



Operative Treatment of Arthrofibrosis of the Knee^{*†}

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The term arthrofibrosis has been used to describe a spectrum of knee conditions in which loss of motion is the major finding^{18,25,33,35,38,41-43,45}. Because different characteristics have been used historically to define this term^{18,25,33,35,38,41-43,45}, the universally accepted definition of this condition remains in question. We believe that arthrofibrosis is best defined as a condition of restricted knee motion characterized by dense proliferative scar formation, in which intra-articular and extra-articular adhesions can progressively spread to limit joint motion. This dense scar tissue can obliterate the parapatellar recesses, suprapatellar pouch, intercondylar notch, and eventually the articular surfaces⁹. Scarring of the infrapatellar fat pad and fibrosis of the patellar ligament can occur, with varying degrees of capsular and quadriceps contracture. Patella infera and chronic patellar entrapment may also develop as a consequence of this process⁹.

We consider loss of motion that is due to a localized intra-articular lesion as a separate clinical entity. Many patients who have this condition primarily lack full extension as the result of a so-called cyclops lesion²² or other anterior intra-articular scarring following reconstruction of the anterior cruciate ligament⁹.

Normal Knee Motion

Normal knee motion has been described as 0 degrees of extension to 135 degrees of flexion, although hyperextension is frequently present to varying degrees¹. In general, however, the best way to ascertain normal motion is to examine the contralateral knee if

it has no abnormal conditions. Maintaining a full range of knee motion requires congruent articular surfaces; adequate muscle function; an articular capsule with suitable capacity and flexibility; effective space in the medial and lateral articular recesses, intercondylar notch, and suprapatellar pouch; and sufficient meniscal motion. Impediments to any of these elements may cause loss of joint motion¹⁴.

Arthrofibrosis

Prevalence

Because most studies of arthrofibrosis that we are aware of included mixed subsets of patients with loss of motion following an injury or operation^{1,2,8,9,13,25,33,35,41-43,45}, the prevalence of true global arthrofibrosis is difficult to determine. Nevertheless, the prevalence of complications related to abnormal motion following reconstruction of the anterior cruciate ligament has reportedly ranged from 4 percent (forty-two of 959) to 35 percent (eleven of thirty-one)^{16,29,39,46}.

Causes of Limited Motion

In order to diagnose arthrofibrosis accurately, other causes of restricted active and passive motion of the knee must first be eliminated. Mechanical causes include loss of articular congruency (for example, due to a fracture), interruption of the extensor or flexor mechanism, a meniscal tear or a loose body, a substantial effusion, a cyclops lesion^{12,22}, and nonisometric placement of the graft during reconstruction of the anterior cruciate ligament^{34,35,43}. Unfortunately, arthrofibrosis may coexist with these problems. Various problems with the placement of an anterior cruciate ligament graft can reduce knee motion, and there are many possible operative solutions (Table I).

Other causes of limitation of active knee motion may be found during the diagnostic workup. The patient may have a neurological deficit in the form of neuropathic apraxia related to the use of a tourniquet or to another previous nerve injury. We saw a patient who had a neurological deficit from a lumbar disc herniation, with loss of knee motion as the primary symptom. The diagnostic

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TABLE I
PROBLEMS WITH PLACEMENT OF AN
ANTERIOR CRUCIATE LIGAMENT GRAFT AND THEIR TREATMENT

Problem	Treatment
Shallow placement of femoral tunnel; limited knee flexion	Excision of graft and revision reconstruction
Anterior placement of tibial tunnel; extension block due to impingement of notch	Notchplasty, partial or complete excision of graft, and revision reconstruction
Inadequate clearance of notch; extension block	Repeat notchplasty
Overtensioning of graft; loss of motion	Excision of graft
Cyclops lesion; impingement of notch and loss of extension	Excision of fibrous nodule

workup for limited knee motion should rule out infection and other inflammatory processes as well.

Any acute injury causing pain may limit active knee motion. Quadriceps inhibition often occurs concomitantly¹¹. In most patients, limitations of motion resolve as the pain and effusion dissipate. In the presence of various pain syndromes, such as complex regional pain syndrome⁴⁴, additional muscle inhibition can occur. This quadriceps inhibition may allow scar tissue to form while the knee is held in a flexed position, and activities of daily living become more difficult to perform.

A knee effusion also limits knee motion by inhibiting the function of the quadriceps muscle¹¹. Atrophy of the quadriceps muscle and flexion contracture usually result and complicate treatment.

Joint immobilization can complicate all of these factors. Disuse may induce abnormal cross-links between collagen fibers at abnormal locations^{43,45}, decreasing their extensibility³ and promoting intra-articular and extra-articular scarring.

The timing of reconstruction of the anterior cruciate ligament in relation to an acute injury is believed to be a factor in loss of motion^{39,46}. Some believe that an operation performed within three weeks after an injury may increase the likelihood that knee motion will be lost^{40,50}, although others disagree²¹. Poor rehabilitation⁴³ preoperatively or postoperatively, with delayed motion protocols, may further increase the risk³⁹. Following reconstruction of the anterior cruciate ligament, loss of extension is most common and frequently is due to a localized intra-articular lesion. This lesion differs from arthrofibrosis throughout the joint (global arthrofibrosis), which manifests as marked limitation of flexion, extension, and patellar glide associated with widespread joint inflammation as well as intra-articular formation of fibrous tissue, which can progress to chondrification and ossification of soft tissues⁹.

Although arthrofibrosis occurs primarily as the result of an abnormal fibrous hyperplasia³⁴, the etiology is more likely multifactorial, and almost all cases are asso-

ciated with an injury or operation. Although the previously mentioned conditions, such as complex regional pain syndrome (which includes reflex sympathetic dystrophy) or infection, may lead to arthrofibrosis^{1,33,43,45}, the disorder may be due to a primary idiopathic overproduction of fibrous tissue.

Clinical Manifestations

Symptoms vary and often do not correlate with the severity of the condition. Stiffness is usually the primary symptom and is often worse in the morning hours. Patients may complain of a warm, swollen knee that is painful with attempted motion. Crepitation and weakness are frequently present, with swelling following prolonged standing or walking. Even when the patient does not have pain, loss of motion and quadriceps weakness can be substantial impediments to the performance of activities of daily living.

Since arthrofibrosis usually occurs after trauma or an operative procedure, pain may be the initial symptom. The presence of pain may complicate preexisting knee stiffness^{9,26}. Pain may be secondary to arthritis and therefore may vary with activity. Pain may also be constant, especially when it is associated with complex regional pain syndrome^{9,26}. Pain alone may prevent certain patients from regaining motion after an operation or injury. Quadriceps function can be decreased or absent because of pain²⁶. Often the knee is held in a flexed position, which encourages tightening of the posterior part of the capsule and the hamstrings. Although pain can be present early, it often becomes more prominent when joint degeneration and arthritis occur as a result of long-standing arthrofibrosis.

Clinical signs are also present in varying degrees. An antalgic, flexed-knee gait is often seen³⁴. Increased warmth around the knee is occasionally noted. Anatomical structures are often difficult to palpate because of tissue-swelling (Fig. 1-A). Although effusion may be present, swelling is more often due to inflamed, thickened capsular and pericapsular tissues. Atrophy of the quadriceps muscle may be severe, depending on the stage of the process, and is accompanied by loss of quadriceps strength. Active and passive knee flexion and extension are often restricted, and medial-lateral and superior-inferior patellar glide is reduced. This restriction of passive motion often has a spring-like end point, reflecting the density and stiffness of the thickened, inflamed, or scarred peripatellar tissue.

If the inflammatory process continues, arthrofibrosis can severely limit knee motion. This is typified by the patient who is subjected to inappropriately intensive and sometimes painful rehabilitation. For example, the total arc of knee motion can be reduced from 130 degrees to 40 degrees over a period of several weeks. Such a progressive course is typical of arthrofibrosis. In contrast, loss of motion due to a focal lesion usually reaches a specific limit and does not worsen.



FIG. 1-A

Figs. 1-A and 1-B: A patient who had global arthrofibrosis.

Fig. 1-A: Photograph demonstrating capsular thickening, typical of global arthrofibrosis, in the left knee. Hypertrophic synovial tissue and scar obliterate normal palpable landmarks and severely limit patellar mobility.

Pathological Findings

Pathological findings at the time of the operation frequently include degeneration of the articular cartilage, abundant formation of fibrous tissue, and soft-tissue chondrification and ossification (Fig. 1-B). Since motion is needed to maintain healthy articular cartilage, loss of motion may lead to softening and degeneration of the hyaline cartilage³⁷. In one study, the prevalence of degenerative arthritis of the knee was lower in patients who were operated on within six months after the index procedure or the injury than in those who were managed later⁹. Grade-II, III, or IV chondromalacia (according to Outerbridge's classification system³²) was found in sixteen of nineteen patients with arthrofibrosis in one study³⁵ and in forty-two (91 percent) of forty-six in another²⁵. Patients with preexisting changes in the articular cartilage may have an acceleration of the degenerative process⁹. Subsequent arthritis may also increase the level of pain. In one series, formation of osteophytes was documented in 89 percent (thirty-one) of thirty-five patients who had arthrofibrosis, and 20 percent (seven) had marked joint-space narrowing compared with the contralateral knee⁹. Soft-tissue calcification or heterotopic ossification, or both, was found in 51 percent (eighteen) of the thirty-five patients. Even with prompt operative treatment and appropriate daily rehabilitation, arthritis may be the end result^{28,34,42,45}.

In some patients, dysfunction of the quadriceps muscle and alterations of the patellar ligament may lead to patella infera, which is a severe complication of arthro-

fibrosis^{28,51}. Treatment of patella infera is difficult. Proximal transfer of the tibial tubercle, lengthening of the patellar ligament, and other methods have been advocated for the treatment of this condition; however, no treatment that we are aware of has proved to be highly successful^{9,34}. Patella infera was found in 9 percent (three) of thirty-five patients⁹ and 15 percent (four) of twenty-six patients³⁴ who had varying degrees of arthrofibrosis.

Loss of Motion

Even minor losses of knee motion may have adverse effects. While it is common to lose both flexion and extension, loss of extension is usually more debilitating. A relatively small extension deficit impedes normal walking function, whereas restricted flexion does not severely affect gait as long as the knee can be flexed at least 60 degrees³. Diminished running speed is associated with loss of flexion of 10 degrees or more⁹, whereas loss of extension of more than 5 degrees may cause patellofemoral pain and a limp during walking³⁶. An extension deficit of more than 10 degrees is poorly tolerated by active people³⁴, and loss of more than 20 degrees may cause a functional limb-length discrepancy⁹. As function of the quadriceps muscle decreases, the ability of the muscle to function as a shock absorber is lessened, which may lead to additional articular degeneration.

Loss of extension usually is associated with scar formation in the anterior compartment of the knee, although fibrosis in the posterior compartment may also contribute to loss of extension (Fig. 2). Fibrosis in the anterior compartment includes proliferative fibrosis of



FIG. 1-B

Lateral (left) and anteroposterior (right) radiographs demonstrating extensive ossification of the infrapatellar fat pad (arrow).

the infrapatellar fat pad and obliteration of the infrapatellar bursa. Fibrous scarring within the intercondylar notch leads to impingement of the anterior cruciate ligament with knee extension^{1,14}. Limitation of meniscal motion, especially in the region of the anterior horns, secondary to fibrosis of the coronary ligaments can also limit extension. Contracture of the posterior capsular structures may limit extension as well, particularly in patients who have a long-standing flexion contracture¹.

Loss of flexion usually is associated with intra-articular fibrosis¹⁴ and scarring in the patellofemoral mechanism (Fig. 3)^{1,14}. Patellar mobility is invariably decreased. This anterior scar usually consists of adhesions of the quadriceps expansion to the lateral and medial recesses¹, adhesions within the suprapatellar bursa¹, adhesions of the quadriceps muscle to the femur^{1,30}, patella infera²⁸, or shortening of the rectus femoris muscle¹. When knee stiffness is due to extra-articular causes, the prognosis for improving motion is less favorable.

Pathophysiology

Arthrofibrosis may occur as the result of the inflammatory cascade due to injury or operative treatment. Although inflammation is undoubtedly present in a large number of individuals, it is not clear why an aggressive form, which can last for months, and later pathological intra-articular and periarticular fibrosis develop in some patients.

Intra-articular adhesions form early in the disease process, and capsular contracture may develop⁸. There

can be extensive involvement, including that of both intra-articular and extra-articular structures to varying degrees. This development may be aggravated by quadriceps dysfunction, contracture, and adhesions, which further decrease motion. In severe cases, the peripatellar recesses, suprapatellar pouch, and intercondylar area are completely obliterated by dense fibrous tissues and adhesions.

Histological examination of synovial tissue from arthrofibrotic knees reveals dense fibrovascular tissue and chondrometaplasia^{9,18}. Less consistent findings include hemosiderin, synovial proliferation, fibrin, and enchondral ossification^{18,22,46}. Similar histological features have been found in association with all degrees of severity of arthrofibrosis¹⁸. Knees that have been affected by the disease for more than six months are more likely to have calcification and enchondral ossification⁹ as chondrometaplasia matures¹⁸. Histological examination of fibronodular lesions (so-called cyclops lesions) after reconstruction of the anterior cruciate ligament demonstrates similar histological findings^{12,22}. This tissue has been described as a dense fibroconnective tissue rich in newly formed vessels, occasionally associated with cartilage, newly formed bone tissue, and necrotic lamellar bone.

Treatment

The goal of operative treatment of arthrofibrosis is restoration of normal motion without inflicting additional damage on the joint. However, the ideal approach

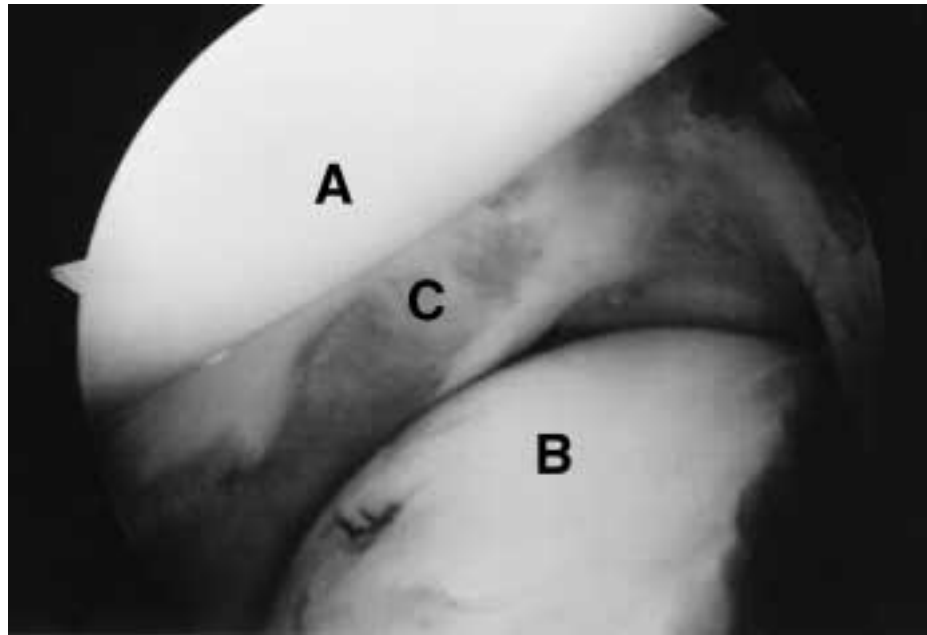


FIG. 2

Arthroscopic view from the superior portal toward the infrapatellar fat pad. The normal soft, yellow fat is replaced by dense fibrous tissue. This scar may be difficult to distinguish from articular surfaces. A = patella, B = medial femoral condyle, and C = fibrotic fat pad.

to arthrofibrosis is prevention³¹. This is best accomplished with use of nonoperative modalities to control pain and inflammation after an injury or operation. These modalities include rest, ice, compression, elevation, aspiration of areas of effusion, nonsteroidal anti-inflammatory medication, and narcotic pain medication. A short-term, tapered oral regimen of steroids may be useful for controlling severe inflammation in some patients. As flexion is more easily obtained than extension, efforts should be directed toward maintaining extension. An operation for an acute injury is best performed after inflammation, edema, and pain have decreased and muscle strength and the range of motion have been regained (typically at one to three weeks after the injury). Preoperative and postoperative immobilization should be kept to a minimum, and early rehabilitation programs are advocated³⁹.

When a plateau has been reached during rehabilitative efforts to restore motion or when there is progressive loss of motion, additional intervention is indicated¹. If appropriate treatment is given early after motion becomes restricted and the inflammatory process is controlled or runs its course, physical therapy emphasizing restoration of a range of motion and, occasionally, gentle manipulation of the knee under anesthesia may result in improvement. When the inflammatory process persists, especially after the third month, these treatments are less successful⁸.

Forced manipulation of an extensively scarred knee may be harmful. Closed manipulation or vigorous attempts to gain passive motion may cause indiscriminate tearing of intra-articular tissue³³, excessive tibiofemoral and patellofemoral compression with the risk of chon-

dral damage or fracture^{8,14,33,43}, rupture of the patellar ligament^{8,33}, and even femoral fracture³³. Manipulation may also initiate complex regional pain syndrome⁸. Experimental evidence in rats and in human cadaveric knees has suggested that forced manipulation can cause tearing of the proliferative connective tissue in a plane different from the original joint space. Additionally, articular cartilage fragments can be sheared off, leaving residual chondral defects on the joint surfaces^{14,15,19}.

Before the advent of arthroscopy, open operative procedures were used to treat limitations of knee motion. While many open methods have been described for treating restricted knee flexion^{10,20,24,27}, these operations can be extensive, with considerable risk of morbidity, and they often necessitate immobilization followed by extensive rehabilitation efforts^{27,47}.

Arthroscopic treatment is the procedure of choice when limitation of motion is nonprogressive and primarily due to a discrete intra-articular lesion. Arthroscopy is less invasive than open procedures, and it allows lysis of intra-articular adhesions under direct visualization^{1,8,13,23,33,35,43}.

In general, arthroscopic treatment has been found to be more effective for improving knee flexion and less so for improving knee extension⁴³. There may be several reasons for this. First, loss of extension may represent inappropriate tensioning or positioning of a cruciate reconstruction in addition to adhesion formation. Second, intercondylar and pericruciate adhesions that contribute to loss of extension¹⁴ can be more difficult to resect adequately without disrupting cruciate integrity. Finally, posterior intra-articular and extra-articular contracture may be an important cause of the loss of exten-

sion, and this is less easily addressed arthroscopically. When there are dense adhesions in a global pattern, operative treatment of arthrofibrosis may entail a combination of arthroscopic and open methods to restore motion of the knee.

Results of Treatment

Appropriate and timely operative treatment generally improves knee motion. Recent reports on the treatment of varying degrees of motion loss have generally described arthroscopic evaluation and excision of fibrous tissue, with retinacular releases, and gentle manipulation of the knee followed by an appropriate rehabilitation program (as will be described in the Postoperative Care section). As demonstrated by many studies, considerable gains in motion are possible after operative treatment^{1,2,8,9,13,25,33,35,41-43,45,49}.

The time-period between initiation of the inflammatory process and appropriate treatment is considered to be a major factor influencing improvement^{1,9}. The ideal interval ranges from three to nine months after the initial operation or injury^{1,8,9}. Operative treatment one year or more after initiation of the process has been less successful in improving the range of motion^{1,43}.

Although the results appear to be better after early intervention, an intense inflammatory reaction develops in some patients following an injury or operation. In these patients, early intensive attempts to regain motion or early operative intervention may exacerbate the un-

derlying condition. Medical treatment of inflammation and pain should be used in conjunction with a program of gentle motion until the inflammatory reaction has decreased.

Because of the heterogeneity of patient groups and the diversity of abnormalities, it is not possible to compare reported outcomes of operative treatment of motion loss secondary to an isolated intra-articular lesion with outcomes of treatment of global arthrofibrosis. Nevertheless, as noted previously, outcomes have generally been favorable. Most patients appear to have some improvement, although the functional outcome is associated with the extent of the arthrofibrosis⁹. Operative treatment of the localized anterior intra-articular variant resulted in better subjective and objective outcomes than did operative treatment of extensive (global) dense scar formation⁹.

Operative Technique

Muscle function, flexion, extension, and patellar mobility should all be documented preoperatively and compared with those of the contralateral, normal knee. With the patient supine and fully anesthetized, the hip should be brought to 90 degrees of flexion, with gravity allowed to flex the knee. Measurement with the limb in this position most accurately reflects the true flexion limit. Next, the hip should be allowed to extend, with the lower extremity held horizontally; while the heel is supported, the limit of extension is measured. Finally,

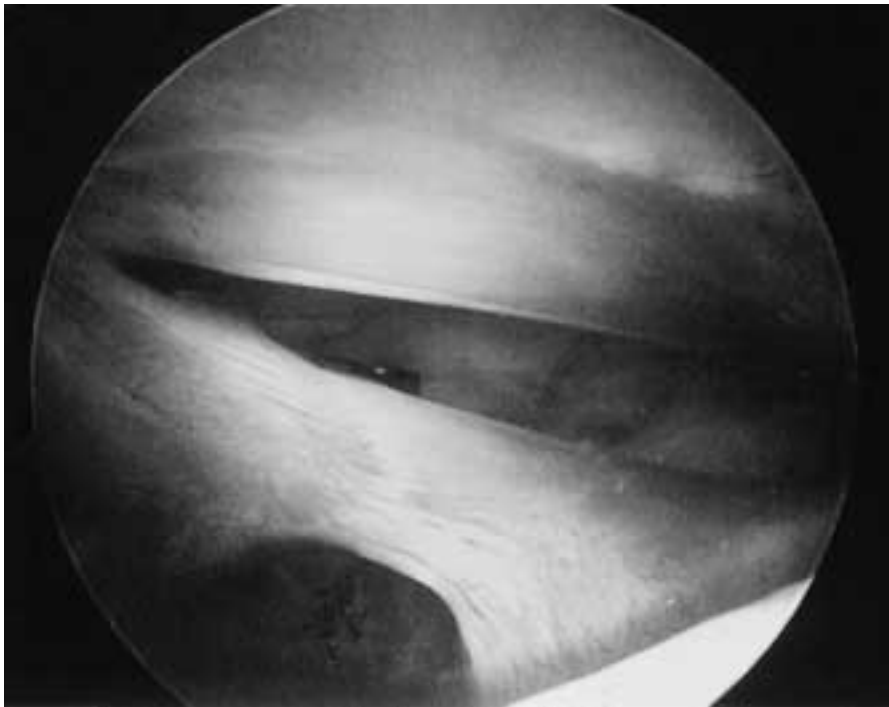


FIG. 3

Arthroscopic view from the inferior portal toward the suprapatellar pouch. In this early case of arthrofibrosis, scar is forming but it incompletely blocks the view of the articularis genu, the normal visual marker for the proximal extent of the suprapatellar pouch. In more advanced cases, the scar forms a solid wall from the proximal edge of the patella to the suprapatellar fat pad, completely walling off most of the suprapatellar pouch.

patellar mobility should be noted with regard to medial-lateral glide, superior-inferior glide, and patellar tilt. The amount of glide that is normally present is influenced by the amount of knee flexion. The greater the flexion angle, the more the patella is captured in the femoral sulcus. In order to make a valid comparison with the unaffected side, the contralateral extremity should be placed in the same degree of flexion and an attempt should be made to assess patellar mobility at this flexion angle. Near the end of the procedure, when full extension is achieved, patellar mobility should again be assessed to confirm that it is symmetrical with that of the other knee at or near full extension.

While a primary goal of operative treatment of arthrofibrosis is the restoration of the range of motion, a second and probably more important goal is the preservation of articular cartilage. Articular cartilage is almost always affected in patients who have severely limited motion. Damage may result from a prolonged loss of motion. Arthrofibrosis is equivalent to internal immobilization of the joint. Therefore, there may be areas of the joint surface that have had lower joint-contact forces for a period of time. This prevents the normal exchange of nutrients by compression-expansion (or biphasic function) of articular cartilage, which may lead to breakdown if the condition persists. Inappropriate rehabilitation may further jeopardize these surfaces because of extreme, excessive contact force¹⁴. Shortening and stiffening of the medial and lateral retinacula can cause marked increases in the patellofemoral contact forces¹⁷.

While it is tempting to try to first force both flexion and extension during a manipulation, the surgeon should keep in mind the degenerative state of the articular cartilage; a joint affected by arthrofibrosis will be less tolerant of the high forces generated by these procedures. Forced manipulation may cause severe, irreparable damage to the articular cartilage surfaces, avulsion of the patellar ligament or the quadriceps tendon, or fractures in older patients who have disuse osteopenia.

Patience and perseverance are the keys to successful treatment of severely stiffened joints. Safe mobilizing procedures are often long, difficult, and tedious. Operative release of fibrotic tissues and débridement of the joint should usually precede an attempt to manipulate a joint. When a specific motion is not achieved with only minimum overpressure, the surgeon should readdress the area of the knee that may be limiting this motion. If motion cannot be achieved with relatively gentle force while the patient is anesthetized, it is unlikely to be achieved by rehabilitation. Iatrogenic complications can be catastrophic in this type of situation.

Entrance into the Joint

Placement of the arthroscopic cannula can be quite difficult in patients who have a flexion contracture and fibrotic retinacula. Placement should never be forced,

particularly when the cannula is passed next to an area of articular cartilage. Stiff and scarred retinacula may prevent passage of an arthroscopic cannula between the patella and the trochlea. If the cannula is forced into this area, it may shear off the softened articular cartilage as it is advanced.

The fat pad is often fibrotic and enlarged to fill the entire anterior compartment of the knee. Initial placement of the arthroscope into the area of the notch is often impossible. Also, the scar may be thick enough to impede normal movement of the arthroscope and instruments once the joint has been penetrated.

Entrance into the joint may be gained through a superior portal, proximal to the patella in the suprapatellar pouch. In moderately involved knees, a sweeping motion of the cannula through the suprapatellar pouch may provide enough working space for visualization and débridement in the suprapatellar area.

Our preferred method for the treatment of a severely contracted knee is to start with a lateral retinacular release. Skin and subcutaneous tissue are separated percutaneously from the underlying retinaculum between the inferior and superolateral portals. A blind lateral release is then performed. A heavy scissors is often necessary to divide this very tough retinacular tissue. This may provide enough visualization space to begin the arthroscopic procedure. The arthroscope should be placed from the inferolateral portal up into the area of the lateral release. The surgeon should avoid passing instruments between the patella and the trochlea until this can be done with minimum force and under direct visualization. If the patellar-trochlear space is still too restricted for adequate arthroscopic evaluation, a partial medial release may be needed. Care should be taken not to detach the vastus lateralis or vastus medialis from the quadriceps tendon as this may cause more quadriceps inhibition. Limiting the superior margin of the retinacular release to the superior pole of the patella laterally and the midpoint of the patella medially usually prevents this complication.

Extension

Full extension is usually more difficult to obtain than flexion. The tissues preventing full extension are often tougher than those encountered in the suprapatellar pouch. There are many causes for loss of extension, and they are more varied than those for loss of flexion. All areas of involvement must be thoroughly addressed to achieve full extension.

The infrapatellar fat pad is often completely obliterated by fibrous and cartilaginous tissue (Figs. 4-A and 4-B). Normal demarcations between the patella, the fat pad, and the menisci may be absent. All scar tissue in the anterior aspect of the knee must be excised without debriding the articular cartilage from the patella or removing the anterior horns of the menisci. It may be difficult to discern the fibrotic fat pad from the patellar

ligament. We sometimes have found it extremely difficult, if not impossible, to debride this tissue arthroscopically. The tissue is so tough (occasionally even ossified) that arthroscopic instruments are often useless for débridement.

A small medial parapatellar tendon arthrotomy is often used to initiate débridement in the anteroinferior aspect of the knee. The medial margin of the patella is palpated, and an incision is made from the inferior aspect of the patella to the tibial tuberosity. A plane between the patellar ligament and densely adherent scar tissue is developed with care, from the medial aspect of the patellar ligament to just beyond the lateral aspect. The patellar ligament can be retracted and protected with a right-angle retractor. All tissue posterior to the ligament from the inferior pole of the patella to the top of the tibial tuberosity must be removed (Figs. 5-A and 5-B). The anterior horns of the menisci and the intermeniscal ligament should be sharply dissected from this dense scar tissue and protected. Often, scissors will not cut this very dense tissue and a number-11 scalpel blade is needed to excise it. The bulk and density of this tissue, which are often similar to those of the normal patellar ligament, can be surprising. In the normal knee, the patellar ligament is separated from the anterior aspect of the tibia by the subpatellar bursa. The tendon is free to move anterior to the tibia proximal to the tuberosity. This space needs to be reestablished in order to allow full patellar mobility. If necessary, distal completion of the medial and lateral retinacular releases may

be undertaken. There is frequently very dense scar tissue from the previously created inferior portals to the anterior joint surface, and this tissue may continue to limit the mobility of the patellar ligament. The surgeon may use either arthroscopic or direct visualization at this point to complete these releases. Adequate lateral release is achieved when the patella can be everted 45 degrees.

Full extension usually is not achieved at this point. Other lesions may still prevent extension. Scarring over the anterior aspect of the anterior cruciate ligament, a cyclops lesion, or obvious impingement at the top of the notch can be addressed next.

It is important, at this point, to address the mobility of the menisci. One of the reasons for a lack of full extension is that the menisci are scarred down in the flexed-knee position. Thompson et al.⁴⁸ showed that, in the normal knee, there is considerable anterior-posterior excursion of both menisci with flexion and extension. If the menisci are held in a posterior position in a knee during flexion, full extension will be blocked. Arthrofibrosis can bind the menisci down and render them immobile in a posteriorly displaced position. This is analogous to a displaced bucket-handle tear. With extension, the meniscus cannot glide anteriorly and therefore acts as a wedge against the femoral condyle, preventing full knee extension. A probe can be placed around the meniscus, and an attempt should be made to displace the meniscus anteriorly. Initially, it will be very immobile. At this point, the surgeon should take



FIG. 4-A

FIG. 4-B

Fig. 4-A: Drawing showing the lateral view of a normal knee. Normal suprapatellar and infrapatellar fat pads are pliable and easily separated from surrounding tissue. The suprapatellar pouch extends proximally to the articularis genu.

Fig. 4-B: Drawing showing the lateral view of a knee with arthrofibrosis. The fat pads are stiff and replaced by dense fibrous tissue. The infrapatellar fat pad is adherent to the patellar ligament, the articular margin of the patella, and the anterior aspects of the menisci. The subpatellar bursa is obliterated. The suprapatellar pouch is divided into a small inferior area and a larger superior area by a wall of scar extending from the proximal pole of the patella to the anterior aspect of the femur.

time to make a gutter from the midportion of the meniscus to the anterior horn by removing all scar superficial to the tibia and deep to the capsule to a depth of four to five millimeters. As this removal progresses, the menisci become more and more mobile when pulled anteriorly with a probe. Once this has been accomplished, nearly full extension should be achieved. If not, a posterior release may be necessary.

A posterior capsular release is occasionally necessary in patients with the most advanced cases of arthrofibrosis with flexion contracture. However, release of the menisci as previously described has greatly lessened the need for posterior approaches.

The posterior oblique ligament should be addressed first. An incision is made in the posteromedial corner of the knee, just posterior to the medial collateral ligament and just anterior to the edge of the sartorius fascia. The sartorius fascia is reflected posteriorly, and the medial head of the gastrocnemius can be visualized. Blunt dissection separates the gastrocnemius muscle from the posterior aspect of the capsule, and the posterior oblique ligament can be visualized. The posterior border of the medial collateral ligament is visualized, and a vertical incision is made along the posterior edge of the medial epicondyle, distally along the posterior edge of the medial collateral ligament. Scissors can be placed under the posterior oblique ligament at this point, and the ligament can then be dissected off the femoral attachment. Extension is assessed at this point, and if it is still limited a posterolateral release may be necessary.

The lateral approach starts at the tip of the fibula and follows the anterior border of the short head of the biceps tendon. The fascia over the biceps is incised, and the short head is visualized and can be retracted posteriorly. The lateral tendon of the gastrocnemius is visualized; it is often intimately associated with the posterior aspect of the capsule on the lateral side. It may be difficult to separate the gastrocnemius tendon from the posterolateral aspect of the capsule. The surgeon can incise the lateral aspect of the capsule along the anterior edge of the lateral head of the gastrocnemius muscle, which surrounds the lateral side of the knee and approaches the lateral epicondyle. Care should be taken not to incise the lateral collateral or popliteofibular ligament. It may be necessary to release the gastrocnemius as well as the posterolateral aspect of the capsule. The posterolateral aspect of the capsule is quite dense, and its partial release often gains the last few degrees of extension. In rare cases, complete division of the posterior aspect of the capsule is necessary.

A posterior capsular release can be accomplished safely through at least two approaches: posteromedial and posterior. The posteromedial approach utilizes the interval that is posterior to the vastus medialis but anterior to the pes anserinus. The junction of the joint capsule and the periosteum, where the posterior femoral condyle meets the femoral diaphysis, is the de-



FIG. 5-A

Drawing showing a medial parapatellar incision used to remove dense anterior scar tissue. Planes must be created between the patellar ligament and the fibrotic fat pad and between the anterior aspect of the tibia and the posterior aspect of the fat pad. All scar tissue from the inferior pole of the patella to the tibial tuberosity must be excised.

sired level for division. Care must be taken to retract the neurovascular bundle posteriorly with the knee in flexion, as the capsule is incised from medial to lateral. This approach is adequate in thin knees.

In large knees, especially those with substantial adipose tissue, it is probably easier to expose the posterior aspect of the capsule through the semimembranosus-medial gastrocnemius interval, with the patient in either the lateral or the prone position^{6,7}. Usually, the medial head of the gastrocnemius muscle and tendon can be retracted laterally with the neurovascular bundle to give safe access to the posterior aspect of the capsule. Occasionally, a partial medial gastrocnemius tenotomy is needed for adequate exposure.

At this point, the tourniquet is released to ensure that a vascular injury has not occurred, pulses are palpated distally and compared with those on the other side, and a gentle manipulation of the knee into flexion and extension is attempted. Motion of the affected knee should now be close to that of the contralateral knee. Large suction drains are used to prevent a postoperative hematoma.

Flexion

Scarring in the suprapatellar pouch and the medial and lateral recesses is the most frequent cause of restriction of flexion. O'Connor³⁰ described a cord of scar that runs from the superior pole of the patella to the femoral shaft just superior to the top of the most proximal articular surface. In many patients, this is not simply a cord of tissue but a complete band of scar that completely obliterates the proximal portion of the suprapatellar pouch. In a normal knee, a view of the suprapatellar pouch from distal portals should reveal the vastus intermedius rising off the femoral shaft. The

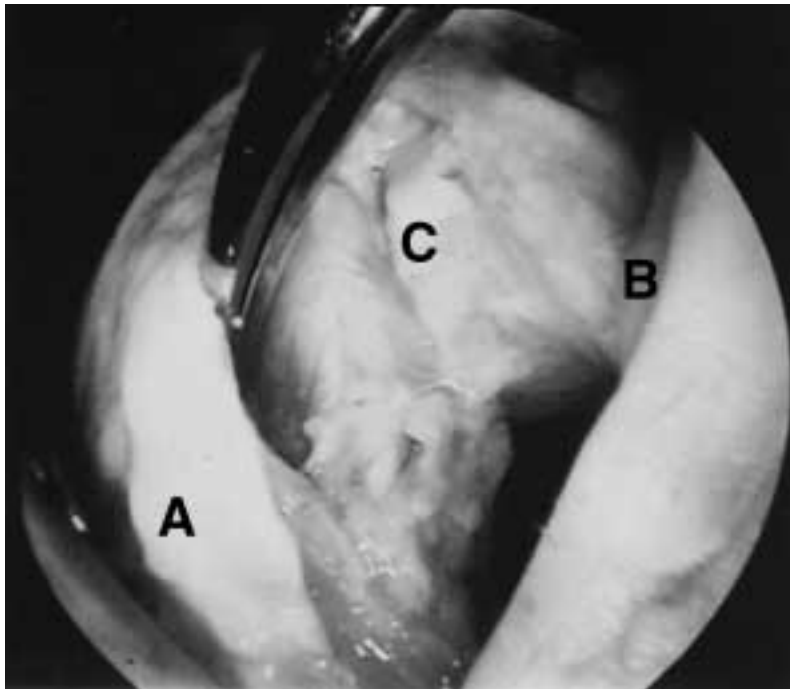


FIG. 5-B

Arthroscopic view showing the patellar ligament (A), the tibial plateau (B), and the fibrotic fat pad (C).

goal of this part of the procedure is to restore the full capacity of the suprapatellar pouch by releasing the remaining adhesions of the patella and the retinaculum to the femur.

Fibrous tissue may be so dense that it is difficult to determine where articular cartilage ends and scar begins. It may be easier to identify the proximal portion of the femoral articular cartilage than the proximal extent of the patellar articular cartilage. Therefore, it is often easiest to begin this débridement by making a hole in the dense curtain of tissue proximal to the trochlea, near the femoral surface or in the center of this tissue. The surgeon may find the tissue to be much too dense to be effectively debrided by even the most intensive use of motorized shavers. Frequently, arthroscopic knives, heavy scissors, or robust baskets are needed to incise this tissue. Once a hole is made in the wall of tissue, the full extent of the suprapatellar pouch can be visualized. It may become necessary to place a knife through the superomedial or superolateral portal to excise this tissue. The free edges of scar are more easily debrided with motorized shavers. Care should be taken to debride the wall of scar completely from the anterior femoral surface to the superior pole of the patella. The more organized fibers of the quadriceps tendon running from the now visualized vastus intermedius to the proximal pole of the patella should be distinguishable from the relatively homogeneous dense scar. Care should be taken not to debride any of the quadriceps tendon or its proximal attachment to muscle.

The next step is to clear both the medial and the lateral recess of scar tissue. Usually, dense bands of fi-

brous tissue run from the sides of the femur to the retinaculum. The surgeon should begin proximally and clear all abnormal tissue from the femur to the retinaculum, working toward the distal extent of the recesses until everything has been debrided to the tibial surface. The scarring process often thickens the retinacula to several times their original size.

At this point, it should be relatively easy to pass the arthroscope from the inferior portal into the suprapatellar pouch and the medial and lateral recesses. The normal volume of these areas should have been re-established, and full inspection of the articular surfaces should be undertaken. The hip should again be brought into 90 degrees of flexion, and gravity should be allowed to flex the knee. A marked increase in knee flexion is usually seen at this point. A gentle manipulation may be done to increase flexion. If flexion is still limited, the suprapatellar pouch and the recesses need to be reinspected, and any remaining scar must be debrided.

Occasionally, gross anterior placement of the femoral tunnel during reconstruction of the anterior cruciate ligament may lead to limited flexion of the knee. Full flexion, therefore, will not be achieved until the graft is partially or completely excised. An adequate notchplasty as well as elimination of any cyclops lesion should be ensured before this is undertaken²².

Postoperative Care

Postoperatively, patients who have had extensive releases may benefit from continuous epidural anesthesia, intra-articular injection of Marcaine (bupiva-

caine; twenty milligrams of 0.25 percent solution), and administration of morphine (ten milligrams for adults). Epidural anesthesia is usually not necessary unless complex regional pain syndrome is suspected, in which case epidural anesthesia is continued for forty-eight to seventy-two hours. When epidural management is used, precautions must be taken to prevent distension of the bladder and pressure ulcers. A continuous-passive-motion machine has been shown to be helpful for some patients after the drains have been removed⁹, although active motion is preferred when it can be accomplished. A continuous-passive-motion machine can be used for as many hours as can be tolerated by the patient. Unfortunately, these devices are not useful for maintaining extension. Since extension is more difficult to achieve than flexion, extension should be emphasized while the patient is still in the hospital. If a continuous-passive-motion machine is not needed, a well padded extension splint may be most comfortable for the first twenty-four hours. The patient may also begin heel-hangs while prone on a table with the distal aspect of the femur (thigh) on the edge of the table. The knee and leg are not supported, and this position encourages full extension. The heel may also be supported on a bolster with the patient supine, and weights or manual pressure may be applied to the knee on an hourly basis for several minutes to achieve full extension. Splinting with the knee in full extension at night may be required to maintain this position.

Excessive force should not be used to achieve a range of motion in the postoperative period. Articular cartilage is still in a compromised state, and forced motion or extended immobilization in extension casts may cause additional damage. Motion is encouraged to restore the health of the articular cartilage. When the patient can achieve flexion and extension in the hospital, the epidural catheter may be removed and the treatment should be switched to adequate pain medication for outpatient use. Postoperative physical therapy should concentrate on maintaining full extension, with

active quadriceps contraction. This may require electrogalvanic muscle stimulation. Effusions should be aspirated if they are large enough to cause pain, inhibit the quadriceps, or limit the range of motion. All reasonable physical therapy measures, such as ice and compressive wrapping to control pain and effusion, should also be applied. The patient may need daily physical therapy for weeks in order to maintain the gains that were achieved by the operative procedure.

The patient may begin toe-touch weight-bearing postoperatively, but the weight should be adjusted according to the condition of the articular cartilage and the degree of control of the quadriceps muscle by the patient.

Complications

The most common complication of operative treatment of arthrofibrosis is the inability to substantially improve motion or the loss of motion after initial improvement^{9,12,42,43}. The rates of reoperation after arthroscopic débridement and manipulation have ranged from one of fifteen to nineteen (43 percent) of forty-four^{13,42,43,45}, and repeat operative procedures have primarily been combinations of closed manipulation and arthroscopic and open techniques. If full extension and flexion cannot be maintained, full extension should be the first goal.

Other complications of operative treatment have been reported in the literature, and the magnitude of these complications has been related to the severity of the arthrofibrosis^{9,13,42,43}. The reported complications have included skin-tearing⁴³, unicortical fracture of the femoral condyle, superficial wound infection, deep wound infection⁹, septic arthritis, peroneal nerve palsy⁹, partial rupture of the patellar ligament, patellofemoral pain syndrome, and postoperative bleeding requiring reoperation^{1,8,9,25,45}. We also consider patients who have operative treatment of arthrofibrosis to be at high risk for deep venous thrombosis, and we recommend mechanical and chemical prophylaxis.

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