect running speed. Severe flexion deficits <90° affect the ability of even the most sedentary patient to sit or climb stairs.

Extension Loss

Extension loss is poorly tolerated and can be more difficult to manage than loss of flexion. As little as 5° of extension loss can produce a noticeable limp during ambulation, strain the quadriceps muscle, and contribute to patellofemoral pain. During weight bearing on a flexed knee, the quadriceps muscle force required to stabilize the knee is 75% of the load on the femoral head at 15° of flexion, 210% at 30°, and 410% at 60°.[3] With increased joint contact pressure, the clinical consequences are increased quadriceps muscle activity and fatigue, and, ultimately, patellofemoral arthrosis.

Measuring Motion Loss

Proper care for the patient with recent ligamentous knee injury or reconstruction requires accurate detection of motion loss. The most common method involves placing a goniometer over the lateral knee joint line in the midsagittal position, using the greater trochanter and lateral malleolus as reference points (Figure 1, A). Several studies have demonstrated high inter- and intraobserver reliability with this method.[4] A second method involves measuring the heel-height difference, which is done with the patient in the prone position (Figure 1, B). In general, 1 cm of heel-height difference correlates to 1° of knee flexion contracture. This technique may be helpful in detecting subtle degrees of motion loss (<10°).[5]

Classification of Motion Loss

Determining the true incidence of motion loss can be difficult, given

Table 1

Sprague Pathoanatomic Classification of Motion Loss

Group	Pathoanatomy
1	Discreet bands or a single sheet of adhesions traversing the suprapatellar pouch
2	Complete obliteration of the suprapatellar pouch and peripatellar gutters with masses of adhesions
3	Multiple bands of adhesions or complete obliteration of the suprapatellar pouch with extracapsular involvement with bands of tissue from proximal patella to anterior femur

Reproduced with permission from Sprague NF III, O'Connor RL, Fox JM: Arthroscopic treatment of postoperative knee fibroarthrosis. *Clin Orthop Relat Res* 1982;166:165-172.

Table 2

Classification of Motion Loss of the Knee Based on Deviation From Full Flexion and Extension

Group	Extension	Flexion	Severity
1	<5°	>110°	Mild
2	5°-10°	90°-110°	Moderate
3	>10°	<90°	Severe

Reproduced with permission from Del Pizzo W, Fox JM, Friedman ML, et al: Operative arthroscopy for the treatment of arthrofibrosis of the knee. *Contemp Orthop* 1985;10:67-72.

the number of available classification schemes. Sprague et al[6] defined motion loss based on a pathoanatomic distribution (Table 1). Later classification schemes focused on knee range of motion (ROM). Del Pizzo et al[7] graded motion loss by evaluating deviation from full flexion and extension, with severe motion loss considered to be >10° from full extension and <90° of flexion (Table 2). Blauth and Jaeger[8] graded motion loss based on full arc of motion as grade I (mild, ROM >120°), grade II (moderate, ROM 80° to 120°), grade III (severe, ROM 40° to 80°), and grade IV (extreme, ROM < 40°). The most recent classification system was introduced by Shelbourne et al,[9] who compared motion loss on the affected side with the normal contralateral limb. The authors identified four types: 1, normal flexion and extension loss <10°; 2, normal flexion and extension loss >10°; 3, flexion loss >25° and extension >10°; and 4, flexion loss >30° and extension loss >10° with patella infera.

Incidence of Motion Loss

Early studies reported that as many as 35% of patients who underwent acute anterior cruciate ligament (ACL) reconstruction or repair developed loss of knee motion.[10] However, with advances in surgical technique and accelerated rehabilitation protocols, the incidence has markedly decreased, to as low as 0% to 4%.[11-14]

Motion loss is more common with multiligamentous high-energy injury than with single-ligament low-energy injury. Noyes et al[15] reported a 23% incidence of motion loss in a group of patients with concomitant ACL reconstruction and medial collateral ligament (MCL) repair. Traumatic knee dislocation, which produces variable patterns of ligamentous instability, results in the highest incidence of motion loss. Sisto and Warren[16] reported motion problems in 6 of 20 patients (30%). Shapiro and Freedman[17] reported a 57% incidence of postdislocation motion loss. In this study, patients required an additional procedure to restore motion.

Risk Factors for Motion Loss

Technical Errors

Proper graft placement in ACL reconstruction is essential to reducing motion loss.[18] Graft placement anterior to the native ACL insertion on the tibia results in impingement on the roof of the intercondylar notch in extension. Lateral placement on the tibia produces impingement on the lateral wall of the intercondylar notch. Placement too far anteromedially has been shown to limit flexion.[19] On the femoral side, the most common error is graft placement too far anterior, which causes excessive strain on the graft, leading to limited flexion and potential graft failure.[20,21]

Other sources of graft impingement include the intercondylar notch and adjacent posterior cruciate ligament (PCL). Whereas moderate notch impingement can cause pain, effusion, and extension loss, severe impingement can cause abrasion and graft failure.[22] Contraction of the quadriceps during knee extension results in increased impingement; thus, a notchplasty should be performed to provide 3 mm of clearance between the graft and the intercondylar roof. Impingement of the ACL graft on the PCL also may limit flexion when the angle of the tibial tunnel is too steep (80°).[23]

Graft Tension

The relationship between motion loss and graft tension remains controversial. Some have suggested that increased graft tension results in excess constraint on the joint.[24] However, in their biomechanical study, Markolf et al[25] showed that although a high degree of graft pretension may

lead to fraying of the graft over the femoral tunnel, it does not lead to a loss of full knee extension. Conversely, undertensioning the graft may produce anteroposterior laxity with subsequent instability, poor graft healing, and failure.

Graft Choice

The relationship between extension loss and graft choice has been questioned. In a prospective, nonrandomized study comparing hamstring and patellar tendon ACL reconstructions, Pinczewski et al[26] reported that 31% of the patellar tendon group and 19% of the hamstring group had extension deficits at 5 years postoperatively. These results were not statistically significant. Sajovic et al[27] found no significant difference in ROM in their recent prospective, randomized trial comparing hamstring and patellar tendon ACL reconstructions at 5-year follow-up. The authors concluded that graft choice is not related to the development of motion loss after ACL reconstruction.

Extra-articular Procedures

Increased trauma around the knee during open arthrotomy has been cited as a risk factor for motion loss after ACL reconstruction. Harner et al[11] theorized that concomitant MCL repair during ACL reconstruction produced motion loss via disruption of the medial capsule and resultant interference with normal knee kinematics. Alternatively, additional trauma to the capsule may cause an elevated fibrotic response as well as increased pain and swelling, resulting in quadriceps inhibition. With extensive extra-articular soft-tissue dissection or trauma, the development of soft-tissue calcification or myositis ossificans also may result in secondary loss of motion[28] (Figure 2). Conversely, Cosgarea et al[29] found that patients who underwent intra-articular procedures (eg, meniscal repair, partial meniscectomy) after ACL reconstruction were not at increased risk for developing arthrofibrosis.

Soft-tissue Injury

The magnitude of soft-tissue and bony injury associated with ligament injury is directly related to the pathogenesis of motion loss. Typical mechanisms include sports-related trauma and knee dislocation secondary to high-energy motor vehicle accidents. The global nature of multiple traumatic injuries often results in delayed surgical timing, compromised wound healing, alterations in surgical approach, and delayed mobilization. Each factor plays a major role in the pathogenesis of motion loss.

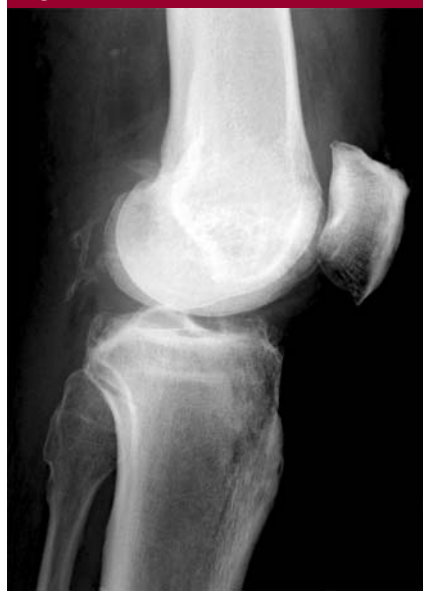
Timing of Surgery

Timing of surgery remains controversial. Many authors differentiate acute from delayed reconstruction at 3 weeks. Delayed surgery allows time for soft-tissue healing, resumption of full ROM, and improved strength.

Shelbourne et al[30] reviewed 169 young athletes after acute ACL reconstruction. Patients in whom ACL ligament reconstruction was done within 1 week of injury had a statistically significant increased incidence of arthrofibrosis ($P < 0.05$) compared with patients in whom ACL reconstruction was delayed >3 weeks.

Harner et al[11] found a 37% rate of postoperative motion loss after acute ACL reconstruction (16 of 43 patients) versus 5% after chronic reconstruction. Wasilewski et al[31] evaluated 87 patients who underwent reconstruction for ACL injury at the acute (<1 month), subacute (1 to 6 months), and chronic (>6 months) stage. Arthrofibrosis was found in 22% of knees managed with ACL reconstruction in the acute stage, in 0% managed in the subacute stage, and in 12.5% managed in the chronic stage. Although Bach et al[24] did not find differences in postoperative motion between acute and chronic ACL reconstructions, they did find a higher overall reoperation rate in the acute cases. They recommend elective subacute reconstruction, provided that motion and effusion goals have been attained.

Figure 2



Lateral radiograph demonstrating posterior heterotopic ossification after multiligamentous reconstruction.

Sterett et al[32] found no association between surgical timing of ligament reconstruction and incidence of motion loss. The authors cited minimal preoperative active ROM (0°-120°), active quadriceps control, and the ability to perform a straight-leg raise as essential determinants of a successful outcome. However, 8% of patients in the acute group required a second surgery for symptomatic scar tissue (6 of 80 patients).

Given the multiple techniques used for reconstruction, variable definitions of timing and classification, and lack of prospective studies, firm conclusions regarding this ongoing debate remain elusive. The key factor remains understanding the mechanism and severity of injury as they relate to the preoperative level of inflammation, strength, and ROM.

Immobilization

Immobilization after knee ligament injury and reconstruction remains a well-established risk factor for motion loss. Hooper and Walton[33] reported that 46% of patients who began motion 2 weeks after surgery experienced loss of motion. In comparison, Zarins and Rowe[34] found nearly complete return of knee extension in their patients, with return of motion after 1 week of cast immobilization following ligament reconstruction. Complications stemming from prolonged casting and the benefits of early motion have led to a shift toward a shorter period of immobilization.

Infection

Infection may cause motion loss after knee ligament surgery. Intra-articular inflammatory mediators responding to infection cause joint synovitis and toxic degeneration to the articular surface. Motion loss is caused directly by fibrous scar formation produced by local cytokine activation, and indirectly by pain produced by joint swelling and irritation.

Complex Regional Pain Syndrome

Complex regional pain syndrome inhibits effective patient participation in proper postoperative rehabilitation. Resultant quadriceps shutdown, muscle atrophy, and avoidance of mobility establish a vicious cycle, leading to the development of intra-articular adhesions and arthrofibrosis. Patella infera is a secondary concern in these patients.

Genetic Risk Factors

Some patients develop motion loss despite efforts to reduce or eliminate the aforementioned risk factors. This has led researchers to consider whether there may be a genetic predisposition to arthrofibrosis after injury. Skutek et al[1] evaluated 17 patients with arthrofibrosis after ACL reconstruction with autologous grafting. Blood samples were taken from each patient, and DNA was evaluated for loci human leukocyte antigen (HLA)–A, –B, –C, and –DQB1. Compared with a control group, patients with arthrofibrosis were less likely to have allelic group HLA-Cw*07 and more likely to have HLA-Cw*08. It is unknown whether these alleles represent potential increased risk for arthrofibrosis by either increased susceptibility or decreased defense mechanisms.

Pathophysiology

Increased understanding of the molecular pathways responsible for orthopaedic disease has provided new insight into the pathogenesis of motion loss. Tissue organization and homeostasis depend on constant signaling of cytokines and locally acting growth factors. These cytokines coordinate cell growth, differentiation, and programmed cell death via constant signaling between local cells (paracrine) as well as among themselves (autocrine).

Transforming growth factor-β (TGF-β), which is released by platelets, plays a critical role in the process of tissue repair.[35] At the site of injury, TGF-β and platelet-derived growth factor initiate a cascade of events resulting in the production of extracellular matrix proteins and protease inhibitors as well as inhibition of proteolytic enzyme production. Formation of extracellular matrix occurs at the site of injury, consisting of an aggregation of collagen, fibronectin, and proteoglycans. With an increase in local concentration, the autoregulatory mechanism of TGF-β results in feedback inhibition. Overexpression of TGF-β can result in progressive deposition of matrix and tissue fibrosis. TGF-β overexpression leads to fibrosis and exists in the other organ systems, such as the kidney, liver, and lung (Figure 3).

Clinical Findings

The term arthrofibrosis describes a wide spectrum of conditions, all of which ultimately result in motion loss. The spectrum of involvement ranges from purely localized to vastly diffuse, with varying degrees of intra- and extra-articular extension.

Localized Intra-articular Extension

Fullerton and Andrews[36] first reported hypertrophy of the ACL graft and protrusion of bone at the tibial attachment site. Jackson and Schaefer[37] further characterized this finding, now known as "cyclops syndrome," as extension loss after ACL reconstruction caused by a mechanical block from hypertrophic fibrous tissue attached to the tibial insertion point of the ACL graft (Figure 4).

Various explanations of the cyclops syndrome have been described in the literature. Anterior graft placement can cause repetitive trauma of graft against bone, leading to impingement on the top of the notch and graft hypertrophy with fibrous tissue proliferation. Fibrous tissue originating from the drilling debris of the tibial tunnel is another possible cause. Fibroproliferative tissue may develop in the patient who does not achieve full extension in the early postoperative period because of pain or immobilization.

Localized Intra-articular With Extra-articular Extension

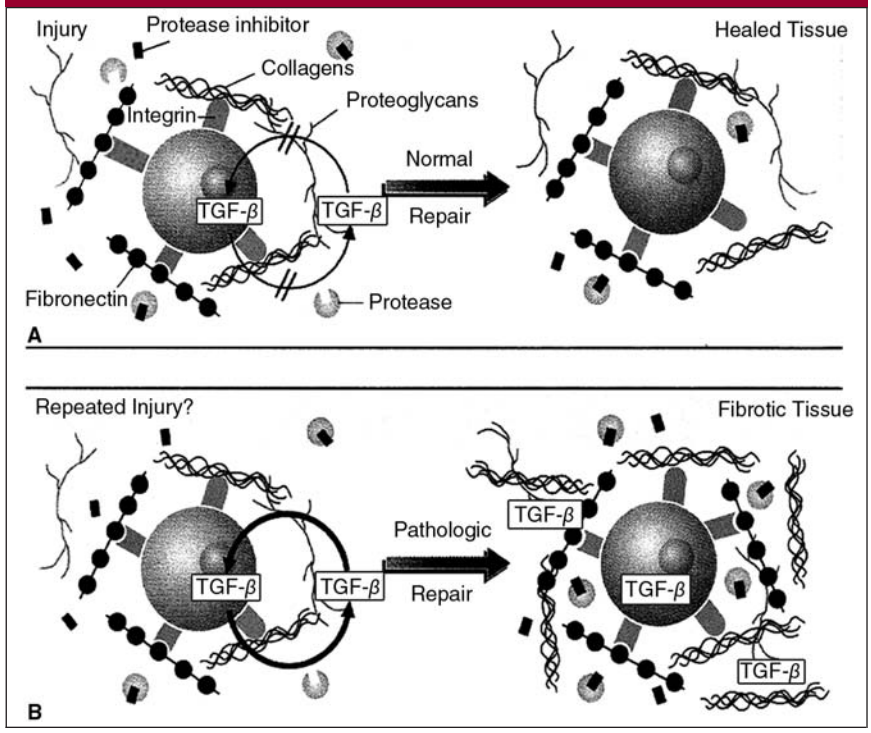
Paulos et al[38] were among the first to describe infrapatellar contraction syndrome (IPCS), which manifests as significant reduction of both flexion (>25°) and extension (>10°) with an associated decrease in patellar mobility, characterized as patellar entrapment. The cause of IPCS was attributed to either exaggerated pathologic fibrous hyperplasia of the anterior soft tissues beyond normal healing or certain risk factors associated with knee surgery (eg, poor

graftisometry, immobilization, muscle weakness).

Paulos et al[38] stratified patients with IPCS into prodromal, active, and residual stages. A patient in the prodromal stage demonstrates periarticular inflammation and edema, quadriceps weakness and lag, poor knee extension, painful ROM, and tenderness over the patellar tendon with a decrease in patellar excursion. A patient in the active stage shows a dramatic decrease in patellar mobility, marked quadriceps atrophy, worsening knee motion, and fat pad induration with a rigid patellar tendon. A patient in the residual stage presents with peripatellar and retinacular tissues that are more supple than in the active stage, with resulting marked quadriceps atrophy and loss of knee flexion and extension. The critical component of the residual stage is the progression of patellofemoral arthrosis and patella infera (Figure 5).

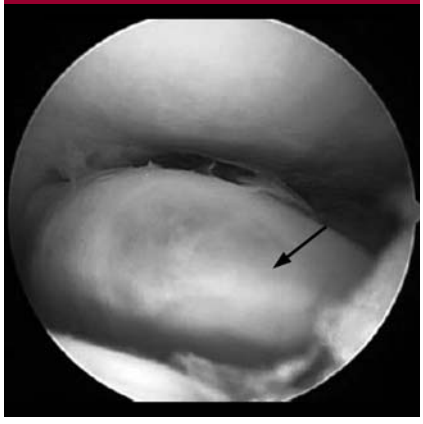
In a series of patients with IPCS, Paulos et al[38] initially used conservative treatment, including inflammation control, followed by gentle

Figure 3



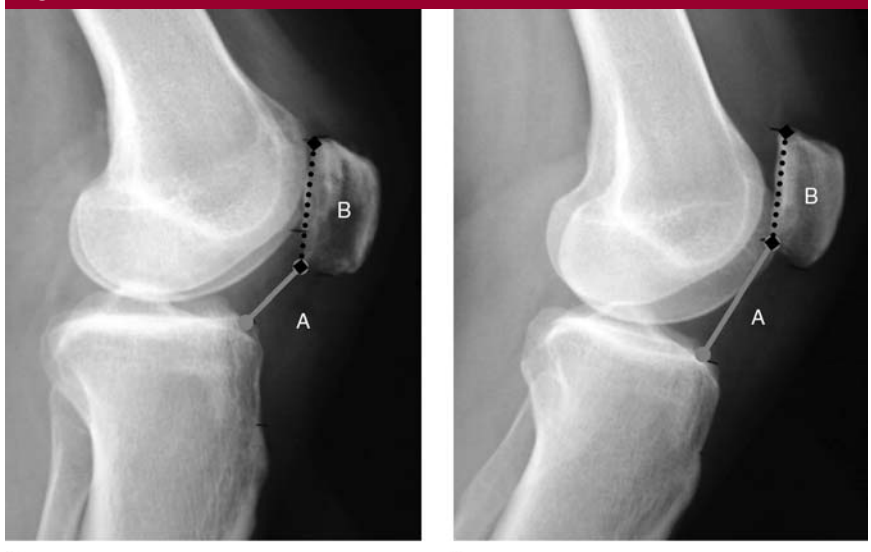
Comparison of the regulatory response of transforming growth factor-β (TGF-β) in a normal **(A)** and a pathologic **(B)** response to tissue repair. (Reproduced with permission from Border WA, Noble NA: Mechanisms of disease: Transforming growth factor [beta] in tissue fibrosis. *N Engl J Med* 1994;331:1286-1292.)

Figure 4



Cyclops lesion (arrow) located anterior to the intercondylar notch. (Reproduced with permission from Ahn JH, Yoo JC, Yang KS, Kim JH, Wang JH: Second-look arthroscopic findings of 208 patients after ACL reconstruction. *Knee Surg Sports Traumatol Arthrosc* 2007;15:242-248.)

Figure 5



Lateral radiographs demonstrating patella infera after patellar tendon repair. The Blackburne-Peel index is shown on presentation (A/B = 0.62) **(A)** and after gait retraining and quadriceps reactivation (A/B = 1.18) **(B)**.

physical therapy, arthroscopic débridement, and manipulation. Patients for whom these treatments were unsuccessful underwent open arthrotomy with débridement; patients with patella infera >8 mm underwent tibial tubercle osteotomy.

Diffuse Intra-articular/ Extra-articular Extension: A Molecular Basis

An exaggerated synovial and inflammatory response may contribute to arthrofibrosis and result in activation and proliferation of fibroblastic cells, which produce elevated levels of type VI collagen and extracellular matrix proteins.[39] With heightened fibroblastic response, tissue shrinkage (ie, scar contraction) occurs both within and outside the joint. A recent study indicates that specific alpha-smooth muscle actin–containing fibroblastic cells play a critical role in tissue contraction associated with wound healing.[40] In arthrofibrotic tissue, the authors found significantly higher numbers of synovial cells ($P \leq 0.05$), significantly less vessel density ($P < 0.001$), and tenfold higher expression of alpha-smooth muscle actin–positive fibroblastic cells ($P \leq 0.001$) compared with control tissue.

Treatment

Immediate Postoperative Motion

Prevention is the most effective means of avoiding the devastating consequences of motion loss after knee ligament injury and surgery. Improved postoperative rehabilitation protocols, including the use of immediate passive and progressive active ROM, have addressed the harmful consequences of prolonged immobilization (Figure 6).

Noyes et al[41] advocated immediate knee motion and early intervention to prevent arthrofibrosis after ACL reconstruction. In a prospective study, 93% of patients regained full ROM (0º to 135º) with the use of active and passive knee motion in the immediate postoperative period (413 of 443 patients). Of the remaining 30 patients, 23 were placed in an early postoperative treatment program, which included hyperflexion and hyperextension exercises and serial extension casting (Figure 7). In eight patients for whom cryotherapy, antiinflammatory medications, elevation, and compression were ineffective, the authors advocated the use of oral steroids. Intensive inpatient physical therapy, manipulation under anesthesia, and arthroscopic débridement were performed in refractory cases. Overall, 98% regained full knee motion, 2% had minor limitations in extension, <1% required arthroscopic release of adhesions, and no patient developed permanent arthrofibrosis.

Postoperative Bracing

Although controversial, the use of a rehabilitation brace after ACL reconstruction is common. In a recent survey of members of the American Orthopaedic Society for Sports Medicine, 85% of respondents prescribed a brace for an average of 3.8 weeks after ACL reconstruction.[42] Whether bracing can prevent loss of extension remains a question. Feller et al[43] found no advantage in restoring knee extension with the use of an extension brace. However, in a recent prospective study comparing bracing from 0° to 90° to bracing locked in full extension during the first week after ACL reconstruction, Melegati et al[44] found that patients braced in full extension for the first week postoperatively had lower heel-height differences at 4- and 8-week follow-up than did patients whose brace was unlocked twice daily for physiotherapy.

In another randomized, prospective study, Mikkelsen et al[45] compared patients braced in full extension (0°) with patients braced in hyperextension (–5°) after ACL reconstruction. At 3 months, 12 of the 22 patients in full extension had extension deficits ≥2° (54%), whereas only 2 of the 22 patients braced in hyperextension had extension deficits (9%). The authors concluded that use of a knee brace in hyperextension for at least 3 weeks after ACL reconstruction is an effective means of preventing postoperative extension loss.

Manipulation Under Anesthesia

Dodds et al[12] reported positive results with knee manipulation in 42 knees with significant flexion or extension deficits after ACL reconstruction with a patellar tendon graft. Outcome after manipulation was related to the severity of extension loss before manipulation; patients with greater extension deficits achieved less overall final extension. The authors recommended manipulation within 12 weeks of ACL reconstruction. More recent reports recommend manipulation within 4 to 12 weeks of reconstruction when <90° of flexion is achieved despite the use of physical therapy.[41]

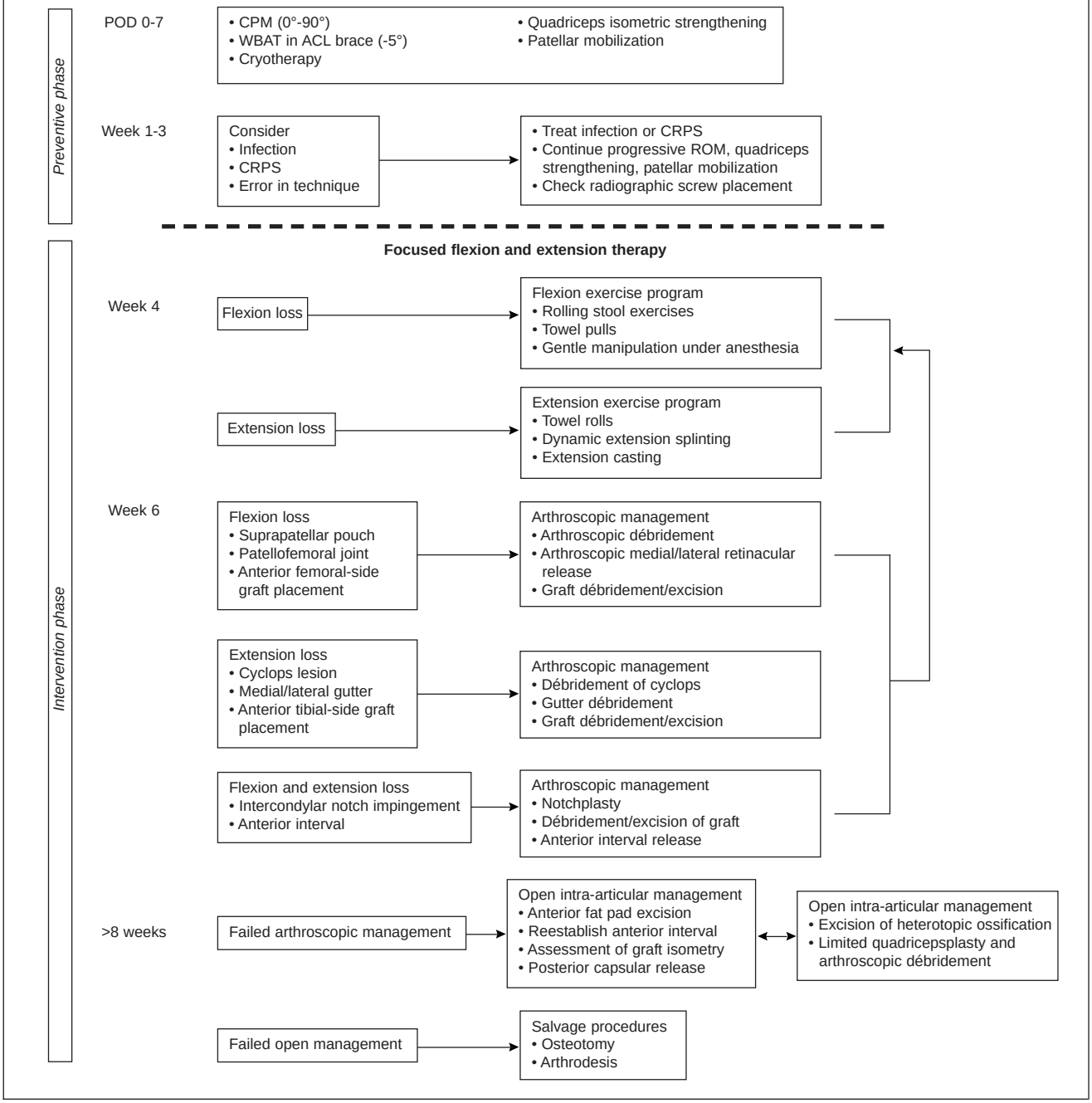
Overaggressive or significantly delayed manipulation should be avoided to prevent complications such as chondral damage, distal femur or patella fracture, patellar tendon rupture, stimulation of myositis ossificans of the quadriceps, and ossification of the MCL.

Manipulation and Arthroscopy

With delayed presentation (>6 weeks), manipulation under anesthesia is most effective when performed in conjunction with arthroscopic lysis of adhesions. Arthroscopic management allows access to focal lesions (eg, ACL nodules, loose bodies) and is helpful in addressing cases of severe diffuse arthrofibrosis refractory to closed methods as well as in avoiding potential catastrophic complications associated with manipulation alone.

Prior to arthroscopy, accurate clinical assessment of motion loss

Figure 6

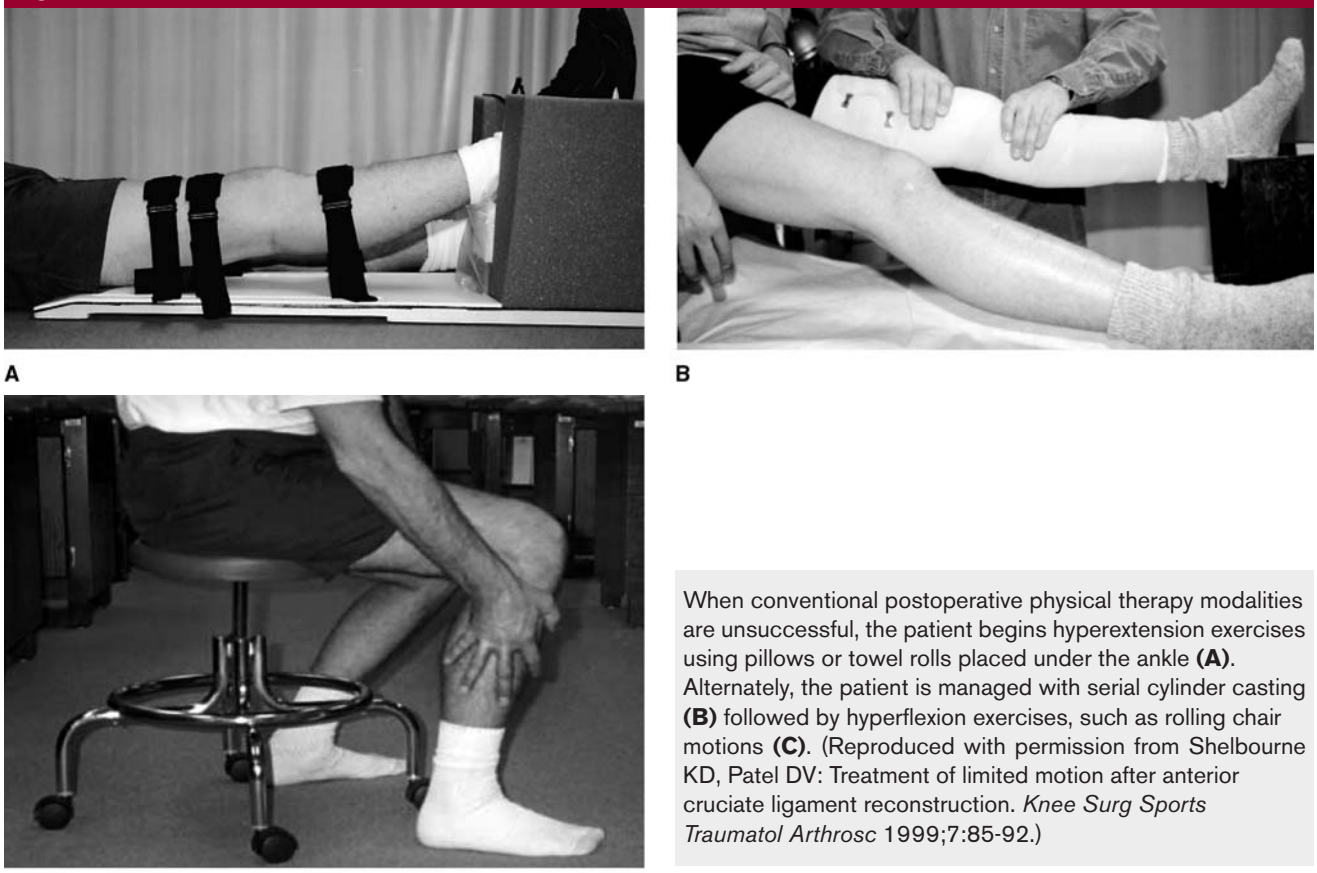


Treatment algorithm for arthrofibrosis highlighting prevention (top) and intervention (bottom) postoperatively. ACL = anterior cruciate ligament, CPM = continuous passive motion, CRPS = complex regional pain syndrome, POD = postoperative day, ROM = range of motion, WBAT = weight bearing as tolerated

aids in identifying the location of arthrofibrosis. Loss of knee flexion often indicates involvement of the suprapatellar pouch, patellofemoral joint, or anterior interval. Involvement of the intercondylar notch can affect both flexion and extension. Extension loss can result from intra-articular nodules adjacent to the tibial graft insertion and arthrofibrosis of the posterior capsule.

Kim et al[46] described a systematic approach when performing arthroscopic débridement of an arthrofibrotic knee. The use of regional

Figure 7



When conventional postoperative physical therapy modalities are unsuccessful, the patient begins hyperextension exercises using pillows or towel rolls placed under the ankle **(A)**. Alternately, the patient is managed with serial cylinder casting **(B)** followed by hyperflexion exercises, such as rolling chair motions **(C)**. (Reproduced with permission from Shelbourne KD, Patel DV: Treatment of limited motion after anterior cruciate ligament reconstruction. *Knee Surg Sports Traumatol Arthrosc* 1999;7:85-92.)

anesthesia can effectively manage perioperative pain and facilitate postoperative rehabilitation.[47] Prior to portal placement, capsular distention is achieved by saline injection into the suprapatellar pouch.

The suprapatellar pouch is reestablished first, followed by the medial and lateral gutters. The anterior interval is identified by releasing the infrapatellar fat pad from the anterior tibia, allowing for reestablishment of the pretibial recess. Medial and/or lateral retinacular release may be required in the patient with reduced patellar mobility or a tight patellofemoral joint.

Once in the intercondylar notch, the surgeon must evaluate for graft impingement caused by notch stenosis. If present, a notchplasty is performed. Scar tissue, ACL or bony nodules, and loose bodies are removed. Evaluation of graft integrity is essential. Depending on the severity of the scarring, the graft may need further débridement, release, or excision. Once complete, the knee should be ranged and motion reassessed.

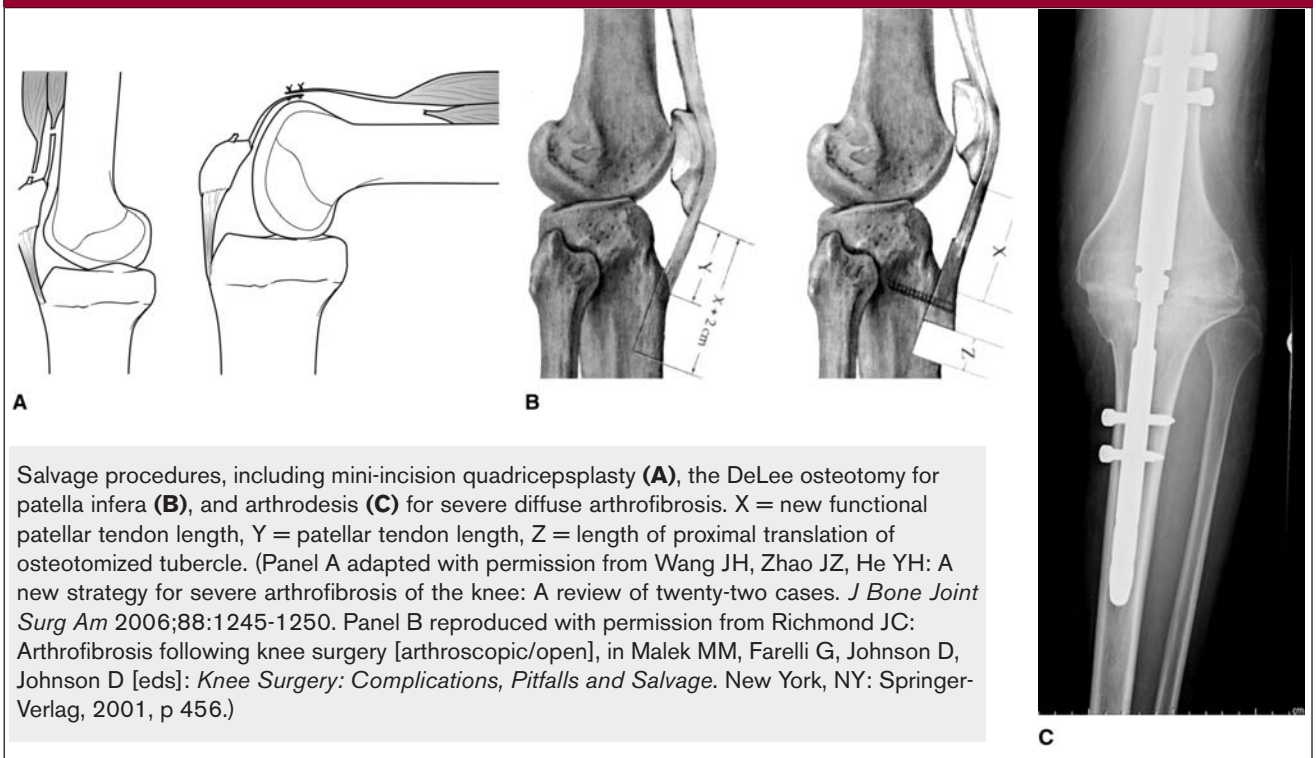
Persistent loss of extension usually indicates posterior capsular involvement. When necessary, a limited open posterior release is performed from a posteromedial and posterolateral incision.[48]

Open Surgical Techniques

Open surgical management of arthrofibrosis represents a salvage option for the rare knee that is refractory to closed and arthroscopic procedures. Of the 207 knees treated for motion loss after knee ligament surgery in a study by Noyes et al,[15] 202 responded to early rehabilitation, manipulation under anesthesia, or arthroscopic lysis of adhesions. Only 2% of patients required open surgical management.

Surgery often demands extensile incisions and large parapatellar arthrotomies. Similar to arthroscopic débridement, a systematic approach should be used. Débridement includes anterior fat pad excision and reestablishment of the pretibial recess, as well as medial and lateral retinacular release. Evaluation of the ACL graft isometry follows, and débridement or excision are often necessary. Finally, release of posterior capsular soft tissues from both the femur and tibia is performed by careful subperiosteal dissection. Failure to recognize and remove any extra-articular causes of motion loss, such as myositis ossificans, heterotopic ossification, or soft-tissue calcifications, can lead to prolonged motion loss after débridement.

Figure 8



Salvage procedures, including mini-incision quadricepsplasty **(A)**, the DeLee osteotomy for patella infera **(B)**, and arthrodesis **(C)** for severe diffuse arthrofibrosis. X = new functional patellar tendon length, Y = patellar tendon length, Z = length of proximal translation of osteotomized tubercle. (Panel A adapted with permission from Wang JH, Zhao JZ, He YH: A new strategy for severe arthrofibrosis of the knee: A review of twenty-two cases. *J Bone Joint Surg Am* 2006;88:1245-1250. Panel B reproduced with permission from Richmond JC: Arthrofibrosis following knee surgery [arthroscopic/open], in Malek MM, Farelli G, Johnson D, Johnson D [eds]: *Knee Surgery: Complications, Pitfalls and Salvage*. New York, NY: Springer-Verlag, 2001, p 456.)

Salvage Procedures

Recently, Wang et al[49] reported their series of mini-incision quadricepsplasty with arthroscopy for patients who presented with severely arthrofibrotic knees with major intra- and extra-articular involvement. The authors used a short, longitudinal incision adjacent to the superolateral corner of the patella to perform a staged release, beginning with the lateral retinaculum and extending to the suprapatellar pouch and across to the medial patellar retinaculum. The last two stages consisted of transection of the vastus intermedius and lengthening of the quadriceps tendon (Figure 8, A). Following release, arthroscopy was performed to complete the intra-articular portion of the procedure. Flexion improved from 5° to 45° preoperatively to 120° to 150° postoperatively.

Proximal sliding tibial tubercle osteotomy (DeLee osteotomy, Figure 8, B) is possible in the patient with patella infera that does not respond to closed, arthroscopic, or open surgical procedures. Arthrodesis is a final salvage procedure in the rare patient for whom no other management option is successful (Figure 8, C).

Postoperative Outcomes

Postoperative outcomes are difficult to interpret, given the different classification systems, variable injury patterns, combined surgical treatment patterns, and different outcome measures. A summary of outcomes after surgical treatment of localized intra-articular lesions, more diffuse intra-articular arthrofibrosis, and arthrofibrosis extending into the extra-articular soft tissues is given in Table 3.

There are few reports on the results of severely arthrofibrotic knees that have undergone multiple surgical treatments. Millett et al[47] reported on eight patients with an average preoperative ROM of 62.5° who underwent open débridement. After radical open débridement (mean follow-up, 57 months), patients gained an average of 62° of motion. Patient satisfaction was high, and Lysholm II scores improved an average of 35 points. However, in five of eight patients, degenerative changes were seen on radiographs at a mean follow-up of 57 months. All patients were able to return to sports, but only one was able to achieve her preinjury level of function.

Summary

The incidence of motion loss after ACL injury and reconstruction has decreased because of greater understanding of surgical timing, improved surgical technique, and advanced rehabilitation protocols. In high-energy injuries and multiligament injuries, however, the incidence of motion loss continues to cause problems for the treating surgeon. Prevention through early motion remains the key element in avoiding motion loss. However, techniques such as manipulation under anesthesia used in conjunction with arthroscopic lysis of adhesions are reliable treatment options. Open surgical débridement is rarely

Table 3

Outcomes After Localized Arthrofibrosis, Diffuse Arthrofibrosis, and Arthrofibrosis With Intra- and Extra-articular Extension

Study	No. of Patients	Extent of Arthrofibrosis	Index Surgery/Injury (No. of Patients)	Average Preoperative ROM	Surgery	Average Postoperative ROM
Jackson and Schaefer[37]	13/230	Cyclops lesion	ACLR with BPTB (13), ITB tenodesis (4), MCL repair (1)	16° to 103°	Arthroscopic débridement of nodule, MUA	4° to 138°
Fisher and Shelbourne[14]	35/959	Cyclops lesion	ACLR with BPTB (42)	6° to 119°	Arthroscopic débridement of nodule	2° to 135°
Klein et al[50]	46	Diffuse intra-articular	ACLR (34), meniscal lesion (4), ACL/PCL (1), patellofemoral (6), HTO (1)	10.4° extension loss, 31.6° flexion loss	Arthroscopic débridement	1.7° extension loss, 7.9° flexion loss
Shelbourne and Johnson[51]	9	Diffuse intra-articular	ACLR with BPTB (8) or HS (1)	23° to 113°	Arthroscopic débridement, MUA	2° to 130°
Hasan et al[52]	17/342	Diffuse intra-articular	ACLR (17)	10° to 123°	Arthroscopic débridement, MUA	3° to 131°
Harner et al[11]	27/244	Diffuse intra-/extra-articular	ACLR (27), extra-articular procedure (11), ITB tenodesis (8), MCL repair or POL reefing (12)	13° to 124°	Arthroscopic débridement (14), open débridement (6), MUA alone (1)	3° to 126°
Cosgarea et al[53]	37	Diffuse intra-/extra-articular	ACLR (23), ACL repair (12), PCL repair (1), MCL repair (1)	14° to 120°	Arthroscopic débridement, percutaneous LOA, lateral release, notchplasty (30), open débridement (7)	3° to 142°
Millett et al[47]	8	Diffuse intra-/extra-articular	Meniscectomy (1), ACLR (4), ORIF with PCL/MCL/LCLR (1), PCL/MCLR (1), ACL/PCLR (1)	19° to 81°	Arthroscopic débridement (6), MUA alone (2), open débridement and soft-tissue release	1° to 125°

ACLR = anterior cruciate ligament reconstruction, BPTB = bone-patellar tendon-bone, HS = hamstring, HTO = high tibial osteotomy, ITB = iliotibial band, LCLR = lateral collateral ligament reconstruction, LOA = lysis of adhesions, MCL = medial collateral ligament, MCLR = medial collateral ligament reconstruction, MUA = manipulation under anesthesia, ORIF = open reduction and internal fixation, PCL = posterior collateral ligament, PCLR = posterior cruciate ligament reconstruction, POL = posterior oblique ligament, ROM = range of motion

necessary and should be considered only as a salvage procedure. Primary motion loss occurs in the patient in whom arthrofibrosis develops despite consideration of all known risk factors. A greater understanding of the pathogenesis of arthrofibrosis and of inflammatory cytokines, such as TGF-β, may lead to novel therapies for managing this difficult problem.

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Citation numbers printed in **bold type** indicate references published within the past 5 years.

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