

Patellofemoral Pain: An Enigma Explained by Homeostasis and Common Sense

William R. Post, MD, and Scott F. Dye, MD

Abstract

We present a rational, scientific, low-risk approach to patellofemoral pain (anterior knee pain) based on an understanding of tissue homeostasis. Loss of tissue homeostasis from overload and/or injury produces pain. Bone overload and synovial inflammation are common sources of such pain. Chondromalacia and malalignment are findings that almost always do not need to be “corrected” to relieve pain. Patience and persistence in nonoperative care results in consistent success. Surgery should be rare and done only after extensive nonoperative management and in the setting of clearly defined pathology. Rational surgical treatment is explained in the context of restoring tissue homeostasis to relieve pain.

Symptoms of patellofemoral pain (PFP) without a readily identifiable cause are perhaps the most common yet vexing clinical complaint heard by orthopedic surgeons worldwide. PFP typically occurs over the anterior knee, is often diffuse, and worsens with prolonged knee flexion and the use of stairs. Some prefer the term *anterior knee pain* (AKP) because we do not always know the pain is patellofemoral in anatomical origin; we know only that it is felt in the anterior knee. Pain is inherently and irreducibly a subjective phenomenon, a function of very discrete central nervous system activity within the sensory area of the contralateral cerebral cortex to the symptomatic knee. Pain is purely subjective and therefore by definition not objectively and consistently measurable between patients. Emotions

play a role in pain as well, and somatization resulting in knee pain is a well-known phenomenon, particularly in adolescent women related to stress or even abuse. There is no imaging study that can be used to guide the rational treatment of pain. The best we can do is to ask patients to draw pain diagrams, which provide useful information proven to correlate with areas of tenderness.¹

Although many have referred to patients with PFP as having patellofemoral pain *syndrome*, we reject that term, as it implies a clearly defined syndrome—a consistent set of symptoms, signs, and test results—that does not exist. More complex AKP cases, such as those involving major trauma, complex regional pain syndrome, or multiple operative procedures, are beyond the scope of this article, though many of the principles discussed are applicable. Surprisingly, despite decades of research and clinical experience with a vast number of patients, there still is controversy regarding the underlying etiology of the symptoms and the best, safest treatment.

Primum non nocere. First, do no harm. Let us understand how to reach that noble goal.

Our Hypothesis: Loss of Homeostasis Causes Pain

Homeostasis is a natural process of maintaining relatively stable and asymptomatic physiologic conditions in all organ systems under fluctuating environmental conditions. We hypothesize that pain is the result when load applied to musculoskeletal tissues exceeds the ability to maintain homeostasis. As in other organ systems, in musculoskeletal tissues homeostasis is restored and maintained with appropriate treatment. To illustrate this hypothesis, Dr. Dye coined the term *envelope of function* (EOF). A combination of magnitude and frequency of load causes loss of homeostasis; with respect to the knee, activity or

Authors' Disclosure Statement: The authors report no actual or potential conflict of interest in relation to this article.

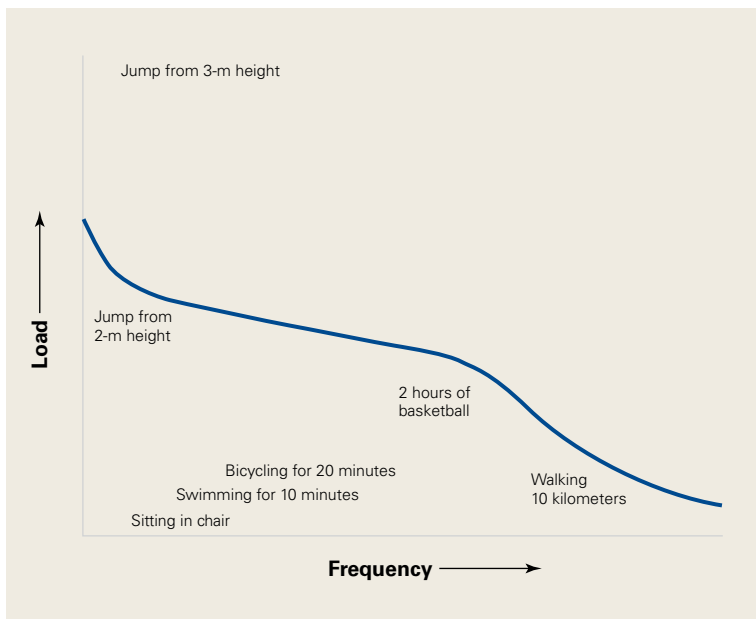


Figure 1. Envelope of function.

Reprinted with permission from *Clin Orthop Relat Res.*²

injury pushes it out of its acceptable EOF in which homeostasis is maintained (**Figure 1**).² When the total amount of load pushes into the zone of supra-physiologic overload, homeostasis is lost and pain occurs. With rest, time, and appropriate treatment, homeostasis can be restored. A simple example is muscle soreness that occurs after overuse and resolves over a few days. When the knee, or any

Take-Home Points

- Loss of tissue homeostasis from overuse or injury produces pain.
- In patients with AKP, treatment should begin with activity modification with the envelope of function; pain-free rehabilitation; an anti-inflammatory program of cold, nonsteroidal anti-inflammatory drugs, and sometimes steroid injection.
- Physical therapy should be done without painful exercise, otherwise it could be counter-productive.
- Patellofemoral syndrome and chondromalacia are not valid clinical diagnoses. A more specific diagnosis based on careful clinical evaluation to determine anatomic origin of pain will better direct treatment.
- Even when lateral retinacular tightness is identified as the probable source of pain, surgery is seldom required.

joint, operates outside its EOF longer or with increased magnitude of load, structural failure may occur. If lack of homeostasis causes pain, the solution to pain is to restore homeostasis.

The therapeutic recommendations that follow from this new biocentric paradigm of joint function are quite different from those associated with hypotheses attributing AKP to chondromalacia and malalignment. This new “common sense” approach, which never encourages treatment that makes symptoms worse, recognizes healing as a complex, rate-limited biological phenomenon that can take time to achieve, especially within a harsh and

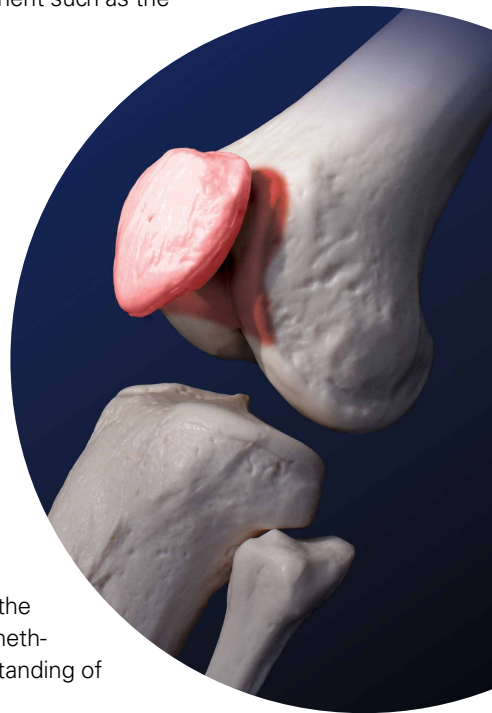
unforgiving biomechanical environment such as the human patellofemoral joint.

Traditional Explanations and Treatment Strategies

In traditional teaching, 2 causes of AKP have been prominent: chondromalacia patella (CMP) (softening of the articular surface of the patella) and malalignment of the extensor mechanism. Ironically, many of the worst AKP cases are iatrogenic, resulting from surgery to “correct” CMP and/or patellofemoral malalignment or maltracking. Even exercises encouraged by ill-informed physical therapists—such as excessive squats and lunges—can easily worsen AKP symptoms. We think the clinical failure of these traditional methods reflects a profound misunderstanding of the most common cause of AKP.

Chondromalacia Patella—Not the Problem

If chondromalacia is the source of AKP, what is it about conservative treatment that “cures” or even improves structurally softened articular cartilage? How can mere activity modification and exercise result in symptom resolution secondary



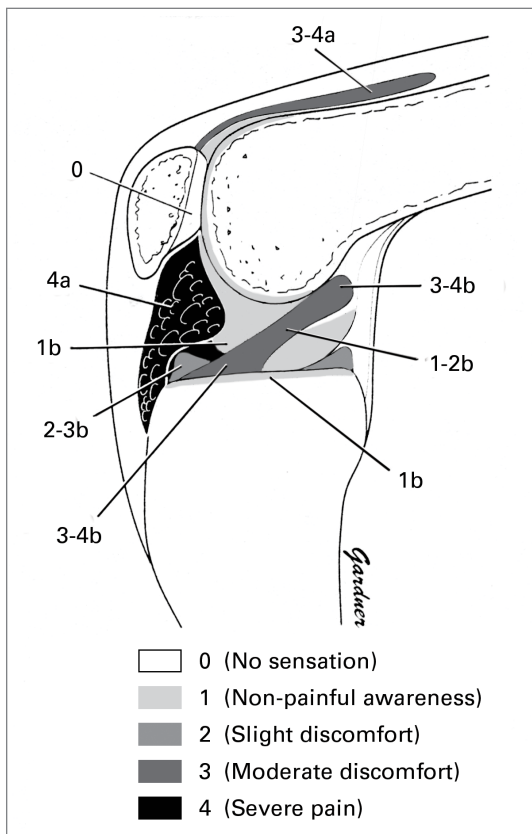


Figure 2. Neurosensory map of internal components of Dr. Dye's knee. Both knees had grade 3+ chondromalacia patella at time of arthroscopic examination. Reprinted with permission from *Am J Sports Med*.³

to improvement in cartilage structure? There is no evidence of this occurring. Nevertheless, patients with this "diagnosis" commonly respond to non-operative treatment.

Dr. Dye has had personal experience in the possible genesis of AKP in CMP. When he was 46 years old, he allowed his asymptomatic knees to be arthroscopically inspected, without intra-articular anesthesia, so that a neurosensory map of their internal components could be drawn (Figure 2).³ Surprisingly, the examination revealed grade 3+ CMP in both knees. During probing of the "pathologic" surfaces, he reported no sensation at all (Figure 3).⁴ Given that articular cartilage is aneural, this was no surprise. CMP alone cannot act as a nociceptive trigger. Although a deficient articular surface may transmit excess load to highly innervated subchondral bone, when excess load fails to cause a loss of homeostasis, symptoms are unlikely. Consistent and concurrent with this finding, each knee appeared normal on technetium 99m-methyl diphosphonate bone scan.



Figure 3. Probing of Dr. Dye's right patella without intra-articular anesthesia. He experienced no pain. Reprinted with permission from *Clin Orthop Relat Res*.⁴

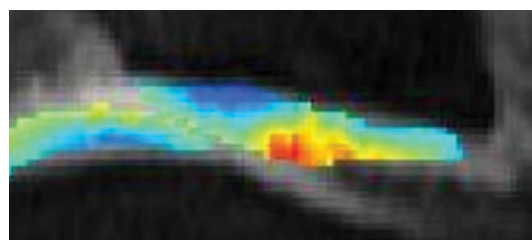


Figure 4. Dr. Dye's positive sagittal T1-p magnetic resonance imaging. Red indicates loss of proteoglycans; blue indicates normal proteoglycan content. Despite the documented diminished proteoglycan content of Dr. Dye's patella, he still remains asymptomatic. Image courtesy of Ben Ma, Department of Orthopedic Surgery and Department of Radiology, University of California, San Francisco.



Figure 5. Dr. Dye's normal, asymptomatic synovium being probed without intra-articular anesthesia. He experienced pain that elicited "involuntary verbal exclamations"

More than 18 years after this neurosensory mapping study, both knees are still asymptomatic, despite substantially reduced proteoglycan content of patellar articular cartilage bilaterally, recently detected with T1-p magnetic resonance imaging (MRI), the current favorite of many who use MRI to track early osteoarthritis (Figure 4). Remarkably, the musculoskeletal system can painlessly deliver millions of load transfer cycles during overt structural failure of one of its main components.⁴ We think Dr. Dye's experience is not an isolated case and that asymptomatic CMP is common.

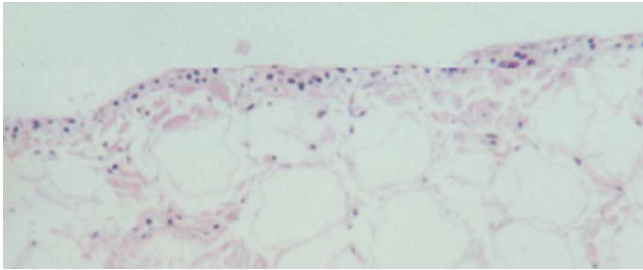


Figure 6. Normal synovial cells on fat-pad tissue (hematoxylin-eosin, original magnification $\times 100$).

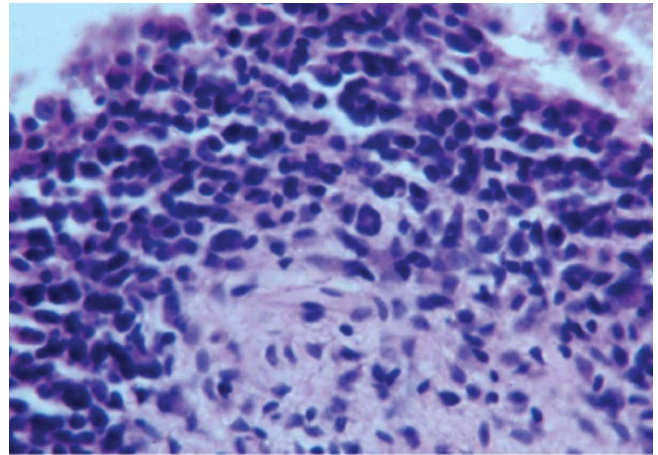


Figure 7. Synovitis (hematoxylin-eosin, original magnification $\times 150$).

Research data and clinical experience confirm that CMP does not in and of itself play a significant role in the genesis or resolution of symptoms in the typical patient with AKP.

Conversely, during the arthroscopy without intra-articular anesthesia, Dr. Dye discovered quickly and dramatically that the synovium and the fat pad were the most sensitive tissues. Light touch on unanesthetized synovial and fat-pad tissues evoked “involuntary verbal exclamations” (**Figure 5**).³ Since then, it has been personally and professionally apparent to him that synovial and fat-pad tissues are potent sources of AKP. Recurrent impingement of the synovium can result in hypertrophy producing thousands of cells instead of the normal few (**Figures 6, 7**). This hypertrophied tissue can impinge between the patella and trochlea as well as around the fat pad. This is a very common cause of persistent AKP in our experience most commonly independent of alignment and chondromalacia.

When MRI of a patient with AKP shows CMP be cautious not to conclude this structural condition is the direct cause of pain. When overload results in loss of homeostasis, breakdown products of damaged articular cartilage can contribute to symptomatic synovial inflammation. In addition, the damaged articular surfaces may fail to efficiently minimize joint friction and load transmission to subchondral bone. Chondromalacia alone, however, cannot be linked to pain.

Malalignment—Not Often the Problem

That brings us to the historically popular concept of patellofemoral “malalignment/maltracking” as a primary cause for AKP. Although this etiology

appeals to many in the orthopedic and physical therapy community,^{5,6} we and others⁷⁻¹⁰ reject the notion that it is common. What objective malalignment changes occur when a patient becomes asymptomatic without operative treatment? Imaging measures of malalignment do not change significantly after effective treatment. In studying patients with AKP in the mid 1980’s, Dr. Dye found no difference between 104 adults with PFP and 79 age- and activity-matched controls with respect to 9 objective indicators of malalignment, including quadriceps (Q) angle, congruence angle, sulcus angle, and subchondral sclerosis of the lateral patellar facet.

The clinical success of McConnell taping, which often produces instant pain relief by using tape to apply loads to the patella and peripatellar soft tissues, is sometimes cited as evidence that maltracking or malalignment is the cause of the pain. We disagree with that conclusion. This pain relief more likely results from relieving pressure and tension on sensitive soft tissues, including synovial, fat-pad, and retinacular tissues—equivalent to, say, using a finger to pull inflamed and swollen bitten cheek tissues away from the teeth, which might repetitively traumatize them. In both cases, healing is not spontaneous; but relieving the sensitive tissue of the exacerbating load is the common principle. We think subtle changes in the tension and impingement of synovial and fat-pad tissues can have profound effects on AKP. Pain relief with McConnell taping no more proves that the source of the pain is malalignment or maltracking than a finger pulling away inflamed and swollen cheek tissues proves that cheek pain is caused by malocclusion.

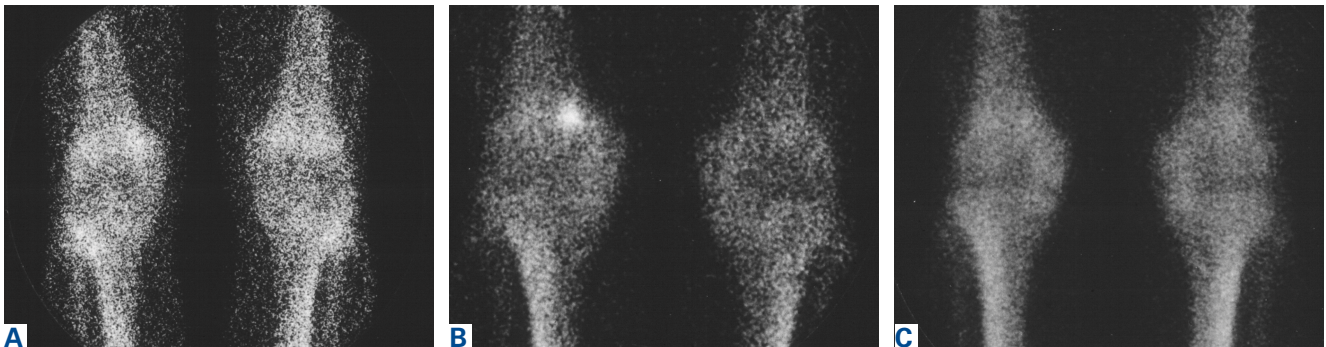


Figure 8. (A) Normal bone scan of both knees before needle penetration of the patella. (B) Intense uptake in patella 7 weeks after needle penetration. (C) Restoration of homeostasis documented by resolution of symptoms and normal bone scan 14 months later.

Reprinted with permission from *Instr Course Lect*.¹¹

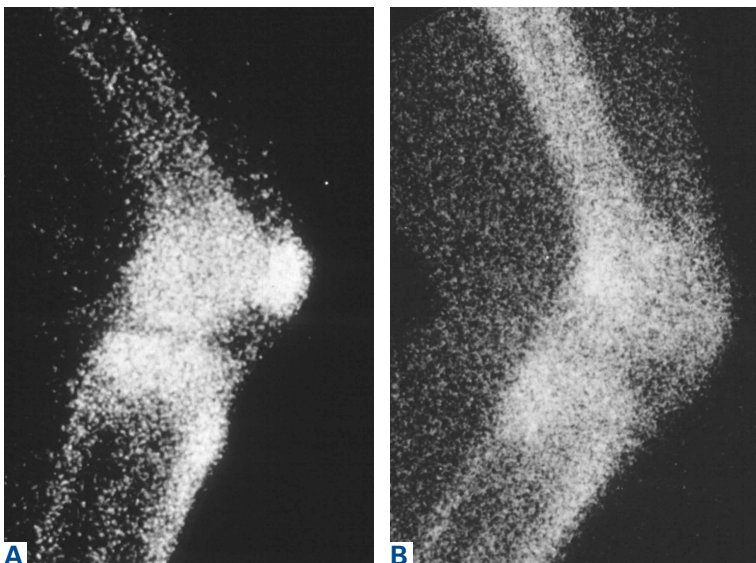


Figure 9. Bone scan (A) before and (B) after 4 months of conservative treatment, which resolved anterior knee pain.

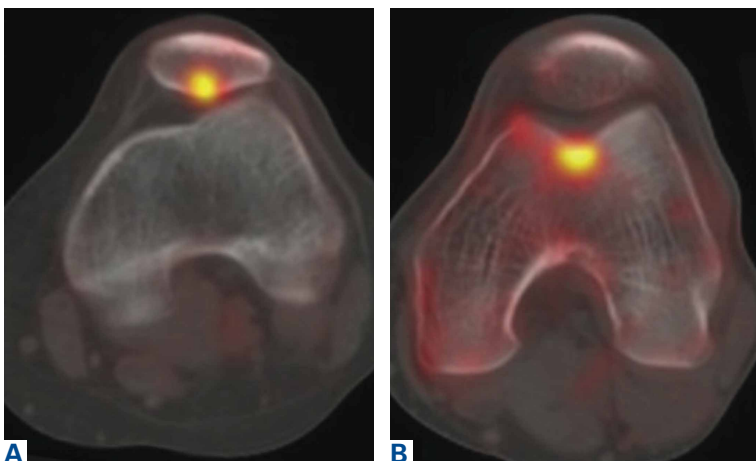


Figure 10. Positron emission tomography–computed tomography images from the Department of Radiology, Stanford University.

Reprinted with permission from *J Orthop Res*.¹²

Patellar Bone Overload—Part of the Problem

Patellar bone has been long assumed to be a source of AKP. To understand this better, Dr. Dye had one of his residents push a 15-gauge needle into the medial facet of his asymptomatic right patella to obtain real-time intraosseous pressure measurements as a control. This was done under local anesthesia, so no pain was felt as the needle entered the patella. However, when an arterial line was connected and flushed prior to pressure measurements, Dr. Dye experienced sharp lancinating pain. Patellar bone is richly innervated, and even mildly increased intraosseous pressure can produce severe symptoms. Dr. Dye’s patella was sore for about 7 months afterward. Bone scan was normal before this study, hot exactly at the needling site 7 weeks after patellar penetration, and normal 14 months later, after return of homeostasis and resolution of symptoms (**Figures 8A-8C**).¹¹

Loss and restoration of osseous homeostasis occur often in AKP patients whose positive patellar bone scans (focal or diffuse) show resolution to normal (homeostasis) after symptom dissipation (**Figures 9A, 9B**). In addition, loss of osseous homeostasis has been documented at higher resolution with positron emission tomography–computed tomography (**Figures 10A, 10B**).¹²

The Mosaic of Anterior Knee Pain

The densely innervated synovial, fat-pad, and patellar bone tissues are nociceptive sources of AKP in the absence of homeostasis. Also causing discomfort are other innervated but less frequently involved structures, including subcutaneous nerves, patellar tendon, quadriceps tendon, medial and lateral retinaculum, prepatellar bursae, and distal anterior thigh musculature. Any or all of these tissues can be involved at any given moment,

just as many tiles comprise a mosaic image. Each patient's mosaic of pathophysiology is unique, and individualized treatment is mandated.

Clinical Applications of Homeostasis and Common Sense

Essential points to be covered in the history include overuse, injury, weight gain, systemic illness (which may produce weakness and deconditioning), prior treatment (especially physical therapy) and response to medications or injections. In the case of prior surgery, preoperative and postoperative identification of the patient's exact symptoms can shed light on the underlying diagnosis and on any symptom changes resulting from treatment.

Sudden pain in the anterior knee can result in pain-mediated reflex quadriceps inhibition and the sensation that the knee is "giving way." Typically, patients describe the knee collapsing into flexion and when asked if their knee is "unstable" after experiencing such episodes they will readily say yes. However, such a knee is not "unstable" in the sense that there is patholaxity that might require surgery. This is a critical distinction to avoid tragically unnecessary surgery.

Careful evaluation for areas of tenderness may direct treatment to focal pathology, such as patellar or quadriceps tendinitis or tendinosis, pathologic medial parapatellar plica, or postoperative neuroma. Palpation and Tinel testing can uncover a neuroma or neuropathy of the infrapatellar branch of the saphenous nerve (**Figure 11**) that no other diagnostic tools can. This simple finding can lead to effective treatment of some chronic and recalcitrant cases. Both authors have seen multiply operated patients for whom subsequent palpation raised the suspicion of a neuroma or neuropathy. After Tinel testing, these patients exclaimed, "That's my pain!"

Poor flexibility, which increases tension and load in peripatellar soft tissues, is very common. In many cases, evaluation of hamstring, prone quadriceps, hip, and gastrocsoleus flexibility with contralateral comparison reveals a need to include stretching in a homeostasis-restoring program.

Insufficient muscular strength and endurance can also result in overload of patellofemoral bony and soft tissues. As all ground reaction force must be absorbed somewhere in the body, and since eccentric muscle contraction absorbs load, other tissues become overloaded if muscle function is insufficient to absorb enough force. Weakness of the hip and core have shown to respond to rehabilitation with resolution to AKP. Proximal weakness screening



Figure 11. Positive Tinel test with sharp pain experienced in distribution of infrapatellar branch of saphenous nerve with tapping on spot marked X.

with step-down or single-leg squat is important.

Joint effusion is an important finding indicative of objective intra-articular pathology and inflammation. Such inflammation may be from overuse resulting in loss of homeostasis (synovitis, cartilage breakdown, symptomatic arthrosis).

Screening examinations for hip and lumbar pathology are mandatory and take only a few minutes.

Treatment Options

Activity Modification

Avoid aggravating the problem. Consider this like a fire. If you are trying to put out a fire (AKP), would you throw sticks (increased activity/aggressive exercise) on it? Of course not. You would turn a hose on it (nonsteroidal anti-inflammatory drug [NSAID] regularly) or perhaps throw a bucket of water (steroid injection) on it. You would not throw gasoline (excessive exercise or activity) on it. Explaining to patients how to remain within their envelope by avoiding any activity that increases symptoms is crucial. No pain no gain is a lie from hell for patients with AKP. Don't throw sticks on the fire.

We are frustrated that patients with PFP are still often told by well-meaning therapists to perform exercises that end up substantially increasing symptoms. Patients are admonished to push forward with "quad strengthening" by any means necessary, including painful lunges and squats, which can exacerbate synovial and fat-pad impingement and put excessive tension on muscle

and tendon tissue, which is ill equipped to absorb the loads. Damaged tissues can usually return to pain-free biological homeostasis if given the opportunity and a reasonable mechanical environment.

Pain-free loading means that each of the hundreds of millions of sensory nerve endings is unperturbed, and is reporting, in effect, "I'm fine in my sector." Minor discomfort is inevitable, but real pain during activity, and exacerbations after activity, is activity outside the EOF. Strive for patients to have "clinically quiet" knees during activity. This common sense approach is often rewarded with dramatic recovery, over time, even in patients with severe AKP. In long-standing cases, patients may take months or even years to recover, but slow and steady progress should be expected. Later, these may be among your most grateful patients.

Cold Therapy

Cold therapy relieves pain, decreases swelling, slows the metabolic rate, is simple, and has few complications. Many AKP-related tissues are superficial, and the application of cold is logical and effective. However, we should not overdo it, either. Cold applied for 20 minutes once or twice daily is sufficient in most cases, at least initially. If it does not help resolve symptoms, it may be abandoned. Likewise, if a patient does not tolerate cryotherapy, it should not be demanded. Some patients respond better to the application of warmth, which is allowed within reason.

Anti-Inflammatory Medication

Inflammation clearly plays a role in the production of pain and swelling in the soft tissues of the anterior knee (synovium, fat pad, patella and quadriceps tendons/peritenon, and retinacular tissues). Consistent use of oral NSAIDs in the absence of medical contraindications can be valuable, and there are benefits to using mild oral NSAIDs (eg, solubilized ibuprofen 400 mg 2 times daily). Prescription NSAIDs should be used short-term, if possible, to avoid complications; long-term use requires medical supervision and laboratory testing. Oral steroids can be used in similar fashion.

Intra-articular steroids (eg, triamcinolone or methylprednisolone 40 mg with a few cubic centimeters of local anesthetic) can be very helpful in quickly reducing inflammation within synovial and fat-pad tissues. In addition, an intra-articular steroid injection is diagnostic when the pain goes away, even if only for the duration of the local anesthetic; this change indicates the pain must be coming from a structure

that is bathed by the intra-articular medication. Longer-term relief provides strong circumstantial evidence of causation related to intra-articular soft-tissue inflammation (loss of homeostasis) and not to chondromalacia or malalignment.

Physical Therapy

Therapy must be performed within the EOF as much as possible. Muscle soreness after a therapeutic workout is acceptable. There can easily be a lag time of 24 hours or more in the production of an activity-induced inflammatory enzyme spike. Therefore, when exercises are being done every other day, the rest days should also be kept well within the EOF. The patient must be essentially pain-free all the time, on exercise days and on rest days. Gentle stretching of tight muscles (especially quadriceps but also hips, hamstrings, and gastrocnemius) and strengthening of hips and core are encouraged. Gentle stretching on rest days is encouraged as well.

The physical therapist must teach the principles of moderating activities of daily living (ADLs) within the EOF (eg, safe use of stairs, safely getting in and out of chairs and vehicles), for it is in these ADLs that many symptomatic patients experience recurrent overload. Total load in ADLs and in therapy must remain within the EOF to maximize the chance of return to homeostasis. Exercise-induced substantial patellofemoral soreness, effusion, or increased temperature in the knee is not acceptable.

Imaging

Advanced imaging in AKP can be a contentious subject. It is too easy to assume images hold the answers. A finding of CMP or alignment abnormality must be viewed with caution, as usually it is not an indication for patellofemoral surgery. You are treating a patient, not a picture. You must be responsible to integrate all available data (history, physical examination, imaging, response to treatment, etc) to make an accurate diagnosis. Always inspect all the imaging data yourself. Do not "push in the mental clutch" but rather do the challenging work of putting all the clinical pieces of the puzzle together to reach the right answer. Do not let the radiologist make the diagnosis!

Radiographs

It is imperative to obtain good-quality radiographs, including axial radiographs of the patella in early flexion, to check for evidence of arthrosis and other joint pathology that may be producing pain. Dr. Post always obtains bilateral knee radiographs to help

understand the degree of any arthrosis or malalignment in the contralateral asymptomatic knee. The information in bilateral radiographs is also instructive for patients. Knowing that the contralateral knee shows the same radiographic changes, or even more, helps them understand that the structural factors as imaged do not dictate symptoms. More advanced or extensive imaging is not needed unless appropriate and patient therapy reaches a stalemate.

Bone Scans

In recalcitrant patients with persistent pain, a bone scan provides sensitive imaging of osseous metabolic activity and thereby clarifies the etiology of the pain. A negative scan rules out the bone as a significant cause, freeing the clinician to concentrate solely on the soft tissues. In a way that MRI can miss, a positive bone scan identifies specific regions that have lost osseous homeostasis and are being overloaded. Microscopically, these regions' changes are very similar to the abnormal bone remodeling that occurs in early-stage stress fractures. Whether focal or diffuse, a positive bone scan means symptoms likely will take longer to reverse than is the case with a negative scan. Often, the stark findings of a positive bone scan can grab the patient's attention and improve understanding and compliance. Focal inferior pole uptake is the most difficult pattern to reverse, perhaps because it may represent the most extreme biomechanical environment of the patellofemoral joint. In Dr. Dye's experience, patients with this pattern may often require drilling of the inferior pole to achieve restoration of tissue homeostasis.

Magnetic Resonance Imaging

MRI can be useful, though scans are commonly read as normal. In some cases, MRI evidence of



Figure 12. T2-weighted magnetic resonance imaging: (A) sagittal view shows synovitis effusion; (B) axial view shows neovascularization of fat pad.

tendinopathy and other intra-articular pathology can direct both operative and nonoperative treatment of AKP. Carefully look for evidence of soft-tissue impingement—such as mild synovial swelling, low-grade effusion, and neovascularization of the fat pad—as in many cases it exists, and has been missed by the radiologist (**Figures 12A, 12B**). View the images yourself and, if necessary, in consultation with a radiologist.

When Surgery Is Needed: General Principles

Although the majority of patients with AKP do not need surgery, some do. Think of surgery as a tool used to create an environment in which homeostasis may be restored. Arthroscopy and meticulous débridement may be used to treat recalcitrant focal synovitis or fat-pad hypertrophy—or focal chondral pathology (eg, unstable flap of articular cartilage) that has produced mechanical symptoms with secondary inflammation. A well-localized area of patellar tendinosis may respond to either arthroscopic or open débridement. A true mechanical alignment abnormality may produce

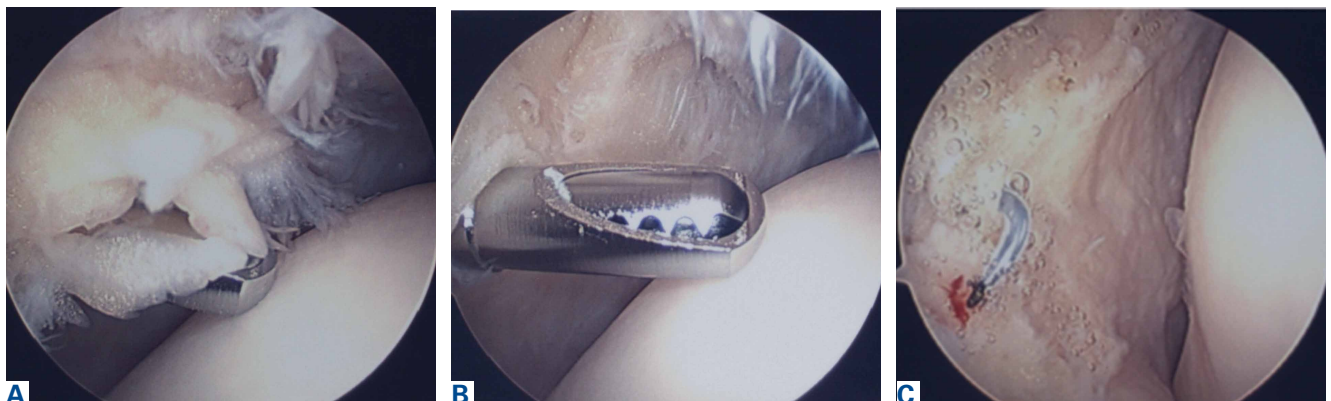


Figure 13. (A) Hypertrophic synovial tissue in position where it could impinge between articular surface and capsule. (B) After meticulous resection of pathologic synovial tissue. (C) Always be certain to assure complete hemostasis.

focal overload to such a degree that the most complete nonoperative programs cannot overcome the loss of homeostasis. In such a case, imaging studies that precisely document overloaded areas and associated malalignment must make sense given the clinical picture, and then must be used in developing a rational surgical plan for unloading bone and soft-tissue pathology to create a mechanical and biological environment for healing and return to homeostasis. At times, the articular damage may be so severe that patellofemoral arthroplasty is the best choice. The exact indications for these procedures are well described elsewhere.¹³

Surgery for Patients With PFP Caused by Recalcitrant Synovitis

As this type of surgery is not often covered in the literature, we offer some treatment pearls here. Arthroscopy for persistent focal synovitis should not be approached lightly; though the mechanics of removing abnormal inflamed synovial tissue may be straightforward, perioperative management and long-term postoperative management are not. The patient must be mentally prepared for the process; blood-thinning agents, fish oil, and turmeric must be discontinued; and hemostasis must be meticulous (**Figures 13A-13C**). A substantial hemarthrosis, which can be very painful, represents a major setback in homeostasis restoration. To ensure there is no active bleeding immediately after surgery, Dr. Dye keeps a small drain in the patient's knee for at least a couple of hours. In a patient with active bleeding, the drain can stay overnight; if there is no bleeding, the drain can be removed before the patient is discharged. The patient must be prepared to take it easy for a while after the procedure to allow cellular repopulation of the raw surface created when the inflamed synovium was removed. As complete restoration of joint homeostasis can take several months, the patient and surgeon must remain patient. Ice, NSAIDs if needed, and rehabilitation within the EOF ensue.

Conclusion

The history of medicine has included many misunderstandings of cause and effect. Trephination was used for headaches, leeches for fever, and, more recently, antacids for *Helicobacter pylori* caused duodenal ulcers. Stimulated by the enigma of AKP, we think our common sense way of thinking about tissue homeostasis in the musculoskeletal system

represents an emerging orthopedic biological paradigm that is applicable to the entire body. We should let the remarkable capacity of vertebrate biology do the "heavy lifting" of healing. The traditional orthopedic emphasis on structure and alignment has a role, but we see it as complementary and secondary to the biological paradigm and find that the evidence presented herein supports our contention. The answer is seen only when one looks beyond the viewbox.

Primum non nocere. Your patients will be most grateful.

Dr. Post is in private practice with Mountaineer Orthopedic Specialists, and a Team Physician, West Virginia University, Morgantown, West Virginia. Dr. Dye is Associate Clinical Professor, Department of Orthopedic Surgery, University of California, San Francisco, California.

Address correspondence to: William R. Post, MD, 2195 Cheat Rd., Suite 2, Morgantown, WV 26508 (tel, 304-594-0456; email, wpost@wvortho.com).

Am J Orthop. 2017;46(2):92-100. Copyright Frontline Medical Communications Inc. 2017. All rights reserved.

References

1. Post WR, Fulkerson J. Knee pain diagrams: correlation with physical examination findings in patients with anterior knee pain. *Arthroscopy.* 1994;10(6):618-623.
2. Dye SF. The knee as a biologic transmission with an envelope of function: a theory. *Clin Orthop Relat Res.* 1996;(325):10-18.
3. 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(6):773-777.
4. Dye SF. The pathophysiology of patellofemoral pain: a tissue homeostasis perspective. *Clin Orthop Relat Res.* 2005;436:100-110.
5. Grelsamer RP. Patellar malalignment. *J Bone Joint Surg Am.* 2000;82-A(11):1639-1650.
6. Powers CM. The influence of altered lower-extremity kinematics on patellofemoral joint dysfunction: a theoretical perspective. *J Orthop Sports Phys Ther.* 2003;33(11):639-646.
7. Sanchis-Alfonso V. *Anterior Knee Pain and Patellar Stability.* London, England: Springer-Verlag; 2006.
8. Post WR. Anterior knee pain: diagnosis and treatment. *J Am Acad Orthop Surg.* 2005;13(8):534-543.
9. Dye SF. Patellofemoral pain current concepts: an overview. *Sports Med Arthrosc Rev.* 2001;9(4):264-272.
10. Dye SF, Staubli HU, Beidert RM, Vaupel GL. The mosaic of pathophysiology causing patellofemoral pain: therapeutic implications. *Oper Tech Sports Med.* 1999;7:46-54.
11. Dye SF, Chew MH. The use of scintigraphy to detect increased osseous metabolic activity about the knee. *Instr Course Lect.* 1994;43:453-469.
12. Draper CE, Fredericson M, Gold GE, et al. Patients with patellofemoral pain exhibit elevated bone metabolic activity at the patellofemoral joint. *J Orthop Res.* 2012;30(2):209-213.
13. Post WR, Teitge R, Amis A. Patellofemoral malalignment: looking beyond the viewbox. *Clin Sports Med.* 2002;21(3):521-546.