Technical Note

Arthroscopic Treatment of the Arthrofibrotic Knee

David H. Kim, M.D., Thomas J. Gill, M.D., and Peter J. Millett, M.D., M.Sc.

Abstract: The management of motion loss of the knee is challenging. A clear understanding of the pathoanatomic causes of motion loss is necessary to establish a careful and rational approach to treatment. Early recognition and physical therapy are effective for the majority of patients, but when these conservative measures fail, operative intervention is indicated. The purpose of this article is to outline a comprehensive approach to the arthroscopic evaluation and treatment of the arthrofibrotic knee. This technique is designed to allow the surgeon to systematically address the numerous causes of motion loss of the knee. **Key Words:** Knee—Arthrofibrosis—Arthroscopy—Stiffness—Motion loss—Lysis of adhesions.

Motion loss of the knee can result from a variety of causes and poses a difficult clinical problem. Early recognition and appropriate treatment can be expected to restore motion and improve function in the majority of patients. An understanding of the pathoanatomic causes of motion loss is necessary to provide successful treatment strategies. Whenever possible, it is important to identify the specific cause and target treatment accordingly.

Nonoperative measures such as rest, ice, anti-inflammatory agents, and subsequent physical therapy are sometimes unsuccessful. In such settings, operative intervention is indicated. Manipulation under anesthesia has been used in the postoperative period to address motion loss with some success.[1,2] However, manual manipulation has significant risk, including complications such as distal femoral fracture and patellar tendon rupture, but this can be prevented by a controlled surgical release of scar tissue as opposed to a gross manual manipulation under anesthesia. The arthroscopic approach is a powerful and controlled method that is effective both for focal, discrete lesions as well as for more global arthrofibrosis. The purpose of this article is to outline our systematic approach to the arthroscopic surgical management of the arthrofibrotic knee.

GENERAL PRINCIPLES

Fibrosis and contractures in different parts of the knee contribute to different types of motion loss. Adhesions in the suprapatellar pouch typically limit patellar mobility and can restrict knee flexion. The proximal extent of the pouch should be approximately 3.5 cm from the superior pole of the patella. A foreshortened pouch can lead to a further loss of knee flexion.

Other structures of the knee that contribute to a loss of flexion are the medial and lateral gutters, and the anterior interval. The anterior interval is the region of the knee posterior to the patellar fat pad and anterior to the anterosuperior tibial plateau. This interval is an underrecognized source of knee flexion loss.

The cruciate ligaments themselves can lead to restrictions in motion, particularly after cruciate ligament reconstruction.[3-8] An anteriorly placed femoral tunnel[9,10] or overtensioned graft[11,12] can limit knee

From the Department of Orthopaedic Surgery (D.H.K., P.J.M.), Brigham and Women's Hospital, Boston, Massachusetts; and the Department of Orthopaedic Surgery (D.H.K., T.J.G., P.J.M.), Massachusetts General Hospital, Boston, Massachusetts, U.S.A.

Address correspondence and reprint requests to Peter J. Millett, M.D., M.Sc., Brigham and Women's Hospital, 75 Francis Street, Boston, MA 02115, U.S.A. E-mail: pmillett@partners.org

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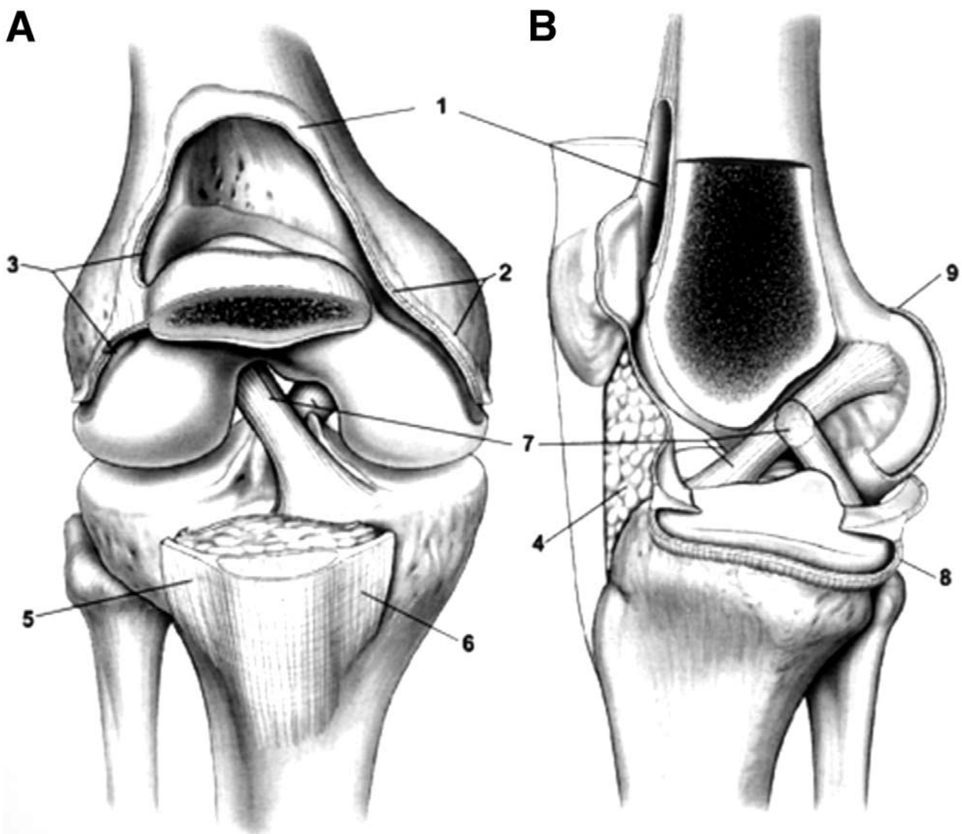

FIGURE 1. Coronal (A) and sagittal (B) illustrations of the knee. Schematic diagram of nine-step systematic arthroscopic evaluation: (1) evaluate and re-establish the suprapatellar pouch; (2) evaluate and re-establish the medial gutter; (3) evaluate and re-establish the lateral gutter; (4) mobilize and debride the infrapatellar fat pad to re-establish the pretibial recess; (5) evaluate the lateral retinaculum and perform lateral retinacular release, if necessary; (6) evaluate the medial retinaculum and perform medial retinacular release, if necessary; (7) evaluate the intracondylar notch, debride scar tissue, and in severe cases, release the anterior cruciate ligament/posterior cruciate ligament, if necessary; (8) evaluate the tibial insertion of posterior capsule, inspect the capsular recess, and perform medial and lateral capsulotomy, if necessary; and (9) evaluate the femoral insertion of the posterior capsule and release capsule, if necessary.

flexion. An anteriorly placed anterior cruciate ligament (ACL) graft on the tibia will impinge on the intercondylar notch and limit knee extension. This impingement could lead to the formation of fibrous ACL nodules (also known as "cyclops" lesions[4]), which further limit extension and can worsen impingement.[4,13-17]

A tight posterior knee capsule can limit knee extension. This area is difficult to approach arthroscopically and could be best addressed through an open approach if needed.

When surgery is undertaken, we perform a systematic nine-step evaluation[18] (Fig 1). First, adhesions in the suprapatellar pouch are identified and released. We seek to restore the normal suprapatellar pouch to allow excursion between the extensor mechanism and the underlying tissues. Next, the medial and lateral gutters are inspected and releases are performed as needed. The anterior interval should be evaluated dynamically as the knee is placed through flexion and extension. This interval between the patellar fat pad and anterior tibia is released to re-establish the pretibial recess. Next, we evaluate the lateral and medial retinacular structures and perform selective releases of any tight lateral and medial retinacular bands of tissue. A complete retinacular release is needed only in severe cases. The intercondylar notch is debrided of scar tissue, particularly fibrous ACL nodules, which lead to notch impingement. In overt cases of graft malposition, the ACL or even the posterior cruciate ligament (PCL) might need to be released. Knee motion is then assessed. If full extension cannot be achieved, the tibial insertion of the posterior capsule is inspected. If the posterior capsule is tight, medial, and occasionally lateral capsulotomies are performed. Finally, we evaluate the femoral insertion of the posterior capsule if full extension is not achieved and an open posteromedial release is performed if needed.

OPERATIVE TECHNIQUE

Anesthesia

We recommend the use of regional epidural anesthesia and the placement of an indwelling epidural

FIGURE 2. Capsular distention: Using a 60-mL syringe and an 18-gauge needle, a total of 180 mL of normal saline is introduced into the knee joint before the placement of the arthroscopic portals.

catheter for intraoperative and postoperative analgesia. This type of anesthesia provides improved localized pain control and allows more intensive physical therapy in the immediate postoperative period.

Capsular Distention

Capsular distension with 120 to 180 mL of saline before arthroscopy is a useful adjunct in the arthroscopic treatment of arthrofibrosis of the knee.[19] With the patient under anesthesia and after the extremity is prepped and draped, the knee joint is palpated. Often the scarring is so severe that it is difficult to make out the usual surface landmarks. Using an 18-gauge needle and 60-mL syringe, normal saline is injected into the suprapatellar pouch from the lateral side (Fig 2). It is important to carefully watch and feel for joint distention as the fluid is injected to ensure that the fluid is entering the true joint space. Fluid should flow easily if the needle is indeed in the true joint space. Normal knees easily accept 180 mL of saline, and an attempt to introduce this volume of fluid into the arthrofibrotic knee should be made. As the capsule is distended with fluid, care is taken to avoid rupturing the true capsule, although intra-articular adhesions could be disrupted. The last 60 mL of fluid is inserted slowly to allow the capsule to stretch over time. Preservation of the true joint capsule prevents extravasation of the fluid during arthroscopy and facilitates visualization.

Portal Placement/Visualization

After the knee is maximally distended with saline, we typically insert an inflow cannula into the knee through a superolateral portal and then initiate flow. This keeps the joint distended and facilitates insertion of the arthroscope through the standard inferolateral portal and also helps with initial visualization of the joint. In severe cases, it is often difficult simply to insert the arthroscope. A standard inferomedial working portal is then established. Although the camera sheath can be used to manually release adhesions, we prefer to use electrocautery to lyse adhesions. This approach will minimize bleeding, which not only helps with visualization, but also decreases the risk of recurrent postoperative scarring.

Systematic Examination

Suprapatellar Pouch: Using electrocautery, we lyse adhesions and release scarring to re-establish the suprapatellar pouch (Fig 3). It is important to remember that the pouch is quite large and should extend 3 to 4 cm proximal to the patella and releases should continue until this is achieved.

Medial and Lateral Gutters: It is important to look for adhesions in this region. Often one can see the adhesions that have formed between the capsule and the femoral condyles. A suction punch or electrocautery can be used to remove adhesions from the lateral gutter.

Anterior Interval: The infrapatellar fat pad and pretibial recess are inspected. We release and mobilize the infrapatellar fat pad from the anterior tibia and re-establish the pretibial recess (Fig 4). With a normal anterior interval, it is important to notice how the intermeniscal ligament glides freely on the anterior surface of the tibia. With anterior interval scarring, adhesions form in the pretibial recess. In experimental models,[20] these adhesions have been shown to increase patellofemoral joint contact pressures. In such instances, an anterior interval release should be performed by releasing the scar tissue from medial to lateral just anterior to the peripheral rim of the anterior horn of each meniscus. We prefer to use bipolar tissue ablation. The release also proceeds from the proximal extent at the level of the meniscus to approximately 1 cm distal along the anterior tibial cortex. Care should be taken to avoid cauterizing or burning the bone of the anterior tibia. Meticulous hemostasis should be maintained, especially around the infrapatellar fat pad.

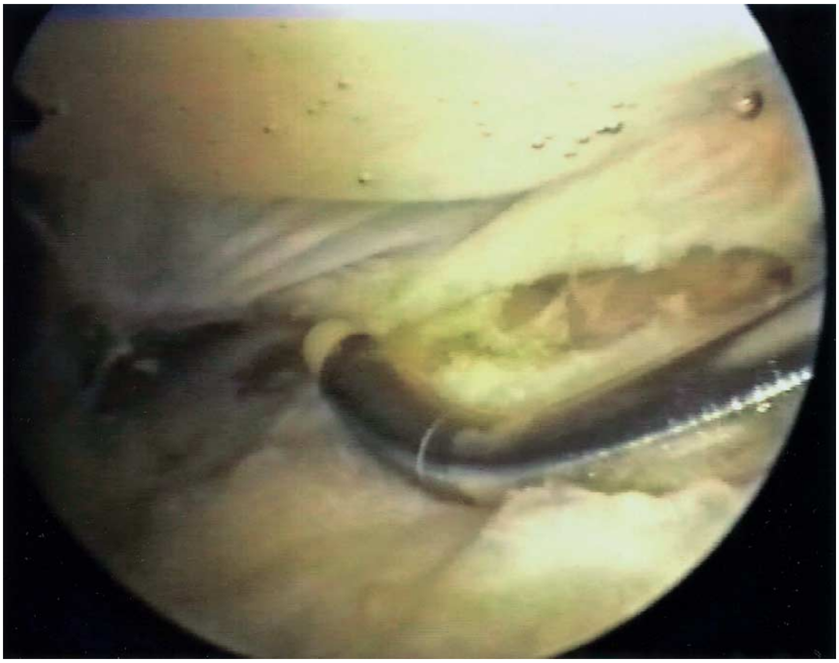

Figure 3. Re-establishment of the suprapatellar pouch: Adhesions and scarring are lysed using electrocautery to re-establish the suprapatellar pouch.

Lateral and Medial Retinaculum: Using electrocautery or a suction punch, we perform selective releases of lateral and medial retinacular structures if they are tight or scarred. This decompresses the patellofemoral joint and improves patellofemoral mobility. It also enhances visualization and permits easier instrumentation as the effective joint space is increased.

Intercondylar Notch: If there is evidence of graft impingement, a notchplasty should be performed. Fibroproliferative ACL nodules (Fig 5A) should be excised using a suction punch, because these nodules can limit extension by impinging on the intercondylar notch. If the cruciate grafts are malpositioned or the native ligaments are excessively scarred, they can be

Figure 4. Re-establishment of the anterior interval: The infrapatellar fat pad and pretibial recess are debrided using an electrocautery device.

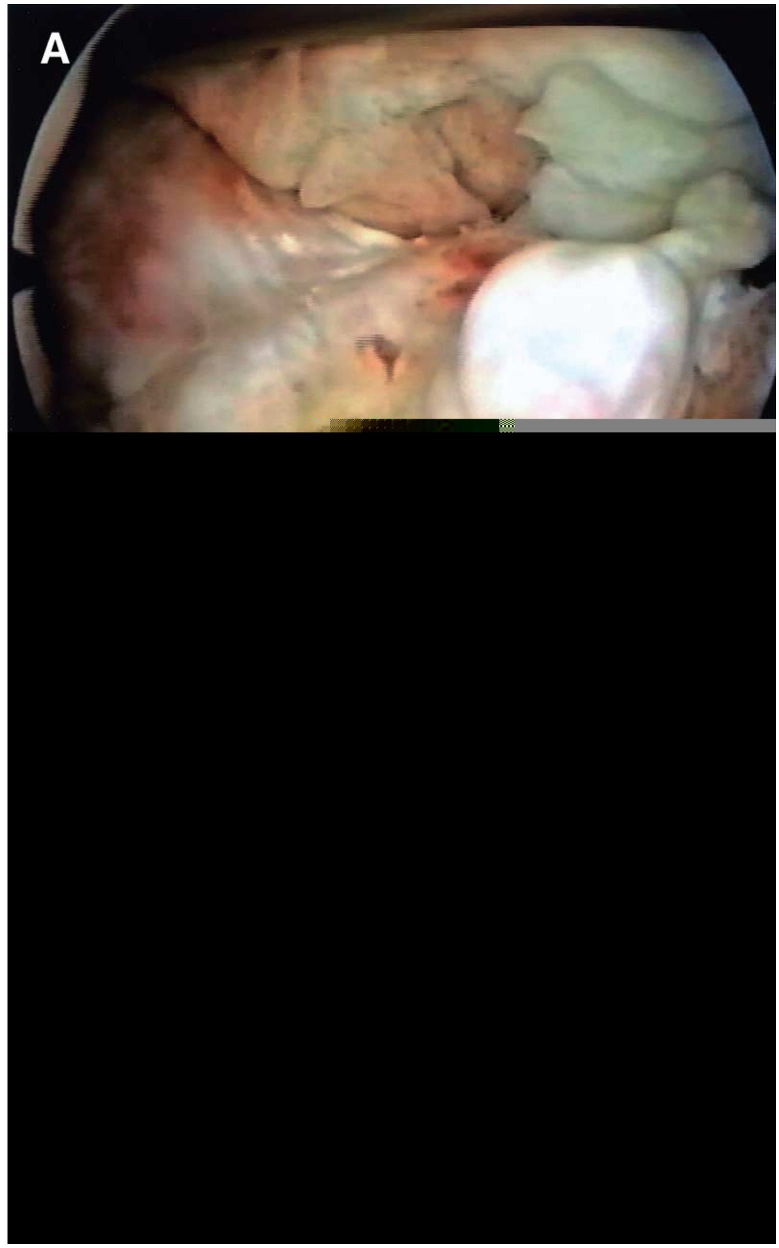

FIGURE 5. (A) Fibroproliferative anterior cruciate ligament nodule: Impingement can be caused by the cyclops lesion. (B) Bony nodule: An arthroscopic bur can be used to remove a bony nodule on the anterior cruciate ligament.

debrided, released, or excised altogether, according to the severity of involvement. Often patients will present with a bony nodule in the intercondylar notch. Such nodules or osteophytes can also impinge in the intercondylar notch and will block knee extension. A high-speed arthroscopic bur (Fig 5B) or an osteotome can be used to remove these bony nodules to prevent further impingement and to re-establish full extension. Generally, peripheral osteophytes are not debrided because this causes unnecessary bleeding and potentiates further adhesions.

Posterior Capsule: After completing the evaluation of the intercondylar notch, the knee should be placed through a range of motion, and flexion and extension should be carefully assessed. If there is persistent loss of extension, consideration should be made for an open capsular release. We prefer to perform this using a limited open approach as described by Steadman et al.[21] Such releases to the posterior medial and posterior lateral capsules should be added if the posterior capsule remains tight. In addition to a capsulotomy, a blunt periosteal elevator is used to strip the capsule from the tibial and femoral insertions. Generally, we begin on the medial side and release the capsule here. If a loss of extension persists, a similar open lateral capsular release is performed.

In our experience, postoperative knee motion will not improve from that obtained in surgery. Therefore, if a loss of extension or flexion persists intraoperatively, every attempt should be made to regain this motion before leaving the operating room.

Postoperative Protocol/Rehabilitation

Patients are typically hospitalized for 48 hours to take advantage of the analgesic properties provided by the indwelling epidural catheter. In the immediate postoperative period, patients are placed in a continuous passive motion (CPM) machine for at least 8 to 10 hours a day to maintain the range of motion obtained in the operating room. CPM is less effective than manual mobilization for the terminal ranges of motion. Home CPM is continued for 2 to 3 weeks. Dynamic bracing can be used to maintain full extension, although these devices are occasionally not well tolerated. Patellar mobility exercises, extensor mechanism exercises, and full passive and active-assisted range of motion exercises are all essential components of therapy, which begin on the first postoperative day.

Daily outpatient physical therapy visits start when patients are discharged and could continue for 6 to 8 weeks. Stationary bicycling could begin immediately, whereas aquatherapy and resistance exercises are begun at 2 weeks if range of motion is maintained and swelling is controlled.

DISCUSSION

The term arthrofibrosis is often used to describe loss of flexion, loss of extension, or both. More precisely, it refers to a specific process in which scar tissue or fibrous adhesions form diffusely within a joint.[22] Risk factors to developing arthrofibrosis of the knee include multiligament injuries and knee dislocations,[23-27] as well as prolonged immobilization, infection, and reflex sympathetic dystrophy.[28,29]

It is important to understand the myriad causes of motion loss so that treatment can be targeted at the specific cause. Causes of extension loss include notch impingement, ACL nodules, and posterior capsule pathology. Causes of flexion loss include suprapatellar adhesions, as well as medial and lateral gutter adhesions. Causes of both extension and flexion loss include improper graft position, infrapatellar contracture syndrome,[30-32] soft tissue calcifications, global arthrofibrosis, reflex sympathetic dystrophy, and infection. As a general rule, causes of loss of extension reside in the intercondylar notch and posterior capsule, whereas causes of loss of flexion reside in the suprapatellar pouch and the medial and lateral gutters.

The arthroscopic treatment of knee arthrofibrosis has been reported as being effective in improving knee range of motion and restoring function with minimal complications. Among the first to report their results were Sprague et al.,[33] who described a series of 24 patients with "fibroarthrosis" who gained an average of 45° of knee flexion with arthroscopic release of adhesions. Several authors have since reported similar results with knee range-of-motion improvements ranging from 45° to 68°.[29,33-42]

In addition, several reports have reviewed the results of arthroscopic treatment of arthrofibrosis, specifically after ACL reconstruction.[4,8,14,15,43] Jackson and Schaefer[4] first described the "cyclops" lesion after ACL reconstruction and reported on 13 patients who underwent arthroscopic debridement with knee manipulation and achieved an average improvement of loss of extension from 16° to 3.8°. Marzo et al.[14] reported on 21 patients who improved on an average loss of extension of 11° to an average of 0° at 1-year follow up after arthroscopic removal of fibrous nodules in the intercondylar notch. Subsequent studies[8,15,43] have all demonstrated marked improvements in function and symptoms with arthroscopic debridement of anterior scar formation in combination with aggressive rehabilitation and serial extension casting.

Arthroscopic treatment is generally most effective when the underlying pathology is predominately intra-articular. When the etiology of the motion loss is multifactorial or involves extra-articular structures, often a combined approach involving both arthroscopic and open techniques is indicated.[21,44-46] Results from this type of treatment are generally less satisfactory than with those reported from solely an arthroscopic approach, mainly because the underlying causes for motion loss are more extensive.

SUMMARY

Arthrofibrosis has a variety of causes and for optimal outcomes, appropriate treatment should be targeted at the specific cause. Our nine-step arthroscopic approach allows the surgeon to systematically address all of the intra-articular pathoanatomy. The first three steps are to evaluate and re-establish the suprapatellar pouch followed by the medial and lateral gutters.

Next, the anterior interval, infrapatellar fat pad, and pretibial recess should be re-established. The medial and lateral retinaculum are evaluated and releases performed if necessary. Electrocautery is essential to minimize bleeding. The intercondylar notch must be carefully evaluated, because this is often a major site of pathology. Scar tissue, ACL nodules, bony nodules, or malpositioned grafts can all cause motion problems. The final two steps involve evaluation of the medial and lateral posterior capsule at both the tibial and femoral insertions. For those patients with persistent loss of extension after comprehensive arthroscopic treatment, limited open releases should be undertaken.

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