Current Concepts

Motion Loss after Ligament Injuries to the Knee
Part II: Prevention and Treatment

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ABSTRACT
This is the second part of a two-part review on motion problems after ligament injuries to the knee. The first part, published in the September/October 2001 issue, discussed normal and abnormal knee motion, terminology, risk factors, and pathoanatomy. The purpose of this article is to review current concepts on prevention and treatment of motion problems, summarizing the recent and pertinent studies that discuss this complicated clinical problem. The first part of this article will discuss the different classification schemes that have been published on motion loss of the knee. Prevention strategies will be discussed next, followed by early recognition. Finally, a discussion of the various treatment options and published results will be presented in detail, together with the authors’ nine-step systematic surgical approach to the stiff knee.

CLASSIFICATION OF MOTION LOSS
Del Pizzo et al.,5 Blauth and Jaeger,2 and Shelbourne et al.24 have all outlined classification schemes for patients with motion loss. Del Pizzo et al. were the first authors to divide patients into groups according to preoperative range of motion and severity of fibrosis. They found a positive correlation between severity of motion loss and degree of fibrosis, using the system of pathoanatomic findings developed by Sprague et al.26 Blauth and Jaeger described a similar four-part classification scheme for patients with motion loss that ranged from mild to extreme, based on the range of motion of the affected knee.

The four-part classification scheme that Shelbourne et al.24 described for patients with arthrofibrosis is useful in that it provides both a descriptive and prognostic guide (Table 1). Seventy-two patients with arthrofibrosis of the knee after previous ACL reconstruction were treated surgically to remove scar tissue. In patients with the most severely affected knees (type 4), 16 had patella infera, greater than 30° of flexion loss, and greater than 10° of extension loss. Not surprisingly, patients with type 4 arthrofibrosis also had less predictable results: five patients failed to regain full extension and in one patient the treatment failed altogether. As a group, however, they still managed an average gain of 18° of extension and 42° of flexion.

These various classification schemes are useful in that they are both descriptive and prognostic. We believe it is essential for the clinician to identify the specific cause of the motion loss so that appropriate management can be initiated. Very often the cause is multifactorial and the surgeon must be prepared to address all problems at the time of surgery. Research studies are of great importance as they provide a basis to discuss possible outcomes with patients and give them a measure of risk or benefit of additional surgery.

PREVENTION
Prevention of motion loss is a key objective that can only come from a detailed understanding of the causes of the problem. Some general principles that we apply to the ACL-injured knee include 1) avoiding ACL reconstruction when the knee lacks full motion, remains swollen, or does not permit a normal gait and 2) waiting until full motion is reestablished before contemplating any surgical recon-
motion and allowing the ligament to heal. This avoids protecting the MCL injury, while regaining necessitating MCL reconstruction as well? Currently, we be delayed to prevent motion problems while potentially the ACL be fixed acutely to promote a stable bed for MCL eral ligament) injury presents a unique dilemma. Should The patient with the combined ACL-MCL (medial collateral ligament) presents a unique dilemma. Should such knees have a higher risk for motion loss. The patient with the combined ACL-MCL (medial collateral ligament) injury presents a unique dilemma. Should the ACL be fixed acutely to promote a stable bed for MCL healing, while risking motion problems, or should surgery be delayed to prevent motion problems while potentially necessitating MCL reconstruction as well? Currently, we recommend protecting the MCL injury, while regaining motion and allowing the ligament to heal. This avoids overstressing an ACL reconstruction and increases the chance for a stable knee. A delay in surgery allows the physician time to assess the degree of MCL healing and the overall knee stability. The MCL generally heals sufficiently so that no surgery is necessary to address it; however, if significant laxity persists with excessive external rotation, an MCL repair or reconstruction may be necessary in combination with an ACL reconstruction (Peyal et al., unpublished data, 2000). In rare instances, patients with combined ACL-MCL injuries heal sufficiently such that symptomatic instability resolves altogether, thereby obviating the need for reconstruction.

There is little controversy regarding graft positioning. When performing an ACL reconstruction, the surgeon should always avoid anterior graft placement on both the fibula and femur. The graft position should also be in-

spected intraoperatively during passive range of motion to avoid notch impingement. Meticulous surgical technique in which precise placement of the graft is performed should prevent the motion problems.

Postoperatively, early motion (particularly complete extension) should be encouraged. An aggressive rehabilitation program that includes mobilization of the patella and early quadriceps muscle function is advocated. Although other surgeons do not advocate brace use after surgery or require brace use only until quadriceps muscle strength returns, we use a hinged knee brace that is locked in extension during ambulation (2 to 3 weeks for bone-patellar tendon-bone autografts and 4 to 6 weeks for hamstring tendon autografts). Having patients apply a splint to the knee nightly until extension is easily achieved is useful for 2 to 3 weeks; however, we believe that casting, prolonged immobilization, and orthotic devices with extension blocks should be avoided. In general, we tailor the patient’s weightbearing status according to the associated injuries, the type of graft used, and the quality of graft fixation. Prevention and early recognition of postoperative hemarthrosis is critical as this can cause quadriceps muscle inhibition, leading to motion problems.

### EARLY RECOGNITION

Most authorities believe that early recognition of and intervention in motion problems lead to improved outcomes. Close monitoring with prone heel hangs or having a 2-week goal of full extension and $120^\circ$ of flexion can lead to earlier detection of motion problems. After any reconstructive procedure, flexion and extension must be systematically assessed, and the motion should be compared with the uninvolved, contralateral knee.

A swollen knee postoperatively can cause motion problems. Torry et al. have recently shown that an intraarticular effusion induces a quadriceps muscle avoidance gait pattern even in healthy persons. Prevention of hemarthrosis postoperatively allows for more comfortable return of knee motion and can prevent quadriceps muscle shutdown. Because the causes for motion loss are broad, we find it useful to subdivide them into problems that cause loss of flexion and problems that cause loss of extension (Table 2).

### TABLE 1

The Classification Scheme for Arthrofibrosis Developed by Shelbourne et al.

<table>
<thead>
<tr>
<th>Type</th>
<th>Flexion (deg)</th>
<th>Extension (deg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Normal</td>
<td>&lt;10</td>
</tr>
<tr>
<td>2</td>
<td>Normal</td>
<td>&gt;10</td>
</tr>
<tr>
<td>3</td>
<td>&gt;25</td>
<td>&gt;10</td>
</tr>
<tr>
<td>4</td>
<td>&gt;30</td>
<td>&gt;10 with patella infera</td>
</tr>
</tbody>
</table>

### TABLE 2

Causes of Motion Loss

<table>
<thead>
<tr>
<th>Loss of Extension</th>
<th>Loss of Flexion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Malpositioned or nonsymmetric graft (anterior tibial tunnel, anterior femoral tunnel)</td>
<td>Suprapatellar adhesions</td>
</tr>
<tr>
<td>Notch impingement</td>
<td>Patellar entrapment</td>
</tr>
<tr>
<td>ACL nodule</td>
<td>Medial and lateral gutter adhesions or fibrosis</td>
</tr>
<tr>
<td>Infrapatellar contracture syndrome</td>
<td>Improper graft position</td>
</tr>
<tr>
<td>Captured joint capsule after meniscal repair</td>
<td>Infrapatellar contracture syndrome</td>
</tr>
<tr>
<td>Posterior capsular scarring</td>
<td>Reflex sympathetic dystrophy</td>
</tr>
<tr>
<td>Hamstring tightness</td>
<td>Soft tissue calcifications of capsule or MCL</td>
</tr>
<tr>
<td>MCL calcification</td>
<td>Postinfection</td>
</tr>
<tr>
<td>Postoperative infection</td>
<td>Quadriceps contracture or myositis</td>
</tr>
<tr>
<td>Reflex sympathetic dystrophy</td>
<td></td>
</tr>
</tbody>
</table>

**TABLE 2**

Causes of Motion Loss
and physical examination alone, although radiographs may be useful to assess tunnel placement and patellofemoral alignment. Important issues such as patella infera and graft malposition may be determined fairly reliably by radiographs. Bony nodules on the tibia and calcifications of the MCL, readily seen on plain films, may provide clues. We always look for loss of patellar motion on physical examination and for patella infera by comparison radiographs. If questions remain or if the diagnosis is unclear, MRI is obtained to more carefully evaluate the soft tissues. Often, an ACL nodule, fat pad scarring, or graft malposition may be noted. We also carefully examine the chondral surfaces so that we may present realistic expectations to the patient. Because the surgeon dealing with these difficult cases should be prepared to deal with all possibilities, all necessary information should be obtained preoperatively.

By monitoring active flexion and extension and palpating for crepitus or a “clunk,” the clinician may be able to detect an ACL nodule. An ACL nodule typically results in pain with knee extension about 2 to 3 months postoperatively. The clinician should be alerted to the possibility of reflex sympathetic dystrophy when patients have pain out of proportion to examination, allodynia, and trophic or sudomotor changes.

**TREATMENT**

The treatment of motion loss after soft tissue injury or surgery to the knee should be targeted to the specific cause. Prevention of motion loss is obviously the best strategy; however, when this fails and motion problems occur, a careful and systematic approach should establish the correct diagnosis. Treatment can thus be targeted accordingly. Most authorities believe that early recognition and prompt intervention will decrease long-term morbidity for the patient.

Many patients in whom motion loss has developed can be treated nonoperatively. Physical therapy and manipulation under anesthesia remain the main nonoperative approaches. Arthroscopic or open surgical treatments are indicated when nonoperative measures have failed or when there is a surgically correctable cause. Only in rare instances is operative intervention used alone; more often, surgical treatment is combined with specific rehabilitation and pain management protocols.

**Nonoperative Treatment**

While most knees with motion problems benefit from intervention, knees that are inflamed and warm, with soft tissue swelling and motion loss, have an active process that should not be forcefully treated. In these circumstances, rest, ice, and antiinflammatory agents are the treatments of choice. In essence, tissue injury has led to the release of cytokines and growth factors that result in stimulation of fibroblasts, leading to fibrosis and joint contractures. Active manipulation at this time will only result in further tissue damage and scarring. In these settings, active range of motion and muscle exercises are permitted, but forceful extension and flexion are avoided. Radiographs obtained at this point should be carefully reviewed for soft tissue calcifications, which may be seen as early as 6 weeks after injury but often require 3 months or more to develop. If calcification of the MCL is noted, with pain and limited motion, it is usually best to avoid aggressive attempts at restoring motion. Gradually, the soft tissue insult will clear and usually allow full return of motion. Pain may persist over the calcified area however for months. On occasion, we have seen extensive calcifications around the joint that have resulted in severe motion loss. These calcifications will subsequently require debridement after the process has matured, which may take 6 to 12 months. A bone scan may be helpful in these instances.

**Treatment with Physical Therapy**

Physical therapy is usually the first-line approach to any postoperative motion problem about the knee. Noyes et al. reported on a rehabilitative treatment plan for early postoperative limitations in knee motion. Knees that did not regain motion as quickly were placed in an early postoperative phased treatment program that included serial casting and aggressive motion exercises. We have used modalities such as hanging weights, quadriceps muscle strengthening, extension casting, and drop-lock braces with variable success. Sliding boards, passive flexion exercises, and continuous passive motion machines may also be helpful. In difficult or complex cases, we use continuous passive motion, although we are careful to remember that continuous passive motion promotes flexion but not extension. If therapy leads to increased swelling, inflammation, and pain, it is best to stop attempts at gaining motion and allow this phase to pass. If therapy is continued, the process will only accelerate with a greater degree of stiffness and motion loss.

**Manipulation Under Anesthesia**

Manipulation of the knee under anesthesia has been used to improve motion in the postoperative period. Currently, we do not recommend this treatment unless it is performed in conjunction with an arthroscopic procedure to remove scar tissue and release the joint capsule. Dodd et al. reported the results of knee manipulations in 42 knees with persistent flexion or extension deficits after intraarticular ACL reconstructions. At the time of manipulation, average flexion increased from 95° to 136° and average extension from 11° to 3°. At final follow-up, average flexion and extension were 127° and 4°, respectively. Final range of motion was not affected by time to manipulation, severity of flexion deficit, or concomitant arthroscopic debridement of adhesions. However, knees with premanipulation extension deficits of 15° or more achieved significantly less final extension than knees with lesser premanipulation deficits. The authors concluded that manipulations were a safe and effective method for improving both flexion and extension in knees that had restricted motion after ACL reconstructions.
We believe that manipulation works best for loss of flexion from mild degrees of arthrofibrosis. Generally, however, we avoid manipulation as a first-line approach and only use it in conjunction with an arthroscopic or open procedure. Focal lesions, such as ACL nodules, malpositioned grafts, or severe arthrofibrosis, respond better to surgery. When manipulation is used, it should be performed gently, as overloading the chondral surfaces by aggressive manipulation can damage the cartilage and lead to further degeneration. With aggressive manipulation, there is also a risk of stimulating myositis ossificans of the quadriceps muscle or ossification of the MCL. When calcifications or ossifications are noted, we believe it is best to stop all rehabilitation and decrease knee motion. The knee should be placed at rest and the patient should be prescribed antiinflammatory medications to avoid the vicious cycle with progressive loss of motion.

Anesthesia and Analgesia

For patients in whom a manipulation or surgical procedure is to be performed, we advocate the use of regional epidural anesthesia and indwelling epidural catheters for postprocedure, patient-controlled analgesia. This type of anesthesia provides better local pain control and therefore allows more intensive physical therapy in the immediate postprocedure period. Our standard patient-controlled analgesia protocol involves fentanyl and mepivacaine at a low-dose, continuous infusion regimen and a patient-controlled, rescue dose, maximum of four doses per hour.

Principles of Surgical Management

When nonoperative measures fail, or when there is a discrete surgically correctable abnormality, we advocate arthroscopic surgery. When surgery is undertaken, we perform a systematic nine-step evaluation, regardless of whether the procedure is performed arthroscopically or as an open procedure. Figure 1 clearly shows and describes each of the nine steps of this evaluation.

Arthroscopic Treatment

Arthroscopic debridement is often successful and can frequently be performed on an outpatient basis. Arthroscopic treatment is best indicated when the block to motion is intraarticular, such as an ACL nodule or an adhesion. A preoperative MRI may be helpful in the diagnostic evaluation. During arthroscopy, careful and thorough inspection of all compartments is required.

In severe cases, where it may be difficult to insert the arthroscope, we reestablish the suprapatellar pouch and the medial and lateral gutters first. This helps with visualization of the knee joint. Next, we recommend that the infrapatellar fat pad be debrided and the recess between the patellar tendon and anterior tibia be reestablished. Care should be taken to preserve the intermeniscal ligament. In cases of excessive scarring and patellar entrapment, we perform releases of the medial and lateral retinaculum. When performing these medial and lateral capsular releases arthroscopically, care should be taken to completely release the tissues as these maneuvers help reestablish patellar motion as well as tibiofemoral motion. Gentle manipulation may also be used as an adjunct.

As a general rule, limitations of extension usually reside in the notch and posterior capsule, while limitations of flexion usually reside in the suprapatellar pouch or gutters. Therefore, the notch should be carefully inspected. If there is evidence of graft impingement, a notchplasty should be performed. Fibroproliferative ACL nodules should be excised, if present. If the cruciate graft or native ligaments are malpositioned or excessively scarred, they should be debrided, released, or excised altogether. Steadman et al. have described limited open procedures to release the posteromedial and posterolateral capsules. These procedures may be added if necessary. In our experience, knee motion will not improve from the motion that was attained in surgery. Therefore, every attempt should be made to achieve satisfactory motion before the patient leaves the operating room.

Results of Arthroscopic Treatment. Arthroscopic treatment results in an excellent outcome with good motion and restoration of function. Arthroscopic treatment results in an excellent outcome with good motion and restoration of function. In 1982, Sprague et al. were among the first authors to describe the arthroscopic treatment of knee fibroarthrosis. Their report details the arthroscopic treatment of 24 patients who had had previous open procedures and had failed nonoperative measures. The authors noted that arthroscopy was particularly useful for the treatment of intraarticular adhesions.

More recently, Marzo and colleagues reported on arthroscopic treatment of symptomatic ACL nodules. Removal of the nodules resulted in improvement of extension from an average of 11° preoperatively to an average of 0° at 1-year follow-up. Side-to-side difference in terminal extension, using the uninvolved limb for comparison, averaged 3°. Fisher and Shelbourne reported on 42 patients who required arthroscopic treatment for symptomatic extension block. Marked improvements in function and symptoms were found after excision of the offending tissue. Jackson and Schaefer reported on 13 patients with ACL nodules who were treated with arthroscopic debridement and manipulation. Postoperatively, average loss of extension improved from 16° to 3.8°. Klein et al. reported on the arthroscopic management of postoperative arthrofibrosis in 46 knees. They reported good-to-excellent results in 76% of patients, with 80% of patients reporting a decrease in pain. Lysholm and Tegner activity scores also improved, although patient satisfaction was good or excellent in only 56.5% of cases.

Open Debridement and Soft Tissue Release

Open procedures may be required to restore knee motion in patients with severe scarring or in those who have failed less-invasive approaches. Chronic motion problems are often difficult to treat, especially in cases with long-standing extension deficits and generalized arthrofibrosis. Open debridement and soft tissue release may be used as a salvage procedure in the treatment of patients with...
severe motion loss who have failed arthroscopic techniques.\textsuperscript{1,9,15}

When there are extensive calcifications of the extraarticular tissues, we advocate open excision to restore motion. Surgery should be delayed 6 to 12 months until the process has matured; maturity is heralded by decreased swelling, pain, and inflammation. All scarred tissue will need to be excised to restore motion. If the excision leads to loss of medial or lateral collateral ligament function, the defect can be reconstructed with a semitendinosus tendon autograft; an Achilles tendon allograft has been used when a semitendinosus tendon autograft is not available.

When open procedures are required to restore motion, it is important to thoroughly debride all scar tissue, both anteriorly and posteriorly. We advocate an anterior extensive approach, when feasible, through a medial parapatellar arthrotomy performed through the medial aspect of the quadriceps tendon, the medial retinaculum, over the medial aspect of the patella, and down onto the anterior tibia. Previous incisions should be incorporated or modified as needed.

Figure 1. Coronal (A) and sagittal (B) diagrams illustrating the nine regions that need to be addressed systematically when surgically treating motion problems of the knee. 1, evaluate and reestablish the suprapatellar pouch; 2, evaluate and reestablish the medial gutter; 3, evaluate and reestablish the lateral gutter; 4, debride and mobilize the infrapatellar fat pad and reestablish the pre-tibial recess; 5, evaluate lateral retinaculum and perform lateral retinacular release if tight or scarred; 6, evaluate medial retinaculum and perform medial retinacular release if tight or scarred; 7, evaluate intercondylar notch, debride scar tissue, and, in severe cases, release ACL or PCL, or both; 8, evaluate tibial insertion of posterior capsule, inspect the capsular recess, perform capsulotomy if necessary; 9, evaluate femoral insertion of posterior capsule and release if necessary.
An extensive subperiosteal medial dissection should be performed around the tibia to the level of the posterior tibial plateau. As has been reported in the arthroplasty literature, we routinely release the semimembranosus tendon in these difficult cases but preserve the superficial MCL. This helps in mobilizing the tibia and in regaining extension. Next, all anterior scar tissue should be debrided, and the medial and lateral gutters should reestablished. Dense scar tissue will invariably be encountered in the infrapatellar fat pad and extensor mechanism. This should be removed carefully while mobilizing the patellar tendon from the anterosuperior border of the tibia. Particular care should be taken to preserve the extensor mechanism. It is often necessary to perform a lateral retinacular release to assist in mobilization of the patella. We do not recommend the use of a quadricepsplasty.

After mobilization of the extensor mechanism, release of the anterior structures, and excision of intraarticular adhesions, passive flexion and extension should be assessed. The cruciate ligaments, whether native or grafts, should then be assessed for malposition and impingement. If the ACL is malpositioned, excessively scarred, or shortened, it is debrided or excised. When the ACL is excised, the tibia can be subluxed anteriorly, so the PCL and posterior aspect of the knee can be explored.

When a flexion contracture exists, the tight posterior capsule can be released by performing a subperiosteal dissection from the femur and tibia. Dense scarred capsular tissue can be “peeled” away subperiosteally, revealing the femur and tibia. We find this very effective when dealing with severe flexion contractures. At this point, if the PCL is malpositioned, blocks motion, or impinges, it can be also excised.

Meticulous hemostasis should be achieved to prevent a postoperative hematoma, as it has been our experience that postoperative hemarthrosis contributes significantly to pain and flexion contractures and may incite a more intense inflammatory response. For similar reasons, we advocate the use of suction drains postoperatively to prevent hemarthrosis.

Results of Open Treatment. We have recently reported on eight patients who had severely restricted motion with extensive intraarticular and periarticular fibrosis. Range of motion averaged 62.5° preoperatively (flexion, 81°; loss of extension, 18.8°). Patients underwent open debridement and soft tissue release as a salvage procedure to restore motion. In this series, there were no complications, although one patient did require late PCL reconstruction for instability. Total motion improved to an arc of 124° postoperatively, while patient satisfaction and function were high. There was a high incidence of patellofemoral arthritis noted at follow-up, with patellar tendons that continued to shorten over time.

In another similar series of patients with severe arthrofibrosis with flexion contractures, patients underwent anterior and posterior open procedures to restore motion. In this series of 21 patients with long-standing symptomatic motion problems, the mean extension deficit of 17° improved to a mean of 2°, with all deficits less than 5°, after the procedure. The knee function correspondingly improved, and no neurovascular complications were observed.

SUMMARY

Limited motion of the knee after ligament injury causes significant pain and functional impairment. Classification systems for the diagnosis and treatment of motion problems have been developed based on physical findings and loss of motion compared with the opposite normal knee. Improved operative techniques and better preoperative and postoperative rehabilitation have improved outcomes. In our opinion, prevention of motion problems is best achieved by delaying ACL reconstruction until the patient has a normal gait, full range of motion, and minimal swelling in the injured knee. An aggressive postoperative rehabilitation program that focuses on regaining full extension through early quadriceps muscle activity and range of motion exercises should also prevent problems. When motion loss does occur, early recognition, proper diagnosis, and targeted treatment may be expected to improve function in most patients.

REFERENCES