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TECHNIQUE

Arthrofibrosis: Evaluation, Prevention, and Treatment

R. Alexander Creighton, MD and Bernard R. Bach, Jr., MD
Division of Sports Medicine
Midwest Orthopaedics at Rush
Rush University Medical Center
Chicago, Illinois, U.S.A.

ABSTRACT

Loss of motion of the knee is a challenging dilemma to both the patient and the treating physician. The causes of arthrofibrosis are many but can usually be traced to a traumatic event, intra-articular/ligament knee surgery, knee arthroplasty, and possibly sepsis. Prevention is the best form of treatment, but when this entity does present, early recognition and a supervised physical therapy program are often successful. If conservative treatment fails, operative intervention is warranted. The purpose of this article is to discuss how to appropriately evaluate a patient with loss of knee motion, understand the pathogenesis and classification of arthrofibrosis, review our operative technique and postoperative management of these difficult patients, review the results in the literature concerning arthrofibrosis, and make the reader aware of possible concerns and the future direction of treatment of patients with arthrofibrosis.

Keywords: arthrofibrosis, knee, motion loss, stiffness, lysis of adhesions, arthroscopy

INTRODUCTION

Loss of motion of the knee joint can be a distressing occurrence in an individual's life. The cause of arthrofibrosis can usually be traced to a traumatic event (intra-articular fracture, injury to extensor mechanism), intra-articular/ligament knee surgery, knee arthroplasty, and possibly sepsis. The average genu recurvatum is 5° for males and 6° for females, and the average flexion is 140° for males and 143° for females.[1] Prior studies incorporating gait analysis have shown that 67° of knee flexion is required in the swing phase of walking, 83° to ascend stairs, 90° to descend stairs, and 93° to rise from a standard chair.[2] The loss of knee flexion is usually better tolerated than the loss of knee extension. Small discrepancies in extension can result in increased energy consumption during gait and cause undue strain on the quadriceps musculature and patellofemoral joint.[3] The purpose of this article is to discuss how to appropriately evaluate a patient with loss of knee motion, understand the pathogenesis and classification of arthrofibrosis, review our operative technique and postoperative management of these difficult patients, review the results in the literature concerning arthrofibrosis, and make the reader aware of possible concerns and the future direction of treatment of patients with arthrofibrosis.

Address correspondence and reprint requests to Bernard R. Bach, Jr., MD, Division of Sports Medicine, Midwest Orthopaedics at Rush, Rush University Medical Center, 1725 W. Harrison St., Suite 1063 Chicago, IL 60612, U.S.A. E-mail: brbachmd@comcast.net

HISTORICAL PERSPECTIVE

Traditionally, arthrofibrosis has been treated with open quadricepsplasty.[4–6] Thompsons' 12 cases of loss of knee motion were related to femur fractures caused by blunt or penetrating trauma. He obtained 10 satisfactory results by performing open excision of scars in vastus intermedius musculature and immediately instituting passive and active motion after the surgery.[4] Nicoll contributed the cause of motion loss to fibrosis of the vastus intermedius at and just proximal to the suprapatellar pouch, intracapsular adhesions between the patella and the femoral condyles, fibrosis of the lateral vasteralis to the lateral condyle, and/or shortening of the rectus femoris.[5] Daoud and colleagues modified the original technique of open quadricepsplasty.[6] They realized the importance of just performing open releases and not sectioning the vastus musculature, which often resulted in extension lags and weak musculature. The first use of arthroscopic management was initiated by Jackson for motion loss related to arthritis.[7] The first arthroscopic series included 24 patients who developed motion restriction after open

operative procedures and was reported by Sprague and associates.[8] Today, it is quite common to perform arthroscopic lysis of adhesions after failure of appropriate nonoperative and rehabilitative management. If the motion gained by arthroscopic releases and manipulation is not adequate, appropriate open releases are then performed.

INDICATIONS, CONTRAINDICATIONS, PATHOGENESIS, AND CLASSIFICATION

The etiology for the development of loss of knee motion is multifactorial. Its presentation may be localized and present with loss of extension or as a generalized fibrosis or capsulitis presenting with severe stiffness. There are preoperative, intraoperative, and postoperative factors that may contribute to arthrofibrosis. Risk factors appear to include operating in the acute injury period, proximal medial collateral ligament (MCL) injury, poor preoperative motion, improper graft placement and surgical technique, and possibly host factors.[9–12] The most common presentation of arthrofibrosis is a patient who has undergone anterior cruciate or multiple-ligament reconstructions. The incidence of knee stiffness after ligament reconstruction has been reported anywhere from 4% to 35%.[13] Shapiro and Freedman[14] noted a 57% incidence of knee motion loss in those patients undergoing combined anterior cruciate ligament (ACL) and posterior cruciate ligament (PCL) reconstructions. The development of an exaggerated scar response is mediated by growth factors and the inflammatory cascade.

A ''cyclops lesion'' is a localized intra-articular fibrous nodule that causes loss of knee extension. It is most often encountered following ACL reconstruction, particularly with anterior placement of the bone tunnel on the tibial side. Histopathology of this lesion early on displays a central area of granulation tissue with occasional osseous or cartilaginous tissue within the lesion (Fig. 1).[15] As the lesion matures, it has the appearance of disorganized fibrous connective tissue with a component of chondroid metaplasia, without granulation tissue and limited vascular ingrowth.[16]

Shelbourne and colleagues[17] described a classification system based on the pattern of knee stiffness. Type I patients have normal flexion and $<10°$ extension loss. Type II patients have normal flexion and $>10°$ extension loss. Type III patients have a combined flexion loss of $>25°$ and extension loss of $>10°$ with patellar tightness. Type IV patients have an extension loss of $>10°$ and total flexion of $<120°$, combined with patella infera contracture syndrome. If motion loss develops, most often nonoperative management is initiated with the use of nonsteroidal anti-inflammatory drugs, intra-articular steroid injections, Medrol dose pack, and institution of a supervised therapy program. Even if the stiffness is caused by an infection, arthroscopic irrigation and debridement can be performed for potentially definitive treatment. Starting appropriate intravenous antibiotics and consultation with an infectious disease specialist will aid in management. The only true contraindication other than patient consent, motivation, and health-related factors is complex regional pain syndrome (CRPS). Operating on a patient's stiffness with CRPS may make this entity worse, especially without appropriate treatment of CRPS.

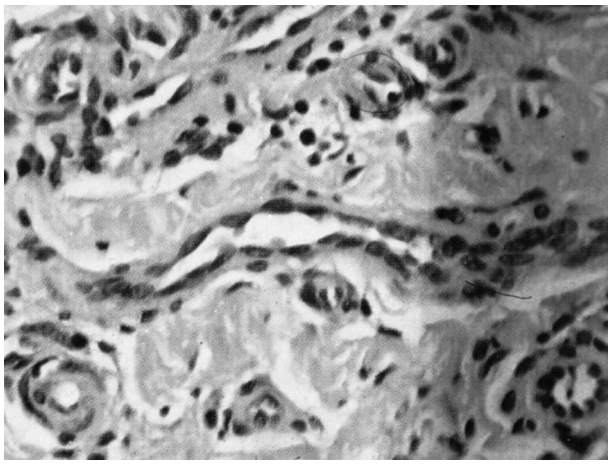

FIGURE 1. An hematoxylin and eosin histologic example of scar noted at the time of resection. Characteristic microvascular ingrowth and fibrous connective tissue is noted. Chondroid metaplasia and osseous islands may also be observed.

PREOPERATIVE PLANNING

A thorough history and physical examination are paramount in evaluating a patient with motion loss. The goal is to elicit the cause of the motion loss. Limitations to extension are generally secondary to pathology in the intercondylar notch region. Limitations of knee flexion are generally secondary to scar development within the medial and/or lateral gutters or within the suprapatellar pouch region.[18] It is also important if the patient has had previous operative intervention to obtain the operative reports. This will potentially aid in understanding the cause of arthrofibrosis (ie, timing of surgery, multiple-ligament reconstruction), associated pathology (cartilage lesions, MCL injury), and technical considerations that a routine patient may not completely understand and convey to the physician. In a patient who has significant pain, consider infection and/or CRPS as a contributing cause of knee stiffness. Diagnostic imaging is required and helpful in evaluation of these patients. Our standard protocol begins with standard weight-bearing anteroposterior,

merchant, long-leg alignment, and bilateral 30° lateral views to assess for patella baja. Magnetic resonance imaging may provide extra information in difficult cases. It is also important to discuss realistic expectations of nonoperative and operative treatment with the patient. This will aid in the patient's understanding of this process, which will help through the difficult rehabilitation that follows surgical intervention.

TECHNIQUE

At our institution, surgical debridement is typically performed as an outpatient procedure and under general anesthesia. After appropriate anesthesia, the operative table is reflexed, the operative leg is left hanging without a leg holder, and the nonoperative limb is placed in a leg holder (Fig. 2). We do not use a leg holder on the operative leg because this will impede the instruments working in the suprapatellar pouch and the ability to perform a manipulation. A nonsterile tourniquet is applied on the operative thigh, the extremity is prepped and draped in a standard fashion, and the leg is exsanguinated and the tourniquet inflated.

Six arthroscopy portals may be used to perform this procedure (Fig. 3). The superomedial portal is an outflow portal, the superolateral portal is the initial working portal, the inferolateral portal is the initial arthroscopy portal, and the inferomedial, posteromedial, and posterolateral portals are used as working portals. Despite the traditional roles of the above portals, any can be used as arthroscopy, outflow, or working portals (Table 1).

The excision of adhesive bands and scar tissue is performed in a systemic fashion starting in the suprapatellar pouch. If the pouch is severely scarred, normal landmarks may be difficult to appreciate. Capsular distension with a 60-mL syringe and an 18-gauge needle, in this instance, may be of benefit to allow easier and safer insertion of the arthroscope and outflow portal.[19] One should first establish the superomedial outflow portal, usually with a sharp trocar. Place the scope through the standard inferolateral portal and direct it into the suprapatellar pouch. If the arthroscope cannot easily be placed into the intercondylar notch and transitioned atraumatically into the suprapatellar pouch, the arthroscope is removed and placed in the suprapatellar pouch via a superolateral portal. The superolateral working portal is next established. It usually is necessary to use a combination of arthroscopic scissors, basket instruments, and electrothermal devices to create edges of the adhered scar tissue before a large shaver can be used effectively (Fig. 4).

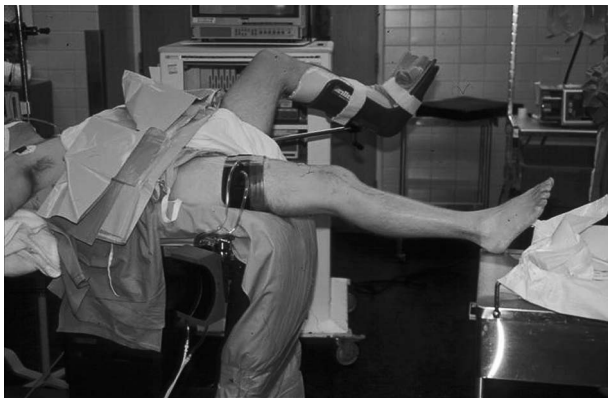

FIGURE 2. The patient is positioned so that the knee can be accessed from the surprapatellar portals. Usage of a leg holder might preclude this is placed too distally on the thigh. We prefer use of a thigh post. The opposite extremity should be carefully positioned to protect the peroneal nerve. The waist and foot of the table are flexed. Flexing the waist of the table takes the lumbar spine out of extension.

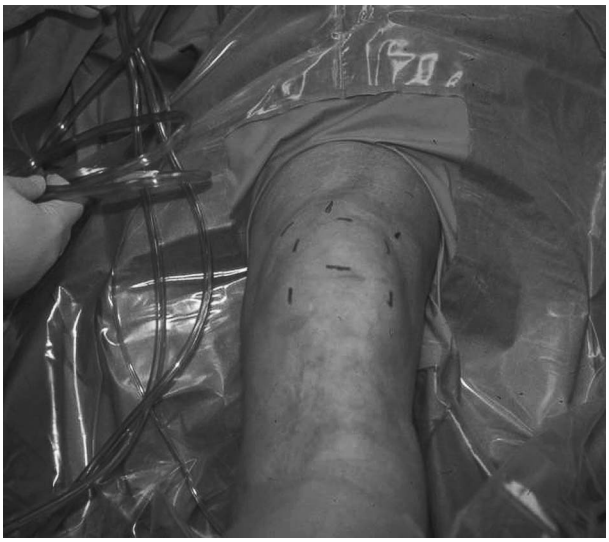

FIGURE 3. A standard superomedial portal is used for pump outflow. Initially the arthroscope is inserted through an inferolateral portal created with the knee flexed an dusing the distal patellar pole as a reference point. If difficulty is encountered easily advancing the arthroscope, one should transition to a superolateral portal for placement of the arthroscope. Initially the inferomedial portal is used as a working portal.

TABLE 1. *Portal usage in arthroscopy technique*

Arthroscopy portal	*Working portal*	*Compartment*
1. Inferolateral	Superolateral	Suprapatellar pouch, lateral gutter
Superolateral*	Superomedial	Suprapatellar pouch
2. Superolateral	Inferolateral	Lateral gutter
3. Inferolateral	Inferomedial	Medial gutter, intercondylar notch
4. Inferolateral	Posteromedial	Posterior recess
5. Inferomedial	Posterolateral	Posterior recess

*If difficulty in placing arthroscope in the inferolateral portal is encountered, establish a superolateral portal instead.

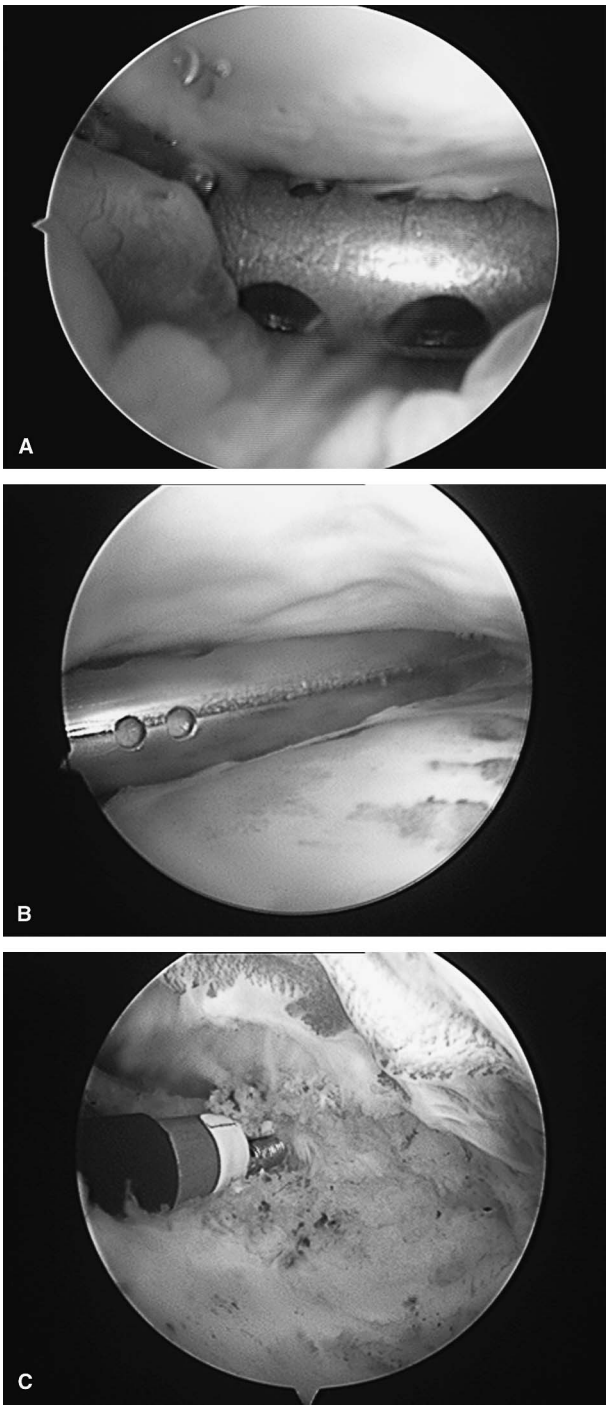

FIGURE 4. A, A superomedial portal is established. It is critical to confirm intraarticular placement of the outflow cannula. Note how contracted the suprapatellar pouch appears. B, Some adhesions may be manually lysed with the outflow cannula. C, Insertion of an electrothermal device, arthroscopic electrocautery, or placement of arthroscopic hand instruments may be used to release the suprapatellar pouch and reconstitute the ''pouch''.

These instruments are used until the scar tissue in the suprapatellar pouch is removed and patellar mobility is reconstituted.

The arthroscope is switched to the superolateral portal, working through the inferolateral portal to free up the lateral gutter. Again, alternating between baskets/scissors and shaver is usually necessary. At this point, establish an inferomedial portal. We prefer to do this under direct visualization with the use of a spinal needle. A shaver is placed into the inferomedial portal, and we continue cleaning out the lateral gutter and then begin debridement of the medial gutter.

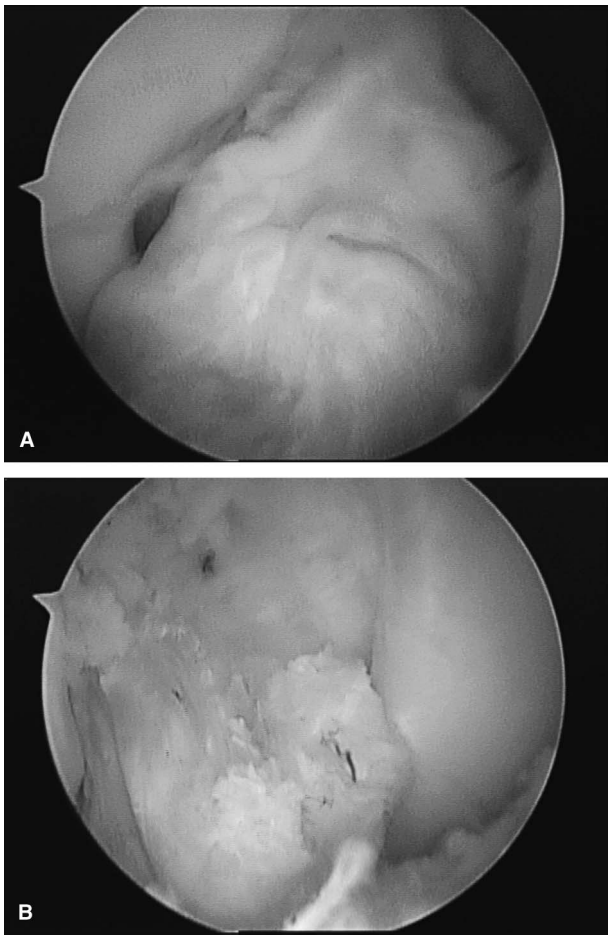

FIGURE 5. A, Scar tissue is noted at the insertion region of the ACL tibial footprint. This may be variable in size and occasionally emanate from the intercondylar apex. Clinically patients may experience a loud painless ''clunk''. Dependent upon the extent of knee flexion contracture, excision of the ''cyclop's nodule'' may result in a significant improvement in terminal knee extension and resolution of the ''clunk''. B, Scar tissue excised from within the intercondylar notch should be visualized from the standard inferolateral and inferomedial portal. It is surprising how one may not appreciate the degree of scarred fat pad anterolaterally when only visualizing from the inferolateral portal.

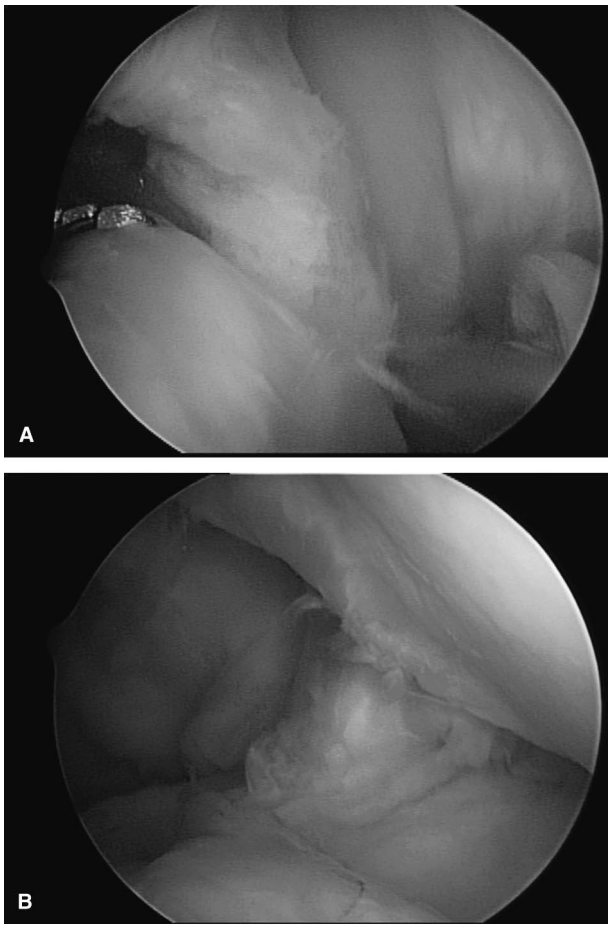

FIGURE 6. A, A sheet of scar tissue is demonstrated. A sheet or cord of tissue may extend from the non articular portion of the distal patella to the anterior tibia. B, Arthroscopic photograph following resection of this sheet of scar tissue.

The arthroscope is switched back to the inferolateral portal, and debridement of the medial gutter and the intercondylar notch is completed. The instruments are placed through the inferomedial portal to perform this part of the procedure. At this time, a ''cyclops lesion'' is easily evaluated at the tibial insertion of the ACL. As the knee is brought into extension, this scar nodule may be noted to slide into the patellotrochlear interval or anterolateral joint region. This nodule may present with an audible ''clunk'' in terminal extension and can be easily differentiated from patellofemoral crepitation (Fig. 5). Appropriate excision is performed, paying attention not to debride the intermeniscal ligament. It is important to release and mobilize the infrapatellar fat pad from the anterior tibia. This re-establishes the pretibial recess, thus decreasing patellofemoral joint contact pressures (Fig. 6).[20] The intercondylar notch should be visualized from both the inferolateral and the inferomedial portals to make sure there is no more scar tissue tethering the patella. Frequently, there is a band that extends from the nonarticular portion of the patella distally toward the tibial plateau. Expansion notchplasty is performed at this time with a combination of an osteotome, arthroscopic shaver, and burr. At this point, evaluate the motion improvement and perform a manipulation (Fig. 7). If the motion is still not adequate, we establish posteromedial and posterolateral portals.

The posterior portals are established using an outside–in technique under direct visualization. For the posteromedial portal, place the arthroscope in the inferolateral portal and position the arthroscope medial to the ACL while internally rotating the tibia. Once in the posterior space of the knee, looking medially, palpate the posteromedial soft spot and direct a spinal needle posterior to the medial epicondyle in a slightly distal and lateral direction. Once the appropriate angle is determined, a disposable arthroscopic cannula is placed into the joint. For

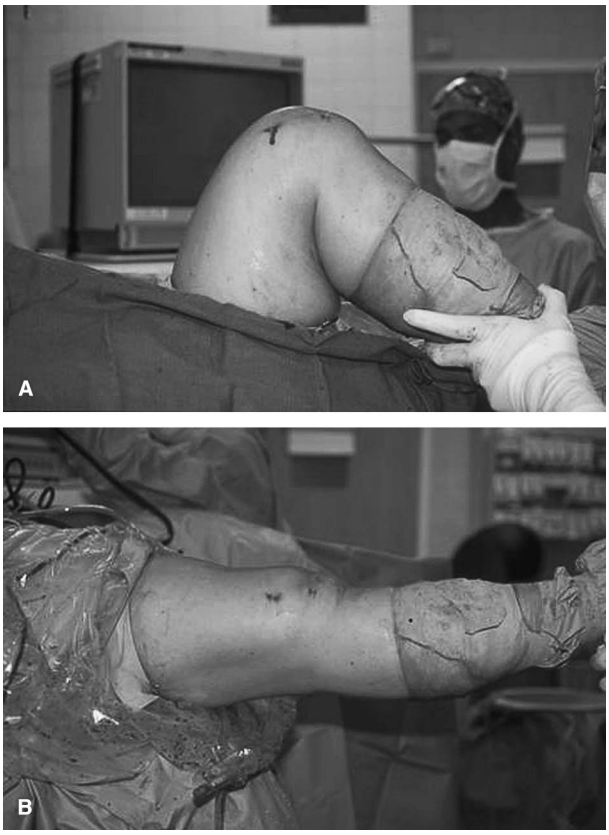

FIGURE 7. A, Following release of the suprapatellar pounch and medial and lateral capsular releases the knee is flexed to determine adequacy of flexion recovery. We document the range of motion established with passive unassisted as well as assisted flexion and document this in the operative report. B, Documentation of extension recovery is assessed. One should assess the degree of patellar mobility. Scar tissue within the intercondylar notch is a primary contributor to loss of knee extension.

the posterolateral portal, the arthroscope is placed in the inferomedial portal; position the arthroscope lateral to the ACL while externally rotating the tibia. Once in the posterior space of the knee and looking laterally, direct a spinal needle just posterior to the lateral condyle, and angle 45° distally and medially. Once the angle is determined, a disposable arthroscopic cannula is placed into the joint. Appropriate excision of scar tissue and removal of any loose bodies are performed, paying particular attention not to injure the posterior neurovascular structures.

After this is accomplished, the posterior cannulas and arthroscope are removed. The knee motion is again assessed, and a repeat manipulation is performed if necessary. If the motion is still not adequate, an open release will be necessary. We follow the anterior extensile approach as described by Millett and associates.[21] A medial parapatellar arthrotomy is performed, and an extensive medial release is performed as in a standard total joint arthroplasty. The periosteum is elevated to include the deep portion of the MCL and the semimembranosus insertion. This maneuver aids in regaining knee extension and mobilizing the tibia. The medial and lateral gutters are evaluated, and any extra-articular or residual intra-articular adhesions are dissected. The infrapatellar fat pad is freed from the patellar tendon and completely removed. A lateral release is next performed to aid in everting the patella. The superior lateral geniculate vessel should be preserved if possible. Flexion and extension are again re-evaluated at this point.

The tourniquet is released at this time, and meticulous hemostasis is obtained. Suction drains are placed to decrease the occurrence of postoperative hemarthrosis, which can contribute to both pain and flexion contractures.[9] The drain is typically left in place for <24 hours. Arthroscopy portals are closed with interrupted 3–0 Prolene suture. If an open procedure was performed, the arthrotomy is left open, but the dermal and subcuticular tissues are tightly closed. A sterile dressing is applied with a cryotherapy device, and a rehabilitative brace is locked in full extension.

RESULTS

As in many reports in the literature, treatment of motion loss of the knee deals with patients of mixed pathologies and causes of knee stiffness. The literature will be analyzed based on the etiology of arthrofibrosis, including an isolated process (ie, cyclops lesion) and a global process (ACL/ligament surgery). The first series of arthroscopic release of adhesions was reported by Sprague and associates.[8] They reviewed 24 patients who had previous operative intervention and presented with severely restricted knee motion. The causes of the restricted knee motion were attributed to extensive intra-articular procedures, sepsis, prolonged immobilization, and poor rehabilitation. The interval between the index surgical procedure and their arthrofibrosis treatment was 33 months. They were able to improve flexion from 70° pre operation to 115° at a mean follow-up of 8 months.

Jackson and Schaefer[15] were the first to describe the ''cyclops lesion.'' They encountered this lesion in 13 of 230 (5%) patients who underwent an ACL reconstruction. These 13 patients presented with a loss of terminal extension and an audible and palpable clunk with attempted terminal extension. They were treated with arthroscopic debridement and knee manipulation. Their preoperative knee range of motion of 0°/16°/103° improved immediately postoperatively to 0°/6°/130° and at a final follow-up to 0°/4°/138°.

Marzo and colleagues[16] at the Hospital for Special Surgery noted an occurrence of a loss of knee motion and a fibrous nodule in 21 of 640 (3%) ACL reconstructions with either bone–patellar tendon–bone or hamstring autografts. All patients were treated with arthroscopic debridement of this nodule, and 10 patients required an additional notchplasty. The majority of the patients obtained a satisfactory outcome, with an average improvement 8° of extension and average extension loss of 3°.

Fisher and Shelbourne[22] treated 42 of 959 (4%) ACL reconstructions for terminal extension loss. The patients were treated with arthroscopic debridement, and their preoperative motion of 0°/6°/119° improved to 2°/0°/135° postoperatively at an average follow-up of 28 months. A cyclops lesion usually becomes apparent in the early postoperative time period. However, Nuccion and Hame[23] reported on a patient who had full recovery after an ACL reconstruction but gradually began to develop loss of extension and pain 4 years postoperatively. The etiology of a cyclops lesion appears to be a multifactorial in origin and may include drilling debris of the tibial tunnel, remnants of the native ACL, and a hypertrophied graft caused by impingement.[24]

A patient's loss of motion may not only be related to a localized scar response but may be caused by a more global adhesion response throughout the joint. Harner and associates[25] reported on 27 patients who developed motion deficits out of 244 (11%) undergoing ACL reconstruction. Twenty-one of the 27 patients were available for follow-up and were compared with a group of patients with ACL reconstructions who did not develop a motion loss. Of the 21 patients, 14 underwent arthroscopic debridement, 6 underwent an open procedure, and 1 underwent a closed manipulation. The preoperative range of motion of 0°/13°/124° improved to 0°/3°/126° in the arthrofibrosis group. They had 67% good/excellent results

compared with 80% good/excellent results in the control group without motion loss.

Shelbourne and Johnson[26] reported on nine patients with a mean follow-up of 31 months who underwent arthroscopic treatment and knee manipulation of arthrofibrosis in an outpatient setting. Eight of nine of these patients underwent an ACL reconstruction within 2 weeks of their injury. Postoperatively, the patients underwent daily extension cast changes. Preoperative range of motion of 0°/23°/112° improved to 2°/0°/130°, and eight of nine patients were able to return to recreational athletics.

Hasan and colleagues[27] at Rush University Medical Center found 17 of 342 (4%) ACL reconstructions to have a symptomatic extension loss of >5°. Thirteen were available for a mean follow-up of 3.9 years, and they were compared with a matched control group of 26 patients who underwent an ACL reconstruction and did not experience an extension loss. The surgery was performed at an average of 12 months from the ACL reconstruction and included arthroscopic debridement of generalized adhesions, excision of a cyclops lesion in 8 patients, and revision notchplasty in 11 patients. All patients were manipulated after debridement, first in flexion and then extension. Preoperative range of motion improved from 0°/10°/123° to 0°/3°/131° postoperatively. Despite a small lingering extension deficit, there were no statistically significant differences in functional rating scales between the arthrofibrosis and control groups at follow-up.

Cosgarea and associates[28] reported on 37 patients who underwent surgical intervention for arthrofibrosis after knee ligament surgery. The authors described a three-stage algorithm. Stage I included arthroscopic management, stage II involved an anterior arthrotomy and extra-articular scar excision, and stage III incorporated a posteromedial arthrotomy. Thirty of 37 patients were treated with arthroscopic intervention, which included debridement, percutaneous lysis of adhesions, a lateral retinacular release, and notchplasty. Seven patients required an anterior arthrotomy, and only one patient required a posteromedial arthrotomy. At an average follow-up of 3.6 years, preoperative motion of 0°/14°/120° improved to 0°/3°/142°. Despite this motion improvement, only 62% had a satisfactory outcome compared with 80% of the control population. Also, radiographic degenerative joint changes were found in 89% of the motion loss group, with 9% having patella infera.

Paulos and colleagues[29] reported on 28 patients with infrapatellar contracture syndrome, a proliferative fibrous hyperplasia of the anterior soft tissue caused by ligament surgery/trauma or prolonged immobilization. The preoperative motion of 0°/17°/98° improved to 0°/5°/133° postoperatively. Despite the motion improvement, no athletes were able to return to their preinjury level of participation, and 90% developed patellofemoral arthrosis. The authors concluded that open debridement is necessary to address the extra-articular component often present in this entity. Also, drop-out casts and forced extension manipulations may contribute to the development of patellofemoral arthrosis.

Paulos and colleagues[30] reported a follow-up study of infrapatellar contracture syndrome that included 75 patients. They performed a DeLee tibial tubercle osteotomy in patients with patella infera of ≥8 mm. The preoperative motion of 0°/16°/96° improved to 0°/2°/121° at final follow-up. In this study, 70% of patients demonstrated radiographic changes of the patellofemoral joint at follow-up. The motion improvement was predictable, but the functional outcome was only fair. The longer the knee was confined in motion, the poorer the result.

Millett and associates[31] reported on eight patients who underwent open debridement and soft tissue release after failure of previous surgical release. The preoperative range of motion of 0°/19°/81° improved postoperatively to 0°/1°/125° at a mean follow-up of 4.8 years. These patients had extensive postoperative management including regional epidural, intravenous corticosteroids for 48 hours postoperatively, continuous passive motion machine 6 hours a day, and custom drop-lock extension braces worn at night. All of the patients reported subjective improvement, but only one was able to return to preinjury level.

SPECIAL CONSIDERATION: STIFF TOTAL KNEE ARTHROPLASTY

The stiff total knee arthroplasty (TKA) is another case scenario that may require operative intervention. This entity is different in that it is usually done for flexion loss rather than extension loss. Total knee replacements require 90° of flexion to function satisfactorily. Paying attention to preoperative motion is the most important factor in determining the patient's postoperative flexion.[32] In stiffness related to arthroplasty, component position and placement can also affect the final range of motion. Factors contributing to loss of knee flexion include an elevated joint line, a flexion gap, and failure to match the tibial slope.[33] The results of treatment of stiffness encountered after TKA are more difficult to interpret. The physician needs to consider preoperative patient factors, component position and ligament balancing, and the onset of the development of stiffness. If stiffness is encountered within the first 3 months and the patient does not have an infection and has appropriately fixed and positioned components, an aggressive physical therapy and home exercise program are recommended.[34,35]

If the physical therapy is not successful or the loss of motion develops after 3 months, manipulation under

anesthesia is often the next step. Fox and Poss[36] evaluated 76 knees in 76 patients who had undergone TKA and experienced a flexion loss. They were able to gain 37° of flexion immediately; however, this dropped to 17° 1 week after manipulation, and this gain in flexion was negligible at 1 year after the manipulation. Daluga and associates[37] reported on 94 knees in 60 patients who gained 35° of flexion after manipulation. These patients were able to maintain this flexion gain at a 24-month mean follow-up. They noticed that if the manipulation was performed >3 months after TKA, the improvement in flexion was less by an average of 7°. Esler and colleagues[38] evaluated 47 knees in 42 patients who had undergone a TKA, and their mean time to manipulation was 11.3 weeks. The patients had an immediate gain of flexion of 34°, which was 33° at 1-year follow-up.

If manipulation alone is not adequate or if there is a significant extension loss, the addition of arthroscopic debridement is the next step of treatment. Campbell[39] treated eight knees in eight patients with arthroscopic lysis of adhesions at 11.6 months from their TKA. At a mean follow-up of 1 year, there was only an 11° gain in flexion and a 5.5° gain in extension. Diduch and associates[40] reported on eight knees in eight patients treated with arthroscopic debridement and manipulation at 7.4 months from TKA. They were able to improve motion by 26° at a mean follow-up of 20 months. If the patient received a PCL-retaining prosthesis and had motion loss, sacrifice of the PCL might be beneficial. Williams and colleagues[41] treated 10 knees in nine patients with arthroscopic PCL sacrifice for motion loss a mean of 29 months from TKA. They were able to improve flexion by 30° and extension by 2.5° at a mean follow-up of 20 months.

If motion loss is determined to be caused by malpositioned or loose components, this is best handled by an arthroplasty specialist. Nicholls and Dorr[42] reported on 13 knees in 12 patients with faulty components and improved their motion an average of 33° with revision TKA. Therefore, for evaluation of a stiff TKA, the orthopedist must take into account the previous factors that contribute to a stiff knee, but pay particular attention to the patient's preoperative knee range of motion and be able to evaluate component position.

TABLE 2. *Pearls and Pitfalls of Knee Stiffness*

1. Avoid acute surgical reconstruction.
2. Recover complete/nearly complete knee motion prior to surgery.
3. Avoid reconstruction until return of quadriceps strength.
4. Exclude displaced meniscus as a cause for preoperative knee flexion contracture.
5. Pain referred to the retropatellar tendon area on forced knee extension is usually from an incarcerated ACL stump.
6. Beware of concomitant MCL injury. Proximal MCL injuries result in slower motion recovery.
7. Improper tunnel placement is a common technical error contributing to motion loss and/or graft failure.
8. Tension and fix the ACL graft in complete extension. Avoid placing a posterior Lachman stress that may overconstrain the graft.
9. If the patient has a locked knee secondary to a displaced meniscus, consider performing a staged meniscal repair and reconstruction, particularly if the patient has a low pain threshold.
10. When performing a meniscal repair, tie the sutures in complete knee extension to avoid capturing capsular tissue.
11. Splint (drop-lock extension brace) the knee in full extension.
12. Consider night extension splinting in the early postoperative course.
13. After reconstruction, if the patient does not have complete extension at 10 days postoperatively, use an extension board and monitor the patient closely.
14. If a patient has not achieved motion goals by 10–14 days postoperatively, monitor weekly.
15. At 6 weeks postoperatively, consider a Medrol dose pack for the patient who is progressing slowly.
16. In the early postoperative phase, aspirate any significant effusion that may contribute to pain, motion loss, or quadriceps shutdown.
17. Consider early surgical debridement if the patient is not progressing and demonstrates a knee flexion contracture.
18. Always obtain preoperative radiographs prior to performing surgery for knee stiffness.
19. Educate your patient preoperatively and postoperatively.
20. In a patient who has significant pain, consider an infection and/or complex regional pain syndrome as a contributing cause of knee stiffness.

COMPLICATIONS

The most worrisome complication that stems from arthrofibrosis is the development of early degenerative joint disease, particularly of the patellofemoral joint. This is more pronounced with the infrapatellar contracture patient population. The scar-contracted knee causes increased joint contact pressures. Also, in a contracted knee, initial placement of arthroscopic instruments can be difficult, possibly causing iatrogenic injury to the cartilage. The development of a postoperative hemarthrosis is a possibility. The hemarthrosis can be painful and act as a block inhibiting the recovery of the knee motion gained at surgery. Meticulous evaluation and coagulation of any bleeding vessels are performed after releasing the tourniquet. The use of a compressive dressing, cryotherapy, and a suction drain may aid in the prevention of a hemarthrosis. However, if extensive releases have been performed, a hemarthrosis will not typically develop secondary to soft tissue extravasation. Another common

scenario is the inability to retain the motion gain after operative intervention. Other potential complications include infection, fracture, extensor mechanism injury, neurovascular injury, and wound dehiscence.

POSTOPERATIVE MANAGEMENT

Postoperative rehabilitation begins in the recovery room, displaying the motion gain to the patient and family while the patient's pain is still controlled. Physical therapy is initiated the same day of surgery, reinforcing the motion gain to the patient. This protocol emphasizes the maintenance of knee extension using prone heel hangs and isometric quadriceps-strengthening exercises. Patellar mobilization and knee sags working on knee flexion are also stressed. If the loss of motion was mild, a knee immobilizer is used in full extension overnight. In moderate knee flexion contractures, extension board splinting is used, and in more severe cases, we employ a bivalved cylinder cast. Hospitalizations and continuous passive motion machines are expensive additions, and we routinely do not use them except for revision or refractory cases.

POSSIBLE CONCERNS AND FUTURE OF THE TECHNIQUE

The best treatment of arthrofibrosis of the knee is prevention. Before any surgical procedure is performed around the knee, the patient should be educated about this potential complication. Paying meticulous attention to the timing of surgery, associated pathology, surgical technique, postoperative rehabilitation, and possibly host factors are musts to avoid this problematic complication (Table 2). There is research being done to identify at-risk patients. Skutek and colleagues[43] believe there to be an association between the development of arthrofibrosis and specific allelic groups of the human leukocyte antigen. Despite taking these measures, stiffness of the knee may still arise. A patient who is informed and motivated will aid in the appropriate diagnosis and treatment protocol for knee stiffness.

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