Arthroscopic Untethering of the Fat Pad of the Knee: Release or Resection of the Infrapatellar Plica (Ligamentum Mucosum) and Related Structures for Anterior Knee Pain

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Abstract: Anterior knee pain (AKP), a multifactorial symptom complex, can be successfully treated surgically. A specific diagnosis often cannot be made, but the pain is linked to an unrecognized common factor in most patients: the mechanical behavior of the non-isometric contents of the anterior compartment of the knee—the fat pad (FP) and infrapatellar plica (IPP). The objective of this presentation is to describe an effective arthroscopic technique that treats AKP by addressing this common factor. The operation consists of release or resection of the IPP, or ligamentum mucosum, which tethers the FP. These highly innervated tissues act together as a hydraulic shock absorber, filling the anterior compartment. They stretch and deform at the extremes of knee motion because of constraint centrally by the non-isometric IPP. These dynamic changes in shape are eliminated when the plica is released or resected. Pain perception is from perturbed nociceptive nerves: pain relief results from de-tensioning these contained nerves by untethering the fat pad. Ascribing pain causation is problematic because morphologic change, such as inflammation, fibrosis, or contracture of these structures, is only present in a minority of cases. Nonetheless, AKP is both physically linked to these central, pain-sensitive structures and relieved by this operation.

Anterior knee pain (AKP), as discussed by Grelsamer et al. (2009), is an unsolved problem in orthopaedics, best treated as a symptom complex without relying on a specific diagnosis or cause. It is very common, with incidence rates far exceeding those of osteoarthritis: it is found in 1 in 14 adolescents and 1 in 10 military members. It is characterized by a deep parapatellar ache, usually associated with squatting, weight-bearing activities, and prolonged sitting. It is often transiently worse during the first few steps after arising, and it may be accompanied by crepitation, weakness, giving way, and catching. The most common physical finding is a positive Hoffa test, and 80% of patients have pain with squatting. It can coexist with, and indeed mimic, other pathology in the knee and is likely multifactorial. No specific investigation is diagnostic. Recent literature has increasingly suggested that there are few structural abnormalities and that the cause of pain is likely neural damage as the “provoking factor.” This article describes a simple, safe operation that addresses AKP associated with derangement of the infrapatellar plica (IPP) and fat pad (FP) in terms of perturbation of the nerves contained in this structural unit. The procedure has a high probability of eliminating the pain, with a complication rate as low as that of diagnostic arthroscopy.

Several authors have suggested that the long-term outcome of AKP, believed by many to be untreatable surgically, may not be benign. The natural history of adolescent AKP has been studied. In many
adolescents, the condition is benign and self-limited; however, in 25% to 45%, pain becomes chronic with long-term sequelae, both physical and mental. These patients may have higher levels of mental distress and perceive lower levels of health; they may adopt the coping skills of chronic pain patients. The adult population may be equally affected, given that more than half the patients with patellofemoral pain reported long-term pain and disability after adequate conservative management. AKP, when prolonged, can be a life-changing condition worthy of further study. Our suggested clinical approach offers a simple arthroscopic procedure, which is safe, potentially allowing a return to normalcy, even after years of AKP.

The procedure involves release or resection of the IPP, or ligamentum mucosum, effectively untethering the FP. It was performed for the first time by the senior author (T.V.S.) during arthroscopy in an adult patient with chronic, disabling AKP. No images were preserved at the time (1990); however, the first clinical case, presented in Video 1, showed remarkably similar arthroscopic findings, as shown in Figure 1. Pristine articular surfaces and menisci were present, with a small zone of inflammation adjacent to the femoral insertion of a normal-appearing, rope-like IPP, which showed subtle changing tension with knee motion. This observation suggested that the structure, rather than being an embryologic remnant of no clinical significance, had an actual function related to this non-isometric mechanical behavior. The IPP was released at its femoral attachment (FA), with rapid and permanent pain relief. This led to the described surgical approach to AKP, as well as the subsequent

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**Fig 1.** Non-isometric mechanical behavior of infrapatellar plica (IPP), viewed arthroscopically (screenshots from Video 1). Arthroscopic views of the notch from the anterolateral portal (A-E) and anteromedial portal (F) are shown in the right knee of a 16-year-old patient with anterior knee pain for 4 years, with an insidious onset, undergoing release and resection of the IPP at its femoral attachment (FA). (A) At 90° of knee flexion, the IPP, of normal structure, is vertical and taut. (B) At 45° of knee flexion, the IPP has 2 parallel elements, classified as “split.” The leading edge is a curved arc suggesting laxity. (C) At 20° of knee flexion, the leading edge straightens as the IPP approaches the apex of the notch; the FA has rotated posteriorly out of view. (D) At 10° to 15° of knee flexion, the IPP contacts and indents the articular surface at the notch; with further extension, the apex of the notch acts as a pulley. From kinematic videos, the IPP increasingly elongates with further extension. The apposing aspect of the IPP and the central body attachment of the fat pad (FP) are compressed against the articular surface of the trochlea, potentially inducing surface reactive change. (E) At 0° of knee flexion and in full extension, the IPP and the central body attachment of the FP to the IPP have rotated out of view and the FP apposes the trochlear articular surface. (F) At 90° of knee flexion, with viewing from the anteromedial portal, the IPP has been released. The FP seen on the left, floating free of the central attachment, is no longer tethered by the IPP at the FA. (CB, central body attachment of FP to IPP; FA, femoral attachment; Infl, small zone of possible inflammation.)
Pathologic structures are relatively uncommon and are approached as follows:

**First principle:** Untether the fat pad, eliminating any central restraint; release and/or resect the IPP and any other attached structure that interferes with the freely reversible deformation and stretch of the fat pad (such as anomalous bands).

**Second principle:** Restore, as much as possible, the anatomic contours of the borders of the anterior compartment, eliminating interference with the fat pad as it deforms with knee motion. This implies removal of local pathology, which is uncommon, such as the following:
- Cyclops lesion attached at the base of the reconstructed ACL
- Arthrofibrotic scarring
- Attached tissues—which may be osseous, cartilaginous, or both (osteochondromatous)—or other soft-tissue lesions (cysts, local PVNS, or any soft-tissue tumor)

**Third principle:** Because the fat pad is a structure of multiple functions, disturb it as little as possible.

**Fourth principle:** Because the IPP–fat pad complex is a potential pain generator, consider untethering the fat pad every time arthroscopy is performed on a knee for undiagnosed pain.

**Table 1. Surgical Principles for Untethering of Fat Pad by Release or Resection of IPP and Related Structures**

| Principle | Description |
|-----------|-------------|
| First principle | Untether the fat pad, eliminating any central restraint; release and/or resect the IPP and any other attached structure that interferes with the freely reversible deformation and stretch of the fat pad (such as anomalous bands). |
| Second principle | Restore, as much as possible, the anatomic contours of the borders of the anterior compartment, eliminating interference with the fat pad as it deforms with knee motion. |
| Third principle | Because the fat pad is a structure of multiple functions, disturb it as little as possible. |
| Fourth principle | Because the IPP–fat pad complex is a potential pain generator, consider untethering the fat pad every time arthroscopy is performed on a knee for undiagnosed pain. |

**Table 2. Steps in Surgical Management for Untethering of Fat Pad**

The contents of the anterior compartment show wide variation, normally containing the IPP and fat pad. Releasing or resecting the IPP untethers the fat pad, modifying the physiology of this normal tissue complex that can become a pain generator. The following steps are recommended: first untether the fat pad and then address observed abnormalities.

To untether the fat pad, release and resect the following structures:

**If the IPP is either separate or split, do as follows:**
- Release its FA, a curved arc of fibrous tissue, using an arthroscopic punch.
- Use a shaver cautiously to remove the fibrous elements of the IPP, avoiding resection of fat pad elements as much as possible.

**If the IPP is a vertical septum, with or without a fenestra, do as follows:**
- Release the FA (as above).
- Use a shaver (hood reversed) to protect the ACL, starting from the FA, carefully removing the attached fibrous and fatty elements down to the intermeniscal ligament.
- If an anomalous band is present, completely remove it, usually with a punch and shaver.

At this point, the fat pad should be sitting free, distracted by filling pressure. You should have an unobstructed view of the normal structures:
- trochlea above, bony notch, ACL, PCL, superior tibia, anterior horns of meniscus, intermeniscal ligament, and fat pad. Usually, the operation is complete at this point.

Pathologic structures are relatively uncommon and are approached as follows:

- The cyclops lesion at the base of the ACL may have fibrous, cartilaginous, and bony elements; a combination of a punch, shaver, and burr allows complete removal.
- A cyst may appear as a soft-tissue prominence which can be unroofed, using a punch, and debrided with a shaver.
- Masses are approached according to their nature.
- Adherent or protuberant arthrofibrotic scar is removed or resected back to the approximate margin of the adjacent tissues.
- Local tissues that appear normal, such as a lipoma, local PVNS, and fibrous or osteocartilaginous masses, are excised.
- Heterotopic ossification is excised; abnormal bone (post-traumatic, osteophytic) can be resected back to the normal bone contour using a burr.

**Surgical Technique**

This section outlines how to perform this operation. As detailed in Table 1, the principles are to untether the fat pad, and to restore the integrity of the contents of the anterior compartment so that there is no restraint to the ability of the semi-liquid fat pad to fill the space and attenuate force. Avoid resection of the fat pad, a structure of many functions, in the absence of properly controlled studies to support this. The Discussion section introduces new aspects of knee physiology that relate to the structure and function of the contents of the anterior compartment of the knee. The operation interrupts this physiology and in so doing provides pain relief in most. Table 2 outlines the steps involved in performing the procedure as is further outlined in detail in this section. Table 3 discusses what might go wrong, and how to avoid this.

**Figure 2** shows the operative setup and approaches. Figure 1 is a composite of screenshots from the first case in Video 1 and correlates with further images from kinematic studies (Figs 3 and 4). Figure 5-8 are screenshots from the second case described in Video 1 and correlate with the steps described in the following sections, outlining how to perform this operation.

**Table 3. What Might Go Wrong, and How to Avoid This**

This clinical experience, spanning 28 years and hundreds of patients from 9-year-old children to elderly patients, is consistent with several reports in the literature of small numbers of patients in whom AKP has been relieved by release of the IPP at its FA.

ACL, anterior cruciate ligament; IPP, infrapatellar plica; PVNS, pigmented villonodular synovitis.
Step 1: Preoperative Setup and Diagnostic Arthroscopy

The patient is positioned supine with a small bolster under the hip to bring the leg into a neutral position, with the anterior surface of the patella parallel to the floor. A tourniquet is applied but rarely inflated. Standard diagnostic arthroscopy is performed using the anterolateral portal (ALP) and anteromedial portal (AMP), with occasional use of the superolateral portal (SLP) (Fig 2).

Step 2: Viewing of Apex of Notch, Classification of IPP, and Observation of Abnormalities

The ALP is established with the knee flexed 90°; at the level of the lower pole of the patella, a longitudinal incision is placed just lateral to the palpable edge of the patellar tendon (Fig 2). After completing the joint survey, the surgeon should approach the anterior compartment from above, noting the integrity of the trochlear articular surface as the apex of the notch comes into view. The trochlear surface may be normal or may show surface changes from linear grooves, beginning at the apex (Fig 8C), to frank osteoarthritis, a presumed effect of increasing compression and shear, arising only in terminal knee extension, induced by tension forces in the adjacent IPP and FP as they distort remarkably (Figs 3 C and D and 9). Figure 5 presents sample screenshots from the second clinical case in Video 1, with views of the apex of the notch and the contents of the anterior compartment of a right knee viewing from the ALP, which show that the IPP type is a fenestrated vertical septum (Fig 5A).

Obtaining a clear view of the apex of the notch can be difficult if there is abundant fat. The IPP tethers the FP and, because it is non-isometric, holds the FP tightly against the distal femur, increasingly approaching end extension. To obviate this, the knee should be flexed 30° to 45°, relaxing the IPP. A probe (blunt trocar or hook) can be inserted through a portal established medially (AMP). The surgeon should use an 18-gauge

| Risks and Pitfalls                                      | How to Avoid Risks and Pitfalls                                                                 |
|--------------------------------------------------------|-----------------------------------------------------------------------------------------------|
| Hemarthrosis, leading to scarring or contracture       | Careful dissection and meticulous hemostasis                                                   |
| Stiffness and/or loss of knee motion postoperatively   | Early mobilization emphasizing active and passive range of motion to full range with an experienced physical therapist |
| Failure to fully achieve optimal strength and range of motion | Understanding that the progressive decrease in function of the limb accompanying chronic AKP may require 12 to 18 mo of rehabilitation; perseverance is required |
| Deep venous thrombosis                                 | Use of postoperative chemical and mechanical prophylaxis as determined by patient risk factors; early mobilization |

AKP, anterior knee pain; IPP, infrapatellar plica.
spinal needle, placed superior to the meniscus, avoiding the FP (Fig 2). The tip of the needle should be advanced to verify that the instruments placed along this line can fully access the IPP. The surgeon should use distraction with the probe to visualize the IPP, which is normally floppy and easily stretched, noting any variance from the categories described by Kim et al., as well as any other abnormalities (anomalies,
cyclops lesions, bony prominences, inflammation, cysts, osteophytes, or other soft-tissue and bony masses). The surgical principles of untethering the FP are outlined in Table 1. The surgical sequence is further discussed in Table 2.

**Step 3: Evaluation of Mechanical Behavior of IPP**

As every human being is unique, anatomic variation is expected and normal. We use Kim’s classification of the IPP (five subtypes), noting that Derganc in 1969 described ten, and Wachtler in 1979 described five. Brooker in 2009 focused on the FP, rightly describing wide variation in the gross anatomy, and an absence of data on what is normal and what is pathologic. This paper focuses on the combined structure and function of the tissue complex of the IPP and FP. It notes that no two knees are the same, and that abnormal tissues are present in a minority of patients with AKP treated with success by this procedure. Behavior can be observed directly through the arthroscope (representative screenshots are shown in Figs 1 and 5-8) and indirectly by kinematic studies (Figs 3 and 4) performed in volunteers undergoing arthroscopy. The 2 clinical cases can be reviewed in Video 1 and the screenshots that follow; in both cases, viewing is performed from the ALP in right knees. The first case, with a split IPP, shows characteristic non-isometric behavior in the composite image in Figure 1. The IPP

![Fig 5. In step 2, we view the apex of the notch, classify the infrapatellar plica (IPP), and note abnormalities. A right knee is shown, viewed through the anterolateral portal, in a 32-year-old male patient with anterior knee pain for 6 years after a direct blow to the front of the knee (second case presented in Video 1). (A) In this gestalt view in mid flexion, an attempt is made to visualize the complete functional unit of the IPP. There is a shallow groove at the apex of the notch and below, the IPP appears “separate”. (B) However, adjusting flexion to 30° gives the complete picture. The IPP is a wall of connective tissue, a vertical septum, in which there is an opening, or fenestra. Above, the femoral attachment of the IPP (FA)—an enthesis—is seen as a curved arc of connective tissue attached to bone. Below and behind is the now relaxed border of the fenestra, connective tissue that merges with the anterior border of the ACL. Adjacent to the IPP is a separate connective tissue band, an anomaly. The IPP is thus a fenestrated vertical septum. (C) At about 60° the more dense superior border of the IPP is apparent, with diaphenous loose connective tissue at the margin of the window. (D) Close-up of the fenestra, with the ACL behind, the rest of the IPP above, and fat pad to the right. (ACL, anterior cruciate ligament.)](https://example.com/fig5)
is increasingly linear and taut approaching full flexion (Fig 1A), is lax in mid-arc (Fig 1B), and then appears to tighten, with its leading edge straightening and approaching the apex of the notch (Fig 1C) and contacting it (Fig 1D) at about 10° to 20° of flexion. The IPP then disappears and rotates out of view (Fig 1E), ending the opportunity for direct viewing.

The vertical septum variant (with or without an opening, or fenestra; Figs 5-8) is physically attached to the ACL, a robust ligament that is nearly isometric\textsuperscript{38}; its mechanical behavior is damped and is not so apparent. The screenshots from the second case in Video 1 are representative of this: In Figure 6A, at 90°, the IPP is vertical but not so clearly stretched; in Figure 6B, at 45°, no laxity is apparent as the leading edge is straight; in Figure 6C, the IPP contacts the articular surface at the apex of the notch at 10° to 15° of knee flexion; and finally, in Figure 6D, the IPP is no longer seen as the central body transition zone and FP are drawn tightly against the trochlea.

The kinematic studies involve the introduction of radiographic contrast medium into the FP and IPP in patients undergoing arthroscopy (Video 1; Figs 3 and 4, showing representative screenshots).\textsuperscript{27,28,30} Lateral fluoroscopy allows indirect observation of the mechanical behavior of the IPP-FP complex, especially
in terminal extension, when the tight apposition of the IPP, central body, and FP against the distal femur blocks the possibility of a direct view. These studies, with awake, mildly sedated volunteers, support direct arthroscopic observations of non-isometric behavior (Fig 1) during knee motion, including stretch in flexion (Fig 1A) and contact of the IPP with the apex of the notch (Fig 1 C and D, Video 1) during extension. In Figure 3B, the ripple seen in the IPP suggests laxity in mid-flexion. What happens in terminal extension cannot be seen directly because the arthroscope is pushed out of the way by the semiliquid FP as it attempts to fill the changing external geometry of the space. What is observed in Figure 3 C and D is remarkable stretch of the IPP and distortion of the FP, with contrast being squeezed out of the base.

With identification completed and mechanical behavior confirmed, the anterior compartment is inspected for abnormalities, which occur rarely. These include bony and soft-tissue anomalies, cysts, heterotopic bone, osteophytes, fibrotic scarring including cyclops lesions and fibrous connective tissue bands, tumors, and contracture of the anterior interval.39

**Step 4: Untethering of FP by Release or Resection of IPP and Restoration of Anatomy of Anterior Compartment**

If the IPP is separate or split, a simple release of the IPP at the FA point is carried out with a Stryker punch (Conquest, 3.4 mm straight or 15° up-angled) and small shaver (Stryker Formula Tomcat Cutter, 4.0 mm) or similar device to sever or remove the connective tissue at the bony attachment, which is not a single point but rather an arc of connective tissue that is inferior to the articular margin at the apex of the notch (Figs 1 B and F, 5B, 7A, and 8B; Video 1). A radiofrequency...
device (Werewolf Flow 50 Wand; Smith & Nephew) can be used to shrink the connective tissue and to establish hemostasis.

If the IPP is a vertical septum or is fenestrated, further resection of the substance of the IPP attached to the ACL is necessary after release at the FA. Any other connective tissues attached to the FP, including anomalous bands (Fig 5B) and post-traumatic fibrous bands, are removed in their entirety. Any mass lesion (cyclops lesion, cyst, or other tissue) is removed to allow the FP unobstructed freedom to fill the space. Meticulous dissection should be performed, with the use of a punch and small shaver to sever or remove the connective tissue at the FA and attached to the ACL. The radiofrequency device can be used for further removal or shrinkage of soft tissues and for hemostasis, which should be complete. A burr is used for bony resection. The final step is a joint survey to verify that the anterior compartment has been restored and that nothing is left tethering the FP, which is observed to sit at a distance from the notch, distracted by the fluid pressure of the pump system (Figs 1F and 8; Video 1). At this point, the procedure is terminated.

Concomitant pathology is addressed as follows: If a medial plica is present, it is not removed unless there is evidence of mechanical interference with the articular surface of the medial femoral condyle or the medial aspect of the patella, or if it appears thickened and inflamed. If a medial shelf is present, it is removed. Any other plica or fibrous adhesion should be assessed; if it is...
anomaly that should be removed because it will be non-isometric and likely symptomatic. An attempt is made to restore the bony contours of the compartment by resecting osteophytes, heterotopic bone, and prominent callus if there has been a previous fracture. If chondromalacia is present, it is debrided sufficiently to produce a stable articular surface using the radiofrequency device in the Coblation setting (Smith & Nephew). Inflamed synovium is debrided.

Postoperative Recovery and Rehabilitation

The postoperative course involves rest and self-directed exercises for the first week, allowing the physiology of the knee to return toward normal. A small effusion may remain for a week or, if there has been substantial dissection, for a longer period. The duration and intensity of the rehabilitation program are individualized. In general, physical therapy is directed toward achieving a full range of motion. Core and global strengthening of the lower extremities is undertaken. In many patients, within 4 weeks, the extremity is near normal and a return to training for sport can begin. The rehabilitation process can be prolonged if the problem has been present for years, but with persistence, full pain-free function can be expected.

Discussion

The operation involves release or resection of the IPP, an intra-articular ligament, which tethers the FP. Knowledge of the structure, function, and physiology of these tissues is important in understanding how this relieves pain. Structure—gross anatomy and histology (Figs 10 and 11)—is linked to function (filling space and force attenuation), observable as mechanical behavior, directly through the arthroscope (Figs 1 and 5-8) and indirectly by kinematic studies performed by the senior author (T.V.S.) (Figs 3 and 4). In these studies, radiographic contrast was placed arthroscopically into the IPP and FP of cadavers and volunteers. The gross and microscopic analyses show that the ligamentous IPP and the FP collectively act as a unit—a deformable, semiliquid, hydraulic shock absorber filling the anterior compartment. Direct observation arthroscopically (Figs 1 and 5-8, Video 1) and kinematic studies (Fig 3, Video 1) show non-isometric behavior, with the
IPP and FP rotating about the FA, that is, the enthesis. The kinematic behavior of the IPP-FP complex when the IPP is separate or split shows stretch and deformity at the extremes of knee motion (Figs 1A and 3C and D), with laxity in between (Figs 1B and 3B), because the central attachment point of the IPP is at the FA. This is well below the instant center of rotation of the knee (ICR), making the IPP-FP complex non-isometric. With motion, contained within the borders of the AC, the IPP-FP complex, tethered at the FA, rotates around and not the ICR (Fig 3, Video 1), leading to stretch and deformity. (B) Sagittal section of an anatomic specimen showing how the AC (bordered by the dashed line) is filled by the IPP-FP complex. The probe is in the infrapatellar bursa. (C) Magnetic resonance imaging correlate, with the knee in full extension. The IPP-FP complex fills the AC and is in tight apposition to the articular surface of the trochlea. (CB, central body [which links FA and FP].)

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This physiology is present in all knees with an IPP (86.5% depending on population). When pain is present, the physical distortion and stretch of the IPP-FP complex perturb the nociceptive neural network, and the symptom complex of AKP results. Pain perception is from activated nociceptive nerves. Ascribing pain causation is problematic because morphologic change, such as inflammation, fibrosis, or contracture of these structures, is present in only a minority of cases. AKP is not well correlated with location around the knee because it is physically linked to the widespread innervation of these central, pain-sensitive structures. Indeed, the neural network involved arises from all the nerves supplying the knee (femoral, tibial, common peroneal, recurrent peroneal, and obturator). Pain generated from the IPP-FP complex can thus be felt anywhere in and about the knee, explaining the varied perceived localization of AKP and why it mimics other pathology. The current general consensus lumps together all the highly innervated soft tissues, including the retinaculum, synovium, and FP, and the medial patellofemoral ligament as the sources of pain. This article focuses on a much more specific concept, that is, that the common link in AKP, when it is associated with the presence of an IPP, is the physiology and function of the highly innervated and sensate IPP-FP complex. This operation, which untethers the FP, alters this physiology, and reliably helps most such patients.
Success is not always achieved, again indicating the complexity of the problem. Complete rehabilitation must be ensured, requiring patience in the very chronic case. The approach, when pain returns or is not relieved, is to re-evaluate clinically. The possibility of neuromatous formation at the portals is eliminated and patellar stability rechecked, given the overlap of AKP with patellar instability. If indicated, further surgery is offered. Psychosocial and secondary gain issues associated with Workers’ Compensation must also be carefully considered.

Many surgeons, faced with chronic recalcitrant AKP, consider this a nonsurgical problem best treated by further conservative management or by pain...
management specialists.\textsuperscript{6,45,46} We have observed that adolescents, whose quality of life has been profoundly disrupted for years, returned to a pain-free state after this simple procedure. A subset of successfully treated adults had experienced adolescent AKP, requiring lifestyle modifications to minimize pain. Many with this symptom complex, despite normal radiographs, had been advised that they had “arthritis.” Even after many years, such patients can be restored to full function, including active sports, with minimal symptoms. Accordingly, our approach is to carefully explain the complex nature of the problem and the risks, benefits, and alternatives, and to suggest the option of arthroscopic treatment. We offer a confirmatory clinical test in which the patient exercises to induce the pain. A dilute local anesthetic mixture containing bupivacaine (8 mL, 0.125%) is injected into the symptomatic knee immediately after exercise. Complete pain relief indicates that the problem is intra-articular, in which case we offer arthroscopy to address any observed pathology. We consider every case individually: The more chronic the pain, the longer that the neural pathways and second-ary, compensatory lifestyle modifications have been established. In such cases, we proceed even if the patient had some remaining pain after injection of the local anesthetic. We have shown success in most patients with the described procedure, with complete pain relief immediately after the operation in most cases, even after many years. The only complication has been transient portal pain, which can be neuroma-like, treated, if persistent, by local injection. The mixture is composed of bupivacaine (3 mL, 0.25%) and 10 mg of methylprednisolone acetate, injected slowly with a 1.5-inch No. 25 needle, starting 1 inch proximal to the painful portal; this is followed by weeks of use of a neoprene sleeve, applying slight pressure to the soft tissues of the knee. Other risks of the procedure are shown in Table 3.

The finding that recalcitrant AKP is relieved in many patients by release or resection of the IPP strongly suggests consideration of this as a first surgical approach in the patient in whom conservative management has failed. AKP is complicated and multifactorial,\textsuperscript{1} but treatment in this way, as a symptom complex with a specific anatomic link to the FP and IPP, allows pain relief for many patients.

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References
1. Grelsamer R, Moss G, Ee G, Donell S. The knee: The patellofemoral syndrome; the same problem as the Loch Ness Monster? Knee 2009;16:301-302.
2. Thomas M, Wood L, Selle J, Peat G. Anterior knee pain in younger adults as a precursor to subsequent patellofemoral osteoarthritis: A systematic review. BMC Musculoskelet Disord 2010;11:201.
3. Smith B, Selle J, Thacker D, et al. Incidence and prevalence of patellofemoral pain: A systematic review and meta-analysis. PLoS One 2018;13:e0190892.
4. Crossley KM, Stefanik JI, Selle J, et al. 2016 Patellofemoral pain consensus statement from the 4th International Patellofemoral Pain Research Retreat, Manchester. Part 1: Terminology, definitions, clinical examination, natural history, patellofemoral osteoarthritis and patient-reported outcome measures. Br J Sports Med 2016;50:839-843.
5. Sanchis-Alfonso V, Roselló-Sastre E. Hypothesis: Anterior knee pain in the young patient-what causes the pain? “Neural model”. Acta Orthop Scand 2003;74:697-703.
6. Smith T, McNamara I, Donell ST. The contemporary management of anterior knee pain and patellofemoral instability. Knee 2013;20:53-515.
7. Sanchis-Alfonso V. Holistic approach to understanding anterior knee pain. Clinical implications. Knee Surg Sports Traumatol Arthrosc 2014;22:2275-2285.
8. Saciri V, Pavlovic V, Zupanc O, Baebler B. Knee arthroscopy in children and adolescents. J Pediatr Orthop B 2001;10:311.
9. Sandow MJ, Goodfellow JW. The natural history of anterior knee pain in adolescents. J Bone Joint Surg Br 1985;67:36.
10. Dye SF. The pathophysiology of patellofemoral pain: A tissue homeostasis perspective. Clin Orthop Relat Res 2005;436:100-110.
11. Price AJ, Jones J, Allum R, et al. Chronic traumatic anterior knee pain. Injury 2000;31:373-378.
12. Stathopulu E, Baildam E. Anterior knee pain: A long-term follow-up. Rheumatology 2003;42:580.
13. Rathleff MS, Rathleff CR, Olesen JL, Rasmussen S, Roos EM. Is knee pain during adolescence a self-limiting condition? Am J Sports Med 2016;44:1165-1171.
14. Luhmann SJ, Schoenecker PL, Dobbs MB, Eric Gordon J. Adolescent patellofemoral pain: Implicating the medial patellofemoral ligament as the main pain generator. J Child Orthop 2008;2:269-277.
15. Kannus P, Natri A, Paakkala T, Järvinen M. An outcome study of chronic patellofemoral pain syndrome. Seven-year follow-up of patients in a randomized, controlled trial. J Bone Joint Surg Am 1999;81:355-363.
16. Natri A, Kannus P, Jarvinen M. Which factors predict the long-term outcome in chronic patellofemoral pain syndrome? A 7-yr prospective follow-up study. Med Sci Sport Exerc 1998;30:1572-1577.
17. Nimon G, Murray D, Sandow M, Goodfellow J. Natural history of anterior knee pain: A 14-to 20-year follow-up of nonoperative management. J Pediatr Orthop 1998;18:118.
18. Post WR. Patellofemoral pain: Results of nonoperative treatment. Clin Orthop Relat Res 2005;436:55-59.
19. Jensen R. Patellofemoral Pain Syndrome. Studies on a Treatment Modality, Somatosensory Function, Pain and Psychological Parameters [doctoral thesis]. Bergen, Norway: University of Bergen, 2009.
20. Carlsson AM, Werner S, Mattlär CE, Edman G, Puukka P, Eriksson E. Personality in patients with long-term patellofemoral pain syndrome. *Knee Surg Sport Traumatol Arthrosc* 1993;1:178-183.

21. Thomee P, Thomee R, Karlsson J. Patellofemoral pain syndrome: Pain, coping strategies and degree of well-being. *Scand J Med Sci Sports* 2002;12:276-281.

22. Lankhorst NE, van Middelkoop M, Crossley KM, et al. Factors that predict a poor outcome 5-8 years after the diagnosis of patellofemoral pain: A multicentre observational analysis. *Br J Sports Med* 2016;50:881-886.

23. Boyd CR, Eakin C, Matheson GO. Infrapatellar plica as a cause of anterior knee pain. *Clin J Sport Med* 2005;15:98-103.

24. Schindler OS. Synovial plicae of the knee. *Curr Orthop* 2004;18:210-219.

25. Hardaker WT, Whipple TL, Bassett FH. Diagnosis and treatment of the plica syndrome of the knee. *J Bone Joint Surg Am* 1980;62:221-225.

26. O’Dwyer KJ, Peace PK. The plica syndrome. *Injury* 1988;19:350-352.

27. Smallman TV, Race A, Ekroth S. Intra-operative visualization of deformation of the infrapatellar plica (IPP), and fat pad—The link to anterior knee pain. *J Bone Joint Surg Br* 2012;94:100 (suppl XXXVIII).

28. Smallman T. In vivo arthroscopic behavior of the infrapatellar plica of the knee. [https://clinicaltrials.gov/ct2/show/NCT00643487](https://clinicaltrials.gov/ct2/show/NCT00643487). Accessed January 1, 2014.

29. Smallman T, Shekitka K, Mann K, Race A. The infrapatellar plica—A new non-isometric intra-articular ligament of the knee. *FASEB J* 2015;29:545.3 (suppl).

30. Smallman T, Mann K, Race A. Intra-operative visualization of deformation of the infrapatellar plica (IPP), and fat pad—The link to anterior knee pain. *FASEB J* 2015;29:865.16.

31. Demirag B, Ozturk C, Karakayali M, Burak Cagatay O, Mehmet KD. Symptomatic infrapatellar plica. *Knee Surg Sport Traumatol Arthrosc* 2006;14:156-160.

32. Kim SJ, Kim JY, Lee JW. Pathologic infrapatellar plica. *Arthroscopy* 2002;18:E25.

33. Kim SJ, Choe WS. Pathological infrapatellar plica: A report of two cases and literature review. *Arthroscopy* 1996;12:236-239.

34. Kim SJ, Min BH, Kim HK. Arthroscopic anatomy of the infrapatellar plica. *Arthroscopy* 1996;12:561-564.

35. Derganc F. Plica synovialis infrapatellaris. *Rev Chir Orthop Reparatrice Appar Mot* 1969;55:633-638 [in French].

36. Wachtler F. Plica synovialis infrapatellaris in man. *Acta Anat (Basel)* 1979;104:451-459 [in German].

37. Brooker B, Morris H, Brukner P, Mazen F, Bunn J. The macroscopic arthroscopic anatomy of the infrapatellar fat pad. *Arthroscopy* 2009;25:839-845.

38. Zavras TD, Race A, Bull AMJ, Amis AA. A comparative study of ‘isometric’ points for anterior cruciate ligament graft attachment. *Knee Surg Sport Traumatol Arthrosc* 2001;9:28-33.

39. Steadman JR, Dragoo JL, Hines SL, Briggs KK. Arthroscopic release for symptomatic scarring of the anterior interval of the knee. *Am J Sports Med* 2008;36:1763-1769.

40. Benjamin M, Moriggl B, Brenner E, Emery P, McGonagle D, Redman S. The “enthesis organ” concept: Why enthesopathies may not present as local insertional disorders. *Arthritis Rheum* 2004;50:3306-3313.

41. Dye SF, Vaupel GL, Dye CC. Conscious neurosensory mapping of the internal structures of the human knee without intraarticular anesthesia. *Am J Sports Med* 1998;26:773-777.

42. Gardner E. The innervation of the knee joint. *Anat Rec* 1948;101:109-130.

43. Breugem SJM, Haverkamp D. Anterior knee pain after a total knee arthroplasty: What can cause this pain? *World J Orthop* 2014;5:163-170.

44. van Raaij TM, de Waal Malefijt J. Anterior opening wedge osteotomy of the proximal tibia for anterior knee pain in idiopathic hyperextension knees. *Int Orthop* 2006;30:248-252.

45. Witvrouw E, Callaghan MJ, Stefanik JJ, et al. Patellofemoral pain: Consensus statement from the 3rd International Patellofemoral Pain Research Retreat held in Vancouver, September 2013. *Br J Sports Med* 2014;48:411-414.

46. Domenech J, Sanchis-Alfonso V, López L, Espejo B. Influence of kinesiophobia and catastrophizing on pain and disability in anterior knee pain patients. *Knee Surg Sport Traumatol Arthrosc* 2013;21:1562-1568.