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Treatment of limited motion after anterior cruciate ligament reconstruction

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Abstract Limited motion or arthrofibrosis after anterior cruciate ligament (ACL) reconstruction causes significant pain and functional impairment. Based on physical findings and loss of motion compared with the opposite normal knee, classification systems for the diagnosis and treatment of arthrofibrosis have been developed. The operative techniques and preoperative and postoperative rehabilitation and management are discussed. Range of motion (ROM) problems after ACL reconstruction

have been minimized by improved surgical techniques and perioperative rehabilitation programs. The most effective treatment for arthrofibrosis is prevention by delaying ACL reconstruction until the patient has a normal gait and full ROM and minimal swelling in the injured knee and by appropriate ROM exercises after surgery.

Key words Anterior cruciate ligament · Arthrofibrosis

Introduction

Limitation of knee motion after anterior cruciate ligament (ACL) reconstruction can cause significant pain and functional impairment. A patient is said to have *arthrofibrosis* when the painful limitation of motion becomes permanent despite efforts such as exercise or treatment modalities. Arthrofibrosis after ACL reconstruction can limit the patient's return to a normal lifestyle. Loss of extension is usually more symptomatic than loss of flexion and is reported to be associated with patellofemoral pain, quadriceps weakness, and overall poor knee function [19]. Limitation of flexion causes difficulty with going down stairs, squatting, and kneeling.

There are several definitions for arthrofibrosis in the literature. Paulos et al. [14] coined the term "infrapatellar contracture syndrome" (IPCS) for patients who evidenced a loss of both flexion and extension, where there was an exaggerated fibrosclerotic healing response associated with patellar entrapment. Harner et al. [7] and Cosgarea et al. [2] defined arthrofibrosis as a loss of knee extension of 10° or more from neutral and knee flexion < 125°. Shelbourne et al. [30] has defined arthrofibrosis as any symp-

tomatic loss of knee extension or flexion compared with the opposite normal knee.

The factors causing limited motion after ACL reconstruction have been discussed in the literature and are summarized in Table 1. A thorough understanding of the possible causes for limited motion is essential for developing a strategy for prevention.

The purpose of this review is to discuss the evaluation and treatment of limited knee motion after ACL reconstruction. Guidelines for preventing or minimizing the po-

Table 1 Factors that can cause limitation of knee motion after anterior cruciate ligament (ACL) reconstruction

| |
|---|
| Arthrofibrosis, infrapatellar contracture syndrome and patella infera [11, 14] |
| Inappropriate graft placement/tensioning [6, 18, 21] |
| "Cyclops" syndrome [8] |
| Acute surgery on a swollen knee [7, 29, 34] |
| Concomitant medial collateral ligament repair [24, 27] |
| Poorly supervised or poorly designed rehabilitation program, prolonged immobilization [6, 12, 20, 29, 34] |
| Reflex sympathetic dystrophy [15, 35] |

tential range of motion (ROM) problems after ACL reconstruction are provided.

Classification systems

For the diagnosis and treatment of arthrofibrosis, two classification systems exist. Paulos et al. [14] produced a classification for severe limitation of knee motion and Shelbourne et al. [30] one for all types of limitations of motion with ACL reconstructive surgery.

For a severer limitation of knee motion, Paulos et al. [14] described three stages of IPCS: the prodromal stage (stage I), the active stage (stage II) and the “burned-out” or residual stage (stage III). The prodromal stage (stage I) occurs 2–8 weeks after surgery. Physical examination typically demonstrates indurated synovial, fat pad, and reticular tissues. These patients usually present with a swelling and tenderness around the patellar tendon, painful ROM, restricted patellar mobility, and quadriceps muscle lag. Failure to progress with rehabilitation is an important clue.

The active stage (stage II) occurs 6–20 weeks after surgery. In this stage, patients have continued peripatellar swelling and induration with severely restricted patellar motion (especially superior glide). Most patients show a “shelf” sign caused by swelling and induration of the anterior tissues extending to the distal attachment of the patellar tendon, thereby resulting in a “step off” between the patellar tendon and the tibial tuberosity.

The “burned-out” or residual stage (stage III) occurs 8 months to years after surgery and is characterized by fat pad atrophy, patellofemoral crepitus, and in most cases patellofemoral arthrosis. Developmental patella infera and quadriceps atrophy are present. Inflamed and indurated peripatellar tissues are less prominent, and patellar mobility is less severely restricted than in stage II.

For all types of limitation of knee motion, Shelbourne et al. [30] described four types of arthrofibrosis associated with ACL reconstruction (Table 2). This classification system is based on motion of the injured knee compared with motion in the non-injured knee including full hyperextension (Fig. 1) [22]. The passive ROM of the knee is recorded as $a^\circ/b^\circ/c^\circ$ with “a” representing the degree of hyperextension, “b” the degree of extension that is short

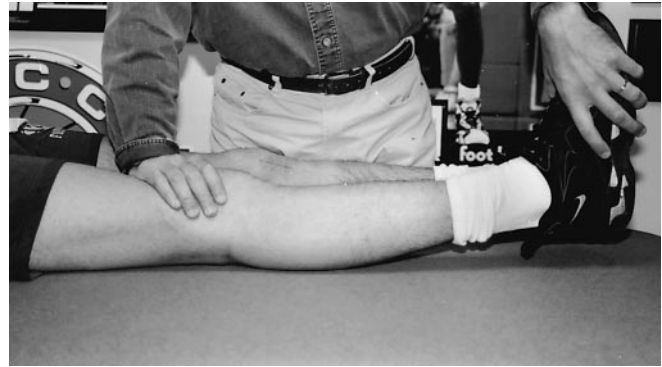


Fig. 1 Full hyperextension of the knee is evaluated by placing one hand above the patella to stabilize the femur and by lifting the foot up passively with the other hand

of 0° , and “c” the degree of flexion present. Motion of $5^\circ/0^\circ/140^\circ$ means that the patient hyperextends 5° of extension past 0° and flexes to 140° . Motion of $0^\circ/5^\circ/140^\circ$ means that the patient lacks 5° of extension from 0° and flexes to 140° .

Patients with type 1 arthrofibrosis have an extension loss of $\leq 10^\circ$ (compared with the opposite knee) that is usually associated with anterior knee pain. In most cases, the patient’s knee can be straightened with pressure, but springs back to a flexed position once the manual pressure is released, and the knee has little, if any, posterior capsular tightness.

Patients with type 2 arthrofibrosis have $> 10^\circ$ extension loss and normal flexion. With applied pressure, the patients’ knee cannot be straightened completely. The extension loss is due to a mechanical block caused by anterior scar formation, a mismatch between a hypertrophic ACL graft including “cyclops” formation [8] and a normal or decreased notch size, and secondary posterior capsular tightness.

Patients with type 3 arthrofibrosis have $>10^\circ$ extension loss and $> 25^\circ$ flexion loss. In addition, the patients’ knees have decreased patellar mobility, tight medial and lateral capsular structures, but no patella infera measured radiographically.

Patients with type 4 arthrofibrosis have similar ROM loss as described for patients with type 3 arthrofibrosis. However, these patients have patella infera measured radiographically compared with the noninvolved knee.

Table 2 Classification of arthrofibrosis [30]

| | Description |
|--------|---|
| Type 1 | $\leq 10^\circ$ extension loss and normal flexion |
| Type 2 | $>10^\circ$ extension loss and normal flexion |
| Type 3 | $>10^\circ$ extension loss and $> 25^\circ$ flexion loss with decreased medial and lateral movement of the patella (patellar tightness) |
| Type 4 | $>10^\circ$ extension loss and $\geq 30^\circ$ of flexion loss and patella infera with marked patellar tightness |

Treatment of arthrofibrosis

Most physicians prescribe physical therapy exercises, patellar mobilization, extension casting, continuous passive motion, and anti-inflammatory drugs to restore as much motion in the knee joint as possible before resorting to surgical intervention. The decision regarding the timing of surgical intervention is variable. Surgery during the in-

flammatory stage is probably contraindicated [14]. Paulos et al. [14] describe the importance of returning the knee to a noninflamed state and strengthening the quadriceps muscles before proceeding with surgery for arthrofibrosis. Progress with therapy should be documented as stalled, and the knee should be allowed to return to a noninflamed state by decreasing the aggressive nature of the rehabilitation efforts.

Various methods of surgical treatment of arthrofibrosis are described in the literature, including open debridement [14], arthroscopic debridement with or without posteromedial and/or posterolateral release [33], combined open and arthroscopic debridement [1], and arthroscopically assisted scar resection [3, 4, 13, 16, 23, 32]. The major open procedures involve extensive hospital resources requiring inpatient hospitalization, prolonged rehabilitation, and use of continuous passive motion machines and continuous epidural catheters for pain control.

Open surgical technique

Paulos et al. [14] reported an open technique for the treatment of patients with stage II or stage III IPCS. In the prodromal stage (stage I, 2–8 weeks after initial surgery), nonoperative treatment was emphasized, consisting of ROM exercises, muscle stimulation, patellar mobilization, and anti-inflammatory medication. In the active stage (stage II, 6–20 weeks after initial surgery), treatment included open intraarticular and extraarticular release of the lateral retinacular, hypertrophied fat pad, and lateral and medial patellomeniscal ligaments followed by continuous passive motion, daily physical therapy, drop-out extension casting, epidural analgesia, and anti-inflammatory medication. In the “burned-out” or residual stage (stage III, 8 months to years after initial surgery), they suggested that surgery should be limited to salvage procedures such as patellectomy and total knee replacement to control pain. They concluded that prevention or early detection and aggressive treatment are important to avoid complications after ACL surgery.

Combined arthroscopic and open surgical technique

Steadman et al. [33] and Cosgarea et al. [1] utilized a combination of arthroscopic and open techniques for restoring ROM. Arthroscopy was performed to remove adhesions in the suprapatellar pouch, medial and lateral gutters, and anteriorly in the intercondylar notch. Arthrotomies were utilized to release anterior extraarticular scar and for posteromedial and posterolateral capsular releases.

Cosgarea et al. [1] reported on 37 patients with arthrofibrosis of the knee after knee ligament surgery. Indications for a lysis of adhesions procedure included flexion

or extension deficits of $\geq 10^\circ$ or when motion failed to improve despite 2 months of intensive physical therapy. After debridement, flexion improved from 83% to 97% of the opposite normal knee. Extension deficits improved from 14° to 3° . Satisfactory functional results were obtained in 23 of the 37 patients (62%). Radiological evidence of degenerative changes in the knee was noted in 89% of the patients. In the study by Steadman et al. [33], following a combination of open and arthroscopic debridement of adhesions, 33 of 44 patients were rated as excellent, 8 good, 1 fair, and 2 poor.

Arthroscopic surgical technique

Some authors recommend arthroscopic surgery in combination with knee manipulation as the treatment for arthrofibrosis of the knee [3, 4, 11, 13, 16, 23, 30–32]. Shelbourne et al. [30] reported on an outpatient arthroscopic technique and rehabilitation program based on the type of arthrofibrosis present (Table 2). Patients with type 1 arthrofibrosis were treated by excision of the hypertrophied mushroom-shaped “cyclops” lesion at the base of ACL graft until the graft fit in the notch without any impingement with the knee in full extension. Patients with type 2 arthrofibrosis required resection of the anterior scar and also resection of the extrasynovial build-up of scar tissue anterior to the proximal tibia. Notchplasty or graft debridement was performed after scar resection if graft impingement was still present on full knee extension.

Patients with type 3 arthrofibrosis had scarring similar to that found in patients with type 2 arthrofibrosis. These patients also had extrasynovial scar formation between the patellar tendon and fat pad, including the fat pad. A blunt probe was used to establish a plane between the patellar tendon and the scar tissue to be resected. The arthroscopic scar resection included removal of scar tissue distally to the upper tibia and anteriorly to the horns of both menisci. Once the retropatellar tendon scar tissue and anterior tibial scar tissue were resected, the fibrotic capsule was excised up to the vastus medialis obliquus and the vastus lateralis insertion to free the patella and the patellar tendon completely. The placement of the ACL graft was considered also. If the graft was found too far anteriorly, the anterior fibers of the graft were resected, and a notchplasty performed laterally and anteriorly to increase the size of the intercondylar notch.

Patients with type 4 arthrofibrosis required a scar resection as described for patients with type 3 arthrofibrosis. A more extensive scar resection both medial and lateral to the patella was required for these patients. In patients with type 3 or type 4 arthrofibrosis, a knee manipulation was performed after completion of the scar resection, to achieve as much knee flexion as possible. A notchplasty was required for all patients with types 2, 3, and 4 arthrofibrosis.

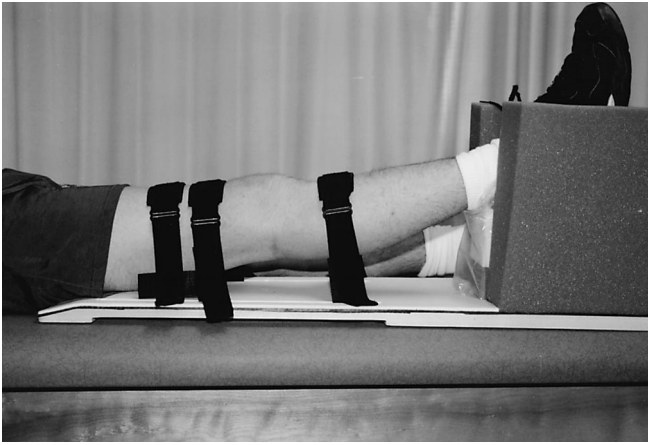


Fig. 2 Extension board is useful for patients who have difficulty regaining full extension at the knee. The heel is elevated while straps above and below the knee joint serve to facilitate knee extension

At the end of the surgical procedure, a well-padded cylinder cast with the knee in full extension was applied for all patients with types 2, 3, and 4 arthrofibrosis. Type 1 arthrofibrosis patients usually regained full knee extension without the need for a cast. All patients were discharged home or to a nearby hotel.

The rehabilitation program after arthroscopic scar resection as described by Shelbourne et al. has been reported in detail [4, 23, 30]. The program emphasized achieving full extension before aggressively improving flexion.

Patients with type 1 arthrofibrosis were seen 2–3 days after scar resection for clinical evaluation, and physical therapy exercises were prescribed to maintain full knee extension. The patients were seen at 1-week intervals, and they were discharged when they were able to maintain full hyperextension easily (usually by 1–2 weeks).

Postoperatively, types 2, 3, and 4 arthrofibrosis patients were seen the following morning, and the extension cast was removed. An extension board (Fig. 2) and prone hangs (Fig. 3) were used to obtain more extension, and a new forced-extension cast was applied. The cast was applied with the patient sitting on a low chair (Fig. 4) to allow maximum relaxation of the posterior knee structures. This treatment was continued daily until full extension (similar to that in the opposite normal knee) or near full extension was obtained. Weight-bearing was allowed as tolerated beginning on the day of surgery. This type of treatment was successful in 1–3 days for patients with type 2 arthrofibrosis and usually in 3–4 days for patients with types 3 and 4 arthrofibrosis.

Once full extension was successfully achieved, it was maintained through a combination of physical therapy exercises, gait training, frequent use of extension board, and a bivalved cast at night. After full extension was maintained easily, aggressive physical therapy exercises to increase knee flexion were commenced.



Fig. 3 Postoperatively, prone hang exercises with weights are used to maintain knee extension



Fig. 4 A cylinder cast is applied to the knee with the patient sitting on a low stool to provide for relaxation of the posterior structures of the knee

Shelbourne et al. [30] report the results of treating 72 patients with arthrofibrosis after ACL reconstruction (performed either at their center or at other centers). The patients were examined at an average follow-up of 35 months (range 28–115 months) after arthroscopic scar resection. The mean interval between the initial ACL surgery and arthroscopic scar resection was 12.5 months (range 2.4–60 months). At the time of latest postoperative follow-up, patients with type 1 arthrofibrosis ($n = 25$) improved from a mean of $0^{\circ}/3^{\circ}/140^{\circ}$ preoperatively to a mean of $4^{\circ}/0^{\circ}/140^{\circ}$ postoperatively; patients with type 2 arthrofibrosis ($n = 16$) improved from $0^{\circ}/11^{\circ}/135^{\circ}$ to $3^{\circ}/0^{\circ}/137^{\circ}$; patients with type 3 arthrofibrosis ($n = 15$) improved from $0^{\circ}/10^{\circ}/111^{\circ}$ to $3^{\circ}/0^{\circ}/139^{\circ}$; and patients with type 4 arthrofibrosis ($n = 16$) from $0^{\circ}/15^{\circ}/103^{\circ}$ to $3^{\circ}/0^{\circ}/130^{\circ}$. There was a considerable improvement in the

mean modified Noyes knee score, stiffness score, self-evaluation score, and functional activity score for patients in all groups [30]. However, patients with type 4 arthrofibrosis had less predictable results, with one patient failing to improve (increasing only 5° of extension and 10° of flexion). The average patellar tendon shortening improved after the scar resection procedure from 15% to 8% compared with the noninvolved knee.

Prevention of arthrofibrosis

The concept of prevention of complications after ACL reconstruction with autogenous, central- 10-mm patellar tendon graft has previously been discussed [12, 17, 26]. Prevention of arthrofibrosis is based on a complete understanding of its causes. The following factors should be considered: graft placement, associated ligamentous injuries, patient selection, timing of surgery and preoperative rehabilitation, postoperative rehabilitation program.

Graft placement

Accurate anatomic placement of the graft is important to prevent ROM problems. If the ACL graft is positioned too anteriorly on the tibial side, impingement of the graft against the roof of the intercondylar notch occurs on extension. If the graft is placed too anteriorly on the femoral side, limitation of knee flexion may occur. We prefer a slightly posterior placement of the tibial tunnel to avoid impingement of the graft against the anterior margin of the notch with the knee in full extension. The femoral tunnel is drilled on the posteromedial aspect of the lateral femoral condyle in such a way that at least 1–2 mm of posterior cortex is left intact.

In addition to accurate graft placement, adequate tensioning and secure graft fixation are also critical. At the completion of the operation, the knee should be taken through a full ROM to ensure full hyperextension and full flexion.

Associated ligamentous injuries

The associated ligamentous injuries (if any) must be considered before planning treatment. The rationale of management of patients with combined ACL/medial collateral ligament (MCL) injuries has previously been reported [24, 27]. In patients with combined ACL/MCL injuries, the torn MCL can be treated nonoperatively, regardless of its severity, and adequate healing of the MCL with good stability can be obtained. The MCL that has avulsed from the femoral condylar origin or a mid-substance MCL tear proximal to the joint line tends to heal with stiffness, and therefore, it is important to restore a full ROM before con-

sidering ACL reconstruction. A delayed reconstruction of the ACL results in a substantially decreased incidence of postoperative complications, especially knee stiffness.

In patients with combined ACL/lateral side injuries, direct anatomic repair of the torn lateral structures and ACL reconstruction should be performed when the acute inflammatory phase of injury has subsided and a good ROM has been obtained (usually by 7–10 days).

Patient selection

Patient selection for ACL surgery is the initial key to avoiding postoperative problems. Not all patients with an ACL tear need to undergo surgery to obtain a functional knee. Factors such as the patient's age, functional and sporting demands on the knee, associated ligamentous and meniscal injury, and the patient's ability to participate in the perioperative rehabilitation program [9, 10, 25–29] must be considered before undertaking ACL reconstruction.

Preoperative counseling of the patient and the family is important. Various treatment options should be reviewed before a decision for surgery is made. Such a discussion minimizes the patient's apprehension towards surgery and allows for early postoperative rehabilitation goals to be obtained easily.

Timing of surgery and preoperative rehabilitation

The importance of timing of surgery has previously been emphasized [9, 10, 25]. It is the physical condition of the knee rather than the number of weeks after injury that should be considered before undertaking ACL reconstruction. Recently, Cosgarea et al. [2] emphasized the importance of the prevention of arthrofibrosis after ACL reconstruction. They reported a retrospective analysis of patients who have undergone ACL reconstruction using the central third patellar tendon graft. Twenty-two of 188 patients (12%) were found to have arthrofibrosis. The incidence of arthrofibrosis was lower when the ACL reconstruction was delayed at least 3 weeks from the initial injury, and when preoperative extension was 10° or better.

Because obtaining full ROM after ACL reconstruction is critical to minimize postoperative subjective knee symptoms [4, 19, 28], predictably obtaining full ROM preoperatively has been a main focus in rehabilitation. The ACL reconstruction should be performed once patients have minimal or no swelling, full ROM (full hyperextension and flexion compared with the opposite normal knee), and a good leg strength and control.

Although good results can be obtained with acute ACL surgery, the delay in ACL reconstruction is justified if one is to avoid or minimize the potential for postoperative ROM problems. Knee stiffness is a difficult problem to

resolve and can often result in a compromised, unpredictable final outcome. The best treatment of knee stiffness is prevention. Therefore, any reasonable way of avoiding or preventing a stiff knee, while still accomplishing the goal of knee stability, is worthwhile. The benefits of delayed surgery far outweigh the risks of proceeding with surgery too soon.

Postoperative rehabilitation program

A postoperative rehabilitation program that emphasizes obtaining full knee ROM, limiting hemarthrosis, and achieving good quadriceps leg control should prevent the complication of arthrofibrosis. Our early postoperative rehabilitation program to achieve full ROM is outlined in Table 3.

Every attempt should be made in the immediate postoperative period to prevent patella infera. Noyes et al. [11] reported the pathomechanics and treatment of developmental patella infera and associated arthrofibrosis after knee surgery. Active quadriceps control and knee flexion are the two most critical factors in preventing patella infera. When there is quadriceps inhibition and/or shutdown, the patellar tendon loses its built-in tensioner and may allow shortening and secondary contracture to develop [17]. Active straight leg raises can help maintain the tension in the patellar tendon. In addition, knee flexion is

equally important in stretching the patellar tendon along with its medial and lateral retinacular structures. Active straight leg raises and passive as well as active knee flexion should prevent significant shortening of the patellar tendon.

Discussion

Arthrofibrosis or a milder form of joint stiffness still remains the most common complication after ACL reconstruction [5, 6, 8, 10, 11, 14, 19]. Arthrofibrosis disrupts the normal knee kinematics and may lead to progressive degenerative changes in the knee [14]. Arthrofibrosis has usually been referred to as a postoperative extension deficit of 10° or more from neutral and knee flexion less than 125° [2, 7]. However, this definition refers to severer forms of arthrofibrosis. Less severe but still symptomatic cases of arthrofibrosis after ACL surgery, with various degrees of restricted knee motion and stiffness, are more common in clinical practice. Patients desire a normal knee postoperatively, and therefore, the goal is to restore full extension (including hyperextension as compared with the opposite normal knee). Therefore, arthrofibrosis has been defined as any symptomatic limitation in knee motion compared with the opposite normal knee [30].

The morbidity associated with a milder loss of knee extension is not well addressed in the literature. Although knees with arthrofibrosis are usually stable by all objective criteria, patients are seldom completely satisfied with their knee function on resumption of daily activities and/or sports. Patients who fail to obtain full knee extension after major ligament surgery experience anterior knee pain, swelling after prolonged standing or walking, crepitus on terminal extension, stiffness, impaired quadriceps muscle strength, altered gait pattern, slower progress in rehabilitation, decreased knee function, and difficulty returning to their desired level of activity [14]. Arthrofibrosis of any type is particularly bothersome to an athletically active individual because of persistent knee pain and quadriceps fatigue with sporting activities.

Loss of knee extension is a definite impairment to knee function and is subjectively distressing to the patient. Most surgeons believe that flexion contractures of greater than 10° are unacceptable. However, in our opinion, any degree of postoperative flexion contracture of the knee is undesirable. The belief that some "minor" degree of extension loss is acceptable as long as the patient has a stable knee and is able to play sports is the main reason we believe that many patients still do not consider their knee normal despite having achieved their postoperative goals.

If full symmetrical hyperextension of the knee is not achieved after surgery, then there is a reason to believe that "something" is preventing this. Since the normal ACL fits perfectly in the notch only during full hyperextension, any loss of knee hyperextension after surgery

Table 3 Postoperative ACL rehabilitation program to achieve full range of motion

| Time after surgery | Prescribed program |
|---------------------------|--|
| Day of surgery | Continuous passive motion (CPM) machine for elevation and gentle motion Cryo/Cuff for compression and cold therapy Full passive extension exercises for 10° min every waking hour Static flexion to at least 90° in the CPM machine 3 times a day Active quadriceps contractions; actively lift leg out of the CPM machine |
| Day after surgery | Same exercises as day of surgery Discharged to home if goals are met: full hyperextension equal to normal knee, flexion to at least 90°, good quadriceps leg control, minimal swelling |
| First week after surgery | Continue with prescribed exercises Limit activities to eating and bathroom privileges |
| Second week after surgery | Additional exercises added to program: prone hangs, wall slides Extension board used if needed Full weight-bearing with normal gait |
| 2–5 weeks after surgery | Additional exercises added to program: heel slides, stationary bicycle, stairmaster |

must be from a misfit of the reconstructed ACL in the intercondylar notch or from secondary contracture of the posterior knee structures. If either one of these problems is allowed to develop postoperatively, it is difficult to restore the knee to normal in the future. Our goal at surgery, therefore, should be to place the ACL graft in a precise location so that the original anatomy of the ACL can be restored, a symmetrical knee hyperextension with good stability can be obtained, and the knee is given a chance to become normal again.

When the ACL graft is accurately placed at surgery, a full ROM including hyperextension can be and should be obtained. There is some concern among surgeons that allowing or encouraging full knee extension postoperatively may lead to excessive stress on the graft with possible long-term laxity developing. Unfortunately, this misconception has led many surgeons to believe that full extension of the knee after surgery (especially if the opposite knee extends beyond 0°) is undesirable. Recently, Rubinstein et al. [18] have reported that restoring and maintaining immediate full hyperextension after ACL reconstruction does not adversely affect the ultimate stability of the knee.

The treatment of any type of arthrofibrosis of the knee can be a frustrating problem for the patient, the surgeon,

and the physical therapist [2]. The patient should be seen frequently in the clinic throughout the treatment period by both the surgeon and the physical therapist so that the degree of improvement can be assessed and the subsequent management plan continued. The patient needs to be cooperative, highly motivated, and patient while being treated for this difficult problem. Patients should be fully educated regarding the total management plan, the success rate, and the prognosis for the ultimate functional status of the knee after the procedure.

Arthrofibrosis of the knee represents a wide continuum of pathology, and a thorough understanding of this disabling condition and a systematic approach to its management are required to obtain a satisfactory functional result. The most effective method of treating arthrofibrosis is preventing its occurrence by appropriate timing of surgery until full ROM is obtained preoperatively and by early postoperative extension exercises. In recent years, ROM problems have been minimized by improved surgical technique and perioperative rehabilitation programs. The goal in ACL surgery is to enable patients to return to a state of full functional activities without compromising long-term stability and function of the knee with minimal or no complications.

References

1. Cosgarea AJ, DeHaven KE, Lovelock JE (1994) The surgical treatment of arthrofibrosis of the knee. *Am J Sports Med* 22:184–191
2. Cosgarea AJ, Sebastianelli WJ, DeHaven KE (1995) Prevention of arthrofibrosis after anterior cruciate ligament reconstruction using the central third patellar tendon autograft. *Am J Sports Med* 23:87–92
3. Del Pizzo W, Fox JM, Friedman MJ, Snyder SJ, Ferkel RD (1985) Operative arthroscopy for the treatment of arthrofibrosis of the knee. *Contemp Orthop* 10:67–72
4. Fisher SE, Shelbourne KD (1993) Arthroscopic treatment of symptomatic extension block complicating anterior cruciate ligament reconstruction. *Am J Sports Med* 21:558–564
5. Fullerton LR Jr, Andrews JR (1984) Mechanical block to extension following augmentation of the anterior cruciate ligament: a case report. *Am J Sports Med* 12:166–168
6. Graf B, Uhr F (1988) Complications of intra-articular anterior cruciate ligament reconstruction. *Clin Sports Med* 7:835–848
7. Harner CD, Irrgang JJ, Paul J, Dearwater S, Fu FH (1992) Loss of motion after anterior cruciate ligament reconstruction. *Am J Sports Med* 20:499–506
8. Jackson DW, Schaefer RK (1990) Cyclops syndrome. Loss of extension following intra-articular anterior cruciate ligament reconstruction. *Arthroscopy* 6:171–178
9. Klootwyk TE, Shelbourne KD, DeCarlo MS (1993) Perioperative rehabilitation considerations. *Oper Tech Sports Med* 1:22–25
10. Mohtadi NGH, Webster-Bogaert S, Fowler PJ (1991) Limitation of motion following anterior cruciate ligament reconstruction: a case-control study. *Am J Sports Med* 19:620–625
11. Noyes FR, Wojtys EM, Marshall MT (1991) The early diagnosis and treatment of developmental patella infera syndrome. *Clin Orthop* 265:241–252
12. Noyes FR, Mangine RE, Barber SD (1992) The early treatment of motion complications after reconstruction of the anterior cruciate ligament. *Clin Orthop* 277:217–228
13. Parisien JS (1988) The role of arthroscopy in the treatment of postoperative fibroarthrosis of the knee joint. *Clin Orthop* 229:185–192
14. Paulos LE, Rosenberg TD, Drawbert J, Manning J, Abbott P (1987) Infrapatellar contracture syndrome: an unrecognized cause of knee stiffness with patella entrapment and patella infera. *Am J Sports Med* 15:331–341
15. Poehling GG, Pollock FE Jr, Koman LA (1988) Reflex sympathetic dystrophy of the knee after sensory nerve injury. *Arthroscopy* 4:31–35
16. Richmond JC, Al Assal M (1991) Arthroscopic management of arthrofibrosis of the knee, including infrapatellar contracture syndrome. *Arthroscopy* 7:144–147
17. Rubinstein RA Jr, Shelbourne KD (1993) Preventing complications and minimizing morbidity after autogenous bone-patellar tendon-bone anterior cruciate ligament reconstruction. *Oper Tech Sports Med* 1:72–78
18. Rubinstein RA Jr, Shelbourne KD, Van Meter CD, McCarroll JR, Rettig AC, Gloyeske RL (1995) Effect on knee stability if full hyperextension is restored immediately after autogenous bone-patellar tendon-bone anterior cruciate ligament reconstruction. *Am J Sports Med* 23:365–368

19. Sachs RA, Daniel DM, Stone ML, Garfein RF (1989) Patellofemoral problems after anterior cruciate ligament reconstruction. *Am J Sports Med* 17:760–765
20. Salter RB, Hamilton HW, Wedge JH, et al (1984) Clinical application of basic research on continuous passive motion for disorders and injuries of synovial joints: a preliminary report of a feasibility study. *J Orthop Res* 1:325–342
21. Sapega AA, Moyer RA, Schneck C, Komalahiranya N (1990) Testing for isometry during reconstruction of the anterior cruciate ligament. Anatomical and biomechanical considerations. *J Bone Joint Surg [Am]* 72:259–267
22. Shelbourne KD, Johnson G (1994) Evaluation of knee extension following anterior cruciate ligament reconstruction. *Orthopedics* 17:205–206
23. Shelbourne KD, Johnson GE (1994) Outpatient surgical management of arthrofibrosis after anterior cruciate ligament surgery. *Am J Sports Med* 22:192–197
24. Shelbourne KD, Patel DV (1995) Management of combined injuries of the anterior cruciate and medial collateral ligament. *J Bone Joint Surg [Am]* 77:800–806
25. Shelbourne KD, Patel DV (1995) Timing of surgery in anterior cruciate ligament-injured knees. *Knee Surg Sports Traumatol Arthrosc* 3:148–156
26. Shelbourne KD, Patel DV (1996) Prevention of complications after autogenous bone-patellar tendon-bone anterior cruciate ligament reconstruction. *Instr Course Lect* 45:253–262
27. Shelbourne KD, Porter DA (1992) Anterior cruciate ligament-medial collateral ligament injury: nonoperative management of medial collateral ligament tears with anterior cruciate ligament reconstruction. *Am J Sports Med* 20:283–286
28. Shelbourne KD, Trumper RV (1998) Preventing anterior knee pain following anterior cruciate ligament reconstruction. *Am J Sports Med* (in press)
29. Shelbourne KD, Wilckens JH, Mol-labashy A, De Carlo M (1991) Arthrofibrosis in acute anterior cruciate ligament reconstruction: the effect of timing of reconstruction and rehabilitation. *Am J Sports Med* 19:332–336
30. Shelbourne KD, Patel DV, Martini DJ (1996) Classification and management of arthrofibrosis of the knee after anterior cruciate ligament reconstruction. *Am J Sports Med* 24:857–862
31. Sprague NF III (1987) Motion-limiting arthrofibrosis of the knee (1987) The role of arthroscopic management. *Clin Sports Med* 6:537–549.
32. Sprague NF III, O'Conner RL, Fox JM (1982) Arthroscopic treatment of post-operative knee fibroarthrosis. *Clin Orthop* 166:165–172
33. Steadman JR, Burns TP, Pelozo J, Sil-liman JF, Fulstone HA (1993) Surgical treatment of arthrofibrosis of the knee. *J Orthop Tech* 1:119–127
34. Strum GM, Freidman MJ, Fox JM, et al (1990) Acute anterior cruciate ligament reconstruction: analysis of complications. *Clin Orthop* 253:184–189
35. Youmans WT (1989) Surgical complication of the patellofemoral articulation. *Clin Sports Med* 8:331–342