Knee pain is one of the most frequent complaints evaluated by orthopedic surgeons. It encompasses a broad range of pathology and can present in a variety of ways. Most of this pain can be attributed to essential structures of the knee, including the menisci, cruciate or collateral ligaments, and articular cartilage. However, there are underrecognized structures in and around the knee that can frequently be a cause of knee pathology and pain. Knee pain stemming from these structures may be missed or incorrectly diagnosed, and these patients often present for second and third opinions because of failure to diagnose and treat the underlying pathology. The synovial plica, suprapatellar pouch, lateral retinaculum, infrapatellar fat pad, and infrapatellar branch of the saphenous nerve are less common but still significant causes of knee pain. Although initial treatment involves various nonoperative modalities, operative treatment is often warranted. Operative and nonoperative management of these soft tissue structures may occur in isolation or with concomitant procedures, including knee ligament reconstruction, total knee arthroplasty, tibial tuberosity osteotomy, or lysis of adhesions. With proper recognition of the role of these structures in knee pain, the orthopedic surgeon can offer a valuable primary or adjunctive treatment option for patients with knee pain, especially those without localizing signs of meniscal, ligamentous, or cartilage damage. [Orthopedics. 2016; 39(1):32-42.]

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pole of the patella and the proximal portion of the patellar tendon and inferiorly to the transverse meniscal ligament, the anterior horns of the medial and lateral menisci, and the peristeum of the tibia.\textsuperscript{3,4} The medial and lateral patellomeniscal ligaments stabilize the fat pad in the coronal plane.\textsuperscript{5,6} Its posterior surface is lined with synovium and forms the ligamentum mucosum (infrapatellar plica).\textsuperscript{2,3} Anastomotic connections from the inferior, superolateral, and superomedial geniculate arteries provide a rich blood supply to the infrapatellar fat pad that can make this structure a significant source of bleeding during arthroscopy.\textsuperscript{7,8} A rich network of innervation to the fat pad is provided by the articular branches of the tibial nerve as well as its intimate connections to the highly innervated synovium.\textsuperscript{9-11} Additionally, the fat pad was a major pain generator during conscious neurosensory mapping of the internal structures of the knee without intra-articular anesthesia.\textsuperscript{12}

**Pathology**

Hoffa’s disease, or infrapatellar fat pad impingement syndrome, results from impingement and inflammation of the extrasyovial infrapatellar fat pad.\textsuperscript{13} Infrapatellar fat pad impingement syndrome can occur as a result of repetitive knee microtrauma or after multiple knee surgeries, including total knee arthroplasty (TKA).\textsuperscript{14} A clear relationship of fat pad pathology, including hypertrophy and fibrosis, with progression of cartilage disorders, including osteoarthritis, has not yet been found. However, an association with fat pad fibrosis or hypertrophy and degeneration of cartilage has been reported.\textsuperscript{14}

**Clinical Evaluation**

Patients with Hoffa’s disease commonly describe burning or aching anterior knee pain near the patellar tendon and the inferior pole of the patella.\textsuperscript{5,15,16} Patients may also present with swelling, clicking, locking, and catching sensations, and they may stand with hyperextended knees to prevent fat pad impingement. A positive Hoffa’s test result, in which the patient has increased pain when the knee is brought from 30° to 60° flexion to full extension while firm pressure is applied inferior to the patella outside the margins of the patellar tendon, is suggestive of Hoffa’s disease.\textsuperscript{5,13,17} In patients who have undergone TKA, a palpable and audible patellar clunk may be present as the hypertrophic fat pad impinges between the patella and the femoral components. To test for patellar clunk, the patient flexes the knee to 120°, which causes scar tissue on the quadriceps tendon to move into the notch of the femoral component. As the patient extends the knee (especially against resistance), the scar tissue catches the anterior edge of the femoral component. Finally, the scar releases with a painful, audible clunk, and the knee can then extend. A patellar clunk may also be present in the setting of a Cyclops-type lesion after anterior cruciate ligament reconstruction.

Magnetic resonance imaging (MRI) is a helpful adjunct to clinical evaluation in confirming the diagnosis of Hoffa’s disease. Edema of the superolateral area of the infrapatellar fat pad is the most important diagnostic MRI criterion for the diagnosis of Hoffa’s fat pad impingement.\textsuperscript{18} A T1-weighted MRI scan shows hypointense enlargement of the infrapatellar fat pad, and a T2-weighted image shows hyperintensity at the same location. In addition, T2 images may show joint effusion and associated intra-articular pathology, including meniscal tears.\textsuperscript{19} A retrospective study of 135 patients investigated the association between MRI findings of Hoffa’s fat pad and patellar articular cartilage abnormalities and reported that abnormal signal intensity in Hoffa’s fat pad has sensitivity of 31% and specificity of 73% for patellar chondropathy.\textsuperscript{20}

**Treatment**

Nonoperative treatment of Hoffa’s disease has a high success rate. This treatment includes nonsteroidal anti-inflammatory drugs and physical therapy to restore normal patellar tracking. Closed chain quadriceps strengthening exercises and gluteal muscle strengthening to improve pelvic stability improve patellar tracking.\textsuperscript{13,21} Taping can also be used to lift the inferior pole out of the infrapatellar fat pad and reduce inflammation.\textsuperscript{13} With the patient supine and the knee extended, the patella is displaced posteriorly, and a piece of tape is applied over the proximal half of the patella. A second piece of tape is applied from the lateral border of the patella over its proximal half to create lateral patellar tilt. A wide V tape is then placed inferior to the patella while the patella is pulled superiority, medially from the tibial tubercle to the medial epicondyle, and laterally from the tibial tubercle to the lateral epicondyle to further unload the fat pad. The goal is to create a “muffin top” to unload the fat pad as much as possible. Injections of lidocaine with methylprednisolone or hydrocortisone can also lead to improvement in pain.\textsuperscript{16,22}

If conservative measures are unsuccessful, several operative interventions can be used. Arthroscopic partial resection of the infrapatellar fat pad has shown good results (Figure 1).\textsuperscript{14,23} Ogilvie-Harris and Giddens\textsuperscript{25} reported good results in 10 of 12 patients at a mean follow-up of 76 months, and Kumar et al\textsuperscript{14} reported that 30 of 34 patients returned to their preinjury Tegner score at a mean follow-up of 68 months after arthroscopic resection of the infrapatellar fat pad. In patients with focal hypertrophic fibrosis of the fat pad, targeted arthroscopic debridement may be warranted and has been associated with good patient satisfaction scores.\textsuperscript{24,25} Anterior interval release, which involves the release of scar tissue connecting the infrapatellar fat pad to the anterior tibia, may also be indicated. Steadman et al\textsuperscript{15} reported that anterior interval arthroscopic release with electrocautery improved patella excursion, resolved flexion contractions, and improved postoperative pain and functional scores in 21 of 25 patients at a mean follow-up of 4 years.\textsuperscript{5} Care
should be taken both to avoid destabilization of the anterior meniscal horn attachment sites and to achieve adequate hemostasis to prevent postoperative hematoma formation. Synovectomy of the posterior portion of the fat pad during TKA in patients with rheumatoid arthritis resulted in decreased pain compared with control subjects and led to fewer complications than were noted in those who underwent total resection of the infrapatellar fat pad. Arthroscopic denervation is another possible treatment. Ogon et al. reported complete resolution of symptoms in 13 of 15 patients at a mean follow-up of 41 months after arthroscopic denervation of the nociceptive nerves, with innervation of the proximal portions of the fat pad and the inferior pole of the patella. Caution must be used to avoid iatrogenic injury to the patellar tendon.

**SYNOVIAL Plica**

**Overview**

Synovial plicae are remnants of the synovial septa that once partitioned the embryonic medial and lateral tibiofemoral compartments and the suprapatellar bursa. Plicae normally diminish in size during fetal development as these 3 compartments mature into the synovial capsule. However, the involution process may be incomplete and plicae can persist into adulthood as inward folds of the synovial lining in up to 4 distinct intra-articular locations: suprapatellar, medial parapatellar, lateral parapatellar, and infrapatellar. The infrapatellar plica, or ligamentum mucosum, is the most common, existing in up to 85% of patients. The medial parapatellar plica is the most commonly symptomatic plica. The incidence of medial parapatellar plicae is 18.5% to 80%.

**Pathology**

Plicae are usually thin and pliable connective tissues that are harmless and unobtrusive. However, they can become thickened and inelastic with inflammatory processes of the synovium, including hemorrhage secondary to trauma or irritation with repeated flexion and extension maneuvers, and in association with other intra-articular pathology. Thickened and inelastic plicae can damage nearby intra-articular structures, a process called pathologic synovial plica syndrome. Pathologic medial parapatellar plicae have been reported to cause abnormal patellofemoral tracking as well as chondromalacia and erosion of the medial femoral condyle as the stiffened plica inappropriately glides over the condyle in flexion. Several classification systems characterize the anatomy of suprapatellar and medial parapatellar plicae. Synovial plica syndrome can be especially challenging to diagnose because the symptoms can mimic more common knee pathology, such as articular cartilage lesions and meniscal tears.

**Clinical Evaluation**

Patients with synovial plica syndrome almost universally present with anterior or anteromedial knee pain. This syndrome is frequently associated with abnormal use of the knee during physical activity and is characterized by pain, swelling, popping, snapping, catching, and locking symptoms that may mimic a meniscal tear. Symptoms often worsen with exercise that loads the patellofemoral joint. On examination, the area over the inflamed plica is tender to palpation, and a cord-like band may be felt in patients with symptomatic medial parapatellar plicae. Reproduction of the discomfort can often be elicited with knee flexion and extension while pressure is applied to the patella either medially or laterally. In the diagnosis of medial plica, MRI has been reported to provide 93% sensitivity and 81% specificity. Plicae appear as low-intensity bands on T1-weighted and T2-weighted MRI images. Axial views on MRI scans are particularly helpful (Figure 2A).

**Treatment**

Nonoperative therapy is the initial treatment strategy and may be most helpful in young patients without structural impingement of the plica. These modalities include rest, nonsteroidal anti-inflammatory drugs, quadriceps strengthening (vastus medialis oblique), hamstring stretching, and corticosteroid injections. In patients who require surgery, resection of the plica is the goal (Figures 2B-C). Excision of the plica with an accessory superolateral portal in combination with a 70° scope may
allow the best visualization, although adequate resection can often be achieved with a 30° scope via the standard inferomedial and inferolateral portals alone (Figure 2D). On arthroscopy, the plica may be easily visualized as a classic plica shelf. Alternatively, it may adhere to the overlying capsule and appear instead as a thickened plica sheet. In the case of a plica sheet, debridement with an arthroscopic shaver pointing away from the capsule helps to develop the interval between the plica and the capsule and allows for appropriate visualization while mitigating the risk of iatrogenic injury to the capsule and subsequent capsular herniation. Once the plica has been identified or separation from the overlying capsule has been achieved, the entire course of the plica should be identified. The use of gentle sweeping motions with the smooth end of the shaver can be particularly helpful to separate the plica both proximally and distally from the capsule in a definite layer without damaging other knee structures. Once full exposure has been achieved, the shaver is used to debride the plica, and electrocautery or bipolar radiofrequency ablation devices are used for hemostasis. When resecting the plica, it is important to take care to avoid damaging the joint capsule during excision. In a recent meta-analysis that included 969 patients treated with open or arthroscopic plica resection at a mean follow-up of 27.5 months, 64% of patients were free of symptoms and returned to unlimited activities, 26% had occasional symptoms that did not affect their activity, and 10% did not show improvement or experienced worsening of symptoms. Complications unique to arthroscopic plica removal can include violation of the joint capsule and/or destabilization of the meniscus with overly aggressive plica resection, in addition to infection, arthrofibrosis, and plica recurrence.

SUPRAPATELLAR POUCH

Overview

The suprapatellar pouch is a continuation of the knee joint cavity and the synovial membrane proximal to the patella. The pouch is anterior to the femur and posterior to the quadriceps tendon, and it extends approximately 4 cm proximal to the superior pole of the patella. It continues on the medial and lateral sides of the patella as the medial and lateral gutters, which are also covered by synovial membrane. On arthroscopy, the entire pouch, along with the medial and lateral gutters, can be visualized with a 30° arthroscope. The synovium should appear as healthy tissue, without signs of synovitis, irritation, or thickening, and the quadriceps tendon should be seen running along the roof of the pouch and inserting into the superior pole of the patella.

Pathology

Suprapatellar pouch pathology includes synovitis, arthrofibrosis, suprapatellar plica syndrome, traumatic lesions, impingement secondary to dysfunction of the articularis genus muscle, and mass lesions. Because its inner surface is lined with synovium, the suprapatellar pouch is susceptible to pathologic processes that cause synovitis, including osteoarthritis, rheumatoid arthritis, and other systemic inflammatory conditions. The suprapatellar pouch is also the most common area of knee arthrofibrosis. Suprapatellar plicae can become hypertrophic and fibrotic, causing shearing forces on the articular cartilage, degenerative changes, and impingement. Traumatic lesions of the pouch may be caused by intra-articular loose bodies or iatrogenic injury. Dysfunction of the articularis genus muscle can also cause knee pain. The articularis genus, which originates on the anterior femoral shaft and inserts on the superior aspect of the suprapatellar pouch, pre-
vents invagination of the pouch beneath the patella. When dysfunctional, this can also lead to pouch impingement, irritation, injury, and pain. Mass lesions in the suprapatellar pouch include lipoma arborescens, 73-76 pigmented villonodular synovitis, 77-79 giant cell tumor, 80 hemangioma, 81 synovial chondromatosis, 29 and synovial cysts. 82,83

Clinical Evaluation

Patients with suprapatellar pouch pathology typically present with anterior knee pain and effusion. In patients with chronic inflammation of the pouch, arthrofibrosis may develop and lead to significantly restricted range of motion. Examination often shows decreased patellar mobility compared with the contralateral knee. Loss of knee flexion on examination is a common finding in these patients and can be measured with the patient prone or supine. Although loss of extension is less common, when present, it causes more severe functional limitations than loss of flexion. 84 Insufflation of the knee with 60 mL saline may also be helpful in the diagnosis of suprapatellar plica or arthrofibrosis. Irrigation drips slowly out of an 18-gauge needle after insufflation of 60 mL saline with normal joint capacity. However, insufflation of 60 mL saline into a knee with significant plica or arthrofibrosis causes pressurized backflow. 85 An MRI scan is the diagnostic imaging modality of choice because it allows proper visualization of the plica and identification of suprapatellar synovitis and provides clues about the etiology of any neoplastic lesions. 58,74,86-91

Treatment

For symptomatic suprapatellar pouch pathology, treatment is aimed at the underlying etiology. Activity modification to decrease loading of the patellofemoral joint, physical therapy, ice therapy, ultrasound, microwave diathermy, bracing, local steroid injections, and nonsteroidal anti-inflammatory drugs are nonoperative treatments that may provide symptomatic relief. 92-95 Surgical therapy is indicated if nonoperative treatment is unsuccessful. In suprapatellar plica syndrome with arthrofibrosis, arthroscopic electrocautery or bipolar radiofrequency ablation is used for resection. Slow resection with electrothermal cautery is recommended, and mobility of the patella in all planes should be significantly improved (Figure 3). Therefore, it is imperative to perform a thorough preoperative examination, both in the preoperative holding area and then once the patient is relaxed intraoperatively, for comparison.

There are several other less common causes of pouch pathology, including pigmented villonodular synovitis, lipoma arborescens, synovial hemangioma, synovial chondromatosis, and cystic disease. The most common is villonodular synovitis, also known as tenosynovial giant cell tumor, which is an intra-articular pathology caused by abnormal proliferation of the synovial membrane. It occurs predominantly in young adults, may affect the suprapatellar pouch, and is treated with excision. In a retrospective series of 41 patients treated for diffuse villonodular synovitis (20 in the open surgery group and 21 in the multidirectional arthroscopy group), at follow-up of 36 months, Gu et al. 96 reported that the multidirectional arthroscopic technique was associated with significantly shorter operative time and hospital stay, less blood loss, and better postoperative International Knee Documentation Society and Lysholm scores than open surgery (P<.001). There were 4 recurrences reported in the open surgery group and 1 in the multidirectional arthroscopy group. All 5 recurrences were treated with a second surgery, without subsequent recurrences. 96

Lateral Patellar Retinaculum

Overview

The lateral patellar retinaculum is an aponeurotic expansion from the vastus lateralis and has a complex anatomy consisting of superficial oblique and deep transverse layers. 97 The superficial oblique layer includes the patellar tendon, the vastus lateralis, and the iliobial band, and it is reported to provide minimal stability to the patella. 97,98 Deep to the superficial oblique layer is the denser, deep transverse layer, consisting of distinct fibrous consolidations, including the lateral patellofemoral ligament, the lateral patellotibial ligament, and the patello-tibial band. 97,98 The lateral patellofemoral ligament is a thickening of the lateral joint capsule and forms the superior border of the deep transverse layer, 99 connecting the patella to the lateral epicondyle to provide superolateral support to the patella. 97,98 It is smaller and more difficult to define than the medial patellofemoral ligament. 100 The reported width and length of the lateral patellofemoral ligament are 16 mm and 42.1 mm, respectively. 99 The lateral patellotibial ligament is located at the in-
Pathology

Lateral patellar compression syndrome results from a relative increase in the tightness of the lateral patellar soft tissue restraints compared with the medial restraints. This results in increased stress on the lateral patella, a finding that Johnson reported to be the consequence of an adolescent growth cycle in which the less compliant lateral patellar soft tissues become a primary restraint to the patella with growth of the distal femoral epiphysis. With increasing stress on the lateral tissue as body weight increases, inflammation of the lateral tissues may lead to scarring and shortening of the lateral complex, with eventual overload of the lateral patellar compartment. Excessive tension in the lateral patellar soft tissues, subchondral bone microfractures, interosseous hypertension, and synovitis above the patellar tubercle, and synovitis above the patellar tubercle, and physical examination findings consistent with tightness of the lateral patellar retinaculum are hallmark findings. Patients with signs of excessive patellar mobility on examination must be carefully evaluated to rule out concurrent patellar instability.

The lateral compression test, J sign, patellar tilt test, and grading patellar glide test (Sage sign) are clinical examinations that can help to elucidate a tight lateral retinacular complex. The lateral compression test attempts to correlate anterolateral knee pain with articular degeneration of the lateral patellofemoral compartment. A positive test result is noted when pain is elicited by compression of the lateral patella as the patient flexes and extends the knee. A tight lateral patellar retinaculum may also cause lateral subluxation of the patella in active terminal extension, which is called the J sign. The patellar tilt test evaluates the tension of lateral restraint. It is performed with the patient supine and the knee in 20° flexion with the trapezius condylar axis parallel to the table. An attempt is made to elevate the lateral aspect of the patella and depress its medial edge. If the lateral retinaculum is excessively tight, the test result will be abnormal and the examiner will be unable to tilt the patella to horizontal (Figure 4A). Finally, the patellar glide test can help to elucidate a tight lateral retinaculum. With the patient supine and the knee in 30° flexion, the patella normally can be manually displaced both medially and laterally. Patellar motion is graded by the number of quarter widths that the patella can be manually displaced. Medially, normal displacement is 1 to 2.5 quadrants. Displacement of less than 1 quadrant indicates a tight lateral restraint (retinaculum), and movement of greater than 3 quadrants suggests a hypermobile patella.

Patellar instability can also cause anterior knee pain and may mimic the symptoms of lateral retinacular tightness. Careful differentiation of patellar instability from a tight lateral retinaculum is critical because performing lateral retinacular release in a patient with patellar instability makes the instability worse. Knee radiographs in the anteroposterior, lateral, and Merchant views can be helpful to investigate the degree of lateral patellar tilt and trochlear and patellar morphology. Long radiographs to include the anterior superior iliac spine can be used to determine the quadriceps angle (Q angle). The tilt angle is determined on the Merchant view and defined as the angle subtended by a line joining the medial and lateral edges of the patella and the horizontal. Tilt of greater than 5° is considered abnormal. The Q angle, which is formed by a line drawn from the anterior superior iliac spine to the central patella and a second line drawn from the central patella to the tibial tubercle, is also important to measure. A value greater than 15° in women and 10° in men may signify a tight lateral retinaculum. Both MRI and computed tomography scans can be useful in assessing the tibial tubercle–trochlear groove distance. A normal tibial tubercle–trochlear groove distance on MRI is 10±1 mm. A value greater than 20 mm should raise concern about possible patellar malalignment and improper patellar tracking. In these patients, a tibial tubercle osteotomy with possible medial patellofemoral ligament reconstruction is needed rather than simple soft tissue release of the lateral patellar retinaculum.

Clinical Evaluation

Lateral patellar compression syndrome is diagnosed based on a history of knee pain, radiographic evidence of excessive patellar tilt, and physical examination findings consistent with tightness of the lateral patellar retinaculum. Anterior knee pain with stair climbing and prolonged sitting (theatre sign), lateral patellar tenderness and crepitus on examination, and inability to evert the lateral edge of the patella are hallmark findings. Patients with signs of excessive patellar mobility on examination must be carefully evaluated to rule out concurrent patellar instability.

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an in vivo MRI-based study, suggesting that strengthening of the vastus medialis oblique may be beneficial. Goh et al. reported in a cadaver study that the patella displaced laterally and increased the load on the lateral patellar facet throughout the range of knee motion when the vastus medialis oblique was inactivated. The only indication for operative intervention for lateral compression syndrome is continued pain despite 6 months of appropriate physical therapy, with a positive lateral patellar tilt test finding on examination and no signs of patellar hypermobility (negative Sage sign). Lateral release procedures can be performed as open, mini-open, or arthroscopic procedures proximal to the vastus lateralis or distal along the patellar tendon (Figure 4B). Patient satisfaction scores (good and excellent) for lateral release range from 14% to 99%. However, the results of these studies are difficult to generalize because the study populations were not homogeneous and some studies did not clearly separate patients with anterior knee pain alone from those with knee pain and patellofemoral instability. The results are generally poor if lateral release is performed as a single procedure in patients with evidence of concurrent patellar instability. In a recent cadaveric biomechanical study, lateral retinacular release did not medialize the patella or reduce peak patellofemoral contact pressure throughout knee extension. In 8 knees without evidence of patellar instability, lateral patellar retinacular release medialized the position of the patella and the peak area of patellofemoral contact only between 60° and 120° and lateralized the patella and the peak contact position with further extension. In patients with evidence of patellar instability, an abnormal Q angle, or an abnormal tibial tubercle–trocchlear groove distance on imaging, distal realignment, repair/reconstruction of the medial patellofemoral ligament, or both may be indicated. Complications have been described in as many as 11% of patients who undergo open or arthroscopic lateral release. These include an increase in medial instability/dislocation, recurrence of instability, quadriceps atrophy, and weakness. Lateral patellar retinacular lengthening as an alternative to release has been reported with both open and arthroscopic techniques. In a recent prospective double-blind study of 28 patients (14 in the open lateral retinaculum lengthening group and 14 in the open lateral retinaculum release group), open lateral retinaculum lengthening resulted in a significantly lower rate of medial patellar subluxation and quadriceps atrophy and better clinical outcomes (lower mean Kujala score) at 2-year follow-up.

**ARTHROFIBROSIS**

**Overview**

Prolonged inflammation as a result of the conditions discussed earlier or in the setting of trauma, degenerative arthritis, or cruciate ligament reconstruction (especially multiligament reconstruction), or in the postoperative period may lead to soft tissue scarring, decreased compliance, and eventual loss of motion, a collection of findings known as arthrofibrosis. The exact prevalence of arthrofibrosis is not known, but it is probably best studied after anterior cruciate ligament reconstruction and TKA. In a recent multicenter study of 980 patients (540 male) who underwent anterior cruciate ligament reconstruction surgery, 53 patients (5.4%) had undergone an arthrofibrosis procedure on the ipsilateral knee at 6 years of follow-up. Other authors estimated this incidence at 4% to 35%. The scarring adhesions seen with arthrofibrosis may cause limitations in knee range of motion. In extreme cases, the entire synovial compartment of the knee may be fibrotic and lead to pain and restriction of motion, despite rehabilitation, exercises, and stretches.

**Pathology**

Abnormal fibrous hyperplasia of intra-articular and extra-articular knee structures leads to restriction of joint motion. Loss of motion may be severe if fibrosis occurs throughout the knee, or it may be moderate if fibrosis is limited to the anterior knee compartment, posterior capsule, or suprapatellar pouch. Generally, fibrosis may cause a lack of flexion, extension, or both. Flexion contractures limit patient function and make the leg functionally short. As the flexion contracture increases, it places excessive strain on the quadriceps muscle to stabilize the knee. A 5° loss of extension may cause patellofemoral pain and a limp during walking. A simulated flexion contracture of 15° significantly increased the energy cost of walking. Additionally, a flexion contracture of 30° significantly changed the kinematics of the trunk. Loss of extension is usually associated with scar formation in the anterior compartment of the knee, and it may also be caused by fibrosis in the posterior compartment.

An extension contracture leads to inability to fully flex the knee.
tractures are usually better tolerated than flexion contractures. Severe flexion deficits of greater than 90° affect the ability to sit or climb stairs.\textsuperscript{132} However, restricted flexion does not severely affect gait as long as the knee can be flexed to 60°.\textsuperscript{133} Loss of flexion is often associated with intra-articular fibrosis and scarring of the patellofemoral joint, and patellar mobility is universally decreased.\textsuperscript{134,135} The consequent pain may lead to the cascade of quadriceps weakness, patellar tendon adaptive shortening, and scarring in the tissues around the patella, with an end stage of permanent patella infera.

**Clinical Examination**

Patients who present with arthrofibrosis should undergo a full history and physical examination. It is important to identify the onset of stiffness, associated injuries and conditions, and previous treatments, especially surgery. Radiographs, including films of the contralateral joint, are helpful and may show heterotopic ossification, joint subluxation, patellofemoral disease, or evidence of osteoarthritis. Symptoms of arthrofibrosis include stiffness, pain, limping, heat, swelling, crepitus, and/or weakness.\textsuperscript{25} Depending on the site of scarring, patellar mobility and/or joint range of motion may be affected, and MRI can help to visualize the knee compartments affected.\textsuperscript{5}

**Treatment**

Nonoperative interventions include compression, elevation, physical therapy, corticosteroid injections, nonsteroidal anti-inflammatory drugs, and cryotherapy. However, in many patients with knee fibrosis, surgery is required. Arthroscopic lysis of adhesions as well as anterior interval, posterior capsular, peripatellar, and suprapatellar release may be indicated and may be performed successfully ([Figure 5]).\textsuperscript{136} In almost all cases, surgery is followed by a strict postoperative rehabilitation plan.

Recent advances in arthroscopic technique led to improved outcomes in patients with intra-articular fibrosis of the knee. Kim and Joo\textsuperscript{137} studied the results of arthroscopic adhesiolysis for arthrofibrosis of the knee and performed an analysis to investigate possible prognostic factors. Their study analyzed 68 patients who underwent arthroscopic adhesiolysis of the knee and were available for at least 1-year follow-up. They studied the influence of possible prognostic factors, including the cause of arthrofibrosis, the duration of disease, and patient age on postoperative recovery of range of motion. They found that 61 patients (89.7%) had an average increase in range of motion of 48.6°; however, the remaining 7 patients (10.3%) showed no increase at final follow-up. The Lysholm knee score and the International Knee Documentation Society subjective knee score increased significantly at final follow-up. Patient satisfaction was high or very high in 89.7% of patients at final follow-up. Additionally, the duration of disease was significantly related to postoperative recovery of range of motion, and longer duration of symptoms was associated with worse postoperative recovery of range of motion. There was no association between the cause of arthrofibrosis and patient age with postoperative increase in range of motion.

**Conclusion**

There is a spectrum of intra-articular pathology related to the nonligamentous soft tissue structures of the knee. These abnormalities may occur as isolated clinical entities or in the setting of concomitant chondral, meniscal, or ligamentous injury or arthrofibrosis. Diagnosis of these less discussed causes of knee pain and dysfunction requires an understanding of the anatomy and pathology of these structures and a high index of clinical suspicion.
With appropriate patient selection and understanding of the available treatment options, good to excellent results may be achieved for most isolated soft tissue abnormalities. Treating these pathologic structures, when present, during surgery for other major knee pathology (i.e., meniscal, ligamentous, chondral) may lead to decreased intra-articular pain, improved patient outcomes, and joint homeostasis.

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