

# Patellofemoral Pain Current Concepts: An Overview

Scott F. Dye, M.D.

---

**Summary:** The conceptualization of factors responsible for the etiology of patellofemoral pain are changing. In the recent past, chondromalacia and malalignment were believed to be most important factors relative to the genesis of anterior knee pain symptoms. However, new evidence from Europe and North America indicates other factors may be more important, such as overuse of anatomically normal patellofemoral structures, leading to the possible loss of both osseous and soft tissue homeostasis. The range of painless loading compatible with tissue homeostasis of a joint without causing structural or physiologic injury is termed the “envelope of function.” Restoration of the envelope of function as safely and predictably as possible is viewed as the primary underlying principle of treatment for patients with patellofemoral pain. **Key Words:** Patellofemoral—Pain—Tissue—Homeostasis—Chondromalacia—Malalignment.

---

Despite recent advances in the understanding and treatment of many musculoskeletal conditions, patients with symptoms of anterior knee pain remain an orthopedic enigma. Disconcertingly, the worst cases of patellofemoral pain and dysfunction often are in patients who have had multiple operative procedures for symptoms that initially were only mild anterior knee discomfort.<sup>1</sup> Perhaps with the exception of surgery for low back pain, no other area in orthopedic surgery has an iatrogenic failure rate as great as that for patients with anterior knee pain. The lack of a safe and predictable approach for these patients represents a genuine orthopedic concern and implies a profound lack of understanding of the causative factors associated with the genesis of patellofemoral pain. The currently accepted treatment approach to patients with patellofemoral pain is based on the concept that observable structural and biomechanical factors are primarily responsible for the symptoms, so treatment addressing those factors should be curative.<sup>2,3</sup>

Two main factors traditionally have been thought to be of casual significance in the genesis of anterior knee pain, including the presence of chondromalacia and/or malalignment of the patellofemoral joint. This com-

monly held belief in the structural and biomechanical genesis of anterior knee pain has served as a justification for treatments that have resulted in worsening of symptoms, including aggressive physical therapy with extension of the knee against resistance to strengthen the vastus medialis obliquus to correct “maltracking.” In addition, the use of various operative procedures such as the lateral release, aggressive chondroplasties, and major proximal and distal realignments have not infrequently resulted in iatrogenic injury, including medial patellar dislocation and reflex sympathetic dystrophy. This article focuses on the majority of patients with patellofemoral pain—those without an overt, easily identifiable cause such as a fracture or advanced arthrosis. Symptoms of patellofemoral instability, exemplified by recurrent patellar dislocation/subluxation, are not within the scope of this article or symposium.

## CHONDROMALACIA

The presence of chondromalacia once was thought to be so commonly associated with anterior knee discomfort that it became the accepted clinical diagnosis for symptoms of patellofemoral pain.<sup>4–6</sup> However, studies have shown that even advanced chondromalacic changes can be totally asymptomatic and that patients with normal appearing articular cartilage can experience substantial anterior knee pain.<sup>7–9</sup> Despite these observations, aggressive chondroplasties still are performed within the patellofemoral joint, including drilling of bone, mosaic-

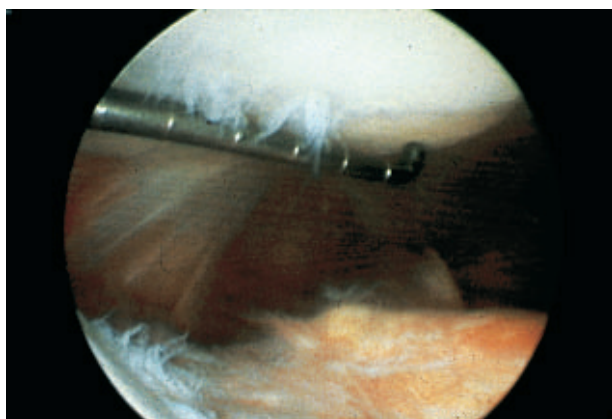
---

From the Department of Orthopedics, University of California, and the Department of Orthopedic Surgery, California Pacific Medical Center, Davies Campus, San Francisco, California.

Address correspondence and reprint requests to: Scott F. Dye, M.D., 45 Castro Street, Suite 117, San Francisco, CA 94114. E-mail: [SFDyeM.D.@aol.com](mailto:SFDyeM.D.@aol.com)

plasties, and autogenous chondrocyte transplantations. However, such procedures have not demonstrated long-term benefit and have resulted in worsening patellofemoral symptoms in many patients. The mere presence of chondromalacia in a patient with patellofemoral pain does not prove that the observed structural damage of articular cartilage is causing these symptoms of discomfort. This author has patellae with documented grade III chondromalacia that are totally asymptomatic, even to direct probing without intraarticular anesthesia<sup>8</sup> (Fig. 1).

Articular cartilage is well known by histologic examination to be aneural, so the absence of sensation to palpation should not be surprising. However, despite this academic understanding, the absence of sensation in the region of advanced structural changes of my own patellar articular cartilage was startling. This finding has influenced my subsequent surgical approach to the presence of chondromalacia in patients with anterior knee pain. I now am much less aggressive regarding surgical debridement of observed damage of articular cartilage than I had been. This is not to say that the presence of chondromalacia cannot be at least indirectly a factor in the genesis of anterior knee pain. Thinning of cartilage can lead to excessive loading of subchondral bone, which because it is an innervated structure, remains a potential source of pain, as noted by Radin<sup>10</sup> in an earlier perceptive article. In addition, the breakdown products of fibrillated cartilage can stimulate cytokine production and associated inflammatory biochemical events within innervated synovium, leading to chemical irritation of nerves and the perception of pain.<sup>11</sup> The frequent swelling of synovial tissues also leads to an increased susceptibility to mechanical irritation (impingement).



**FIG. 1.** Probing of the author's unanesthetized right patella, showing grade III chondromalacia. No sensation was experienced during palpation of the articular cartilage. (Reprinted with permission.)

## MALALIGNMENT

The concept that is perhaps most widely accepted is that some form of malalignment between the patella and femur, even though it may be subtle, is of primary causal significance in the genesis of anterior knee pain.<sup>3,12</sup> This view, although biomechanically appealing to many orthopedic surgeons and physical therapists, has not, in this author's experience, held up to close scientific scrutiny or a dispassionate logical analysis. Even worse, the belief in the concept of malalignment as a necessary, but not always sufficient, condition for the presence of patellofemoral pain has served as the justification and even encouragement for the unwary orthopedic surgeon to perform operative procedures designed to correct the supposed malalignment. Proponents of the malalignment theory also believe that if such a malalignment-oriented procedure fails, it may be because of an overcorrection or undercorrection, again encouraging additional surgical perturbation of an already iatrogenically injured joint. Such a perspective has led to well-meaning but ill-advised multiple surgical attempts to get the patellofemoral position "just right" only to further damage and traumatize this region of the knee.

This author's suspicion regarding the lack of importance of the observable indicators of patellofemoral malalignment dates nearly 20 years, when our research group at Letterman Army Medical Center in San Francisco performed a clinical and imaging evaluation of patients with patellofemoral pain compared with that of control subjects. Several supposed indicators of malalignment, including a high Q angle, a high congruence angle, and the presence of a meniscus of osseous sclerosis of the lateral facet on axial radiographs, were not found with a statistically greater frequency in the symptomatic population than in the asymptomatic control group.<sup>13</sup>

More recent work by Thomee et al.<sup>14</sup> confirms this view that factors other than malalignment (i.e., overuse of anatomically normal patellofemoral joints) are the cause of most symptoms of patellofemoral pain. However, one of the major proponents of the malalignment theory states that he has very rarely seen patients with normal patellofemoral alignment who have symptoms caused by overuse.<sup>3</sup> Anatomic research by Stäubli et al.<sup>15</sup> has thrown into question the primary method of determining the presence of malalignment, which for years has been based on the measurement of *osseous* landmarks of the patellofemoral joint. They have shown that the articular cartilage morphology does not necessarily match the osseous morphology, as will be addressed in separate articles in this symposium. Thus, when one de-

termines that tilting of the patella is present by measuring such osseous landmarks, the cartilage surfaces may in fact be mating perfectly. If one then performs an operation to untilt the patella to achieve osseous radiographic normalcy—supposedly correcting the malalignment—one may in fact be creating an iatrogenic malalignment, which may result in worsening of symptoms.

### LOGICAL ANALYSIS

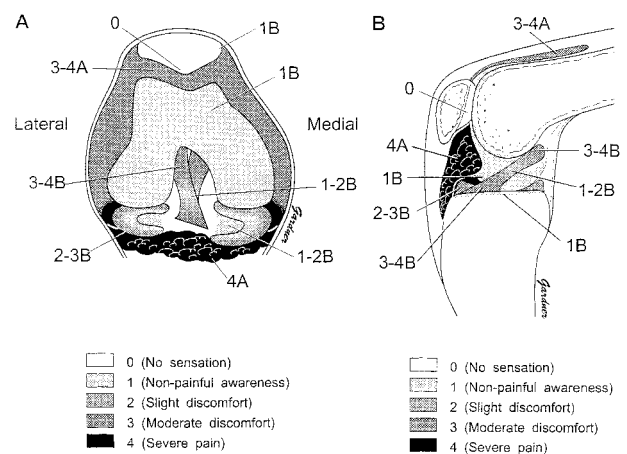
If the presence of observable factors of malalignment is so important in the genesis of anterior knee pain, why does one find patients with bilateral radiographically determined patellofemoral malalignment (patellar tilts) with only unilateral symptoms? Why do more than 90% of patients with anterior knee pain who have a diagnosis of malalignment as the cause have a successful response to conservative therapy, even though there has been no documentation of long-term restoration /or correction of the supposed causative underlying indicators of malalignment (for example a high Q angle or a shallow trochlea). Patients have lived and adapted to their unique biomechanical factors their entire lives. Just because one examines them at a time when symptoms are present does not mean that these so-called indicators of malalignment are causal in the genesis of pain. Even successful conservative treatment, such as the pain-relieving patellofemoral taping technique of McConnell,<sup>3</sup> does not necessarily work by correcting malalignment but may in fact decrease patellofemoral pain by mechanically relieving (unpinching) swollen and irritated peripatellar tissues, which eventually heal, resulting in long-term pain relief without a permanent change in patellofemoral alignment characteristics.

In addition, supposed secondary indicators of the so-called excessive lateral pressure facet syndrome (presumed to be the most common form of malalignment), including the radiographic findings of perpendicularization of lateral facet trabeculae and a meniscus of osseous sclerosis of the lateral facet, were not found to be present at a greater frequency in the symptomatic group than in the asymptomatic control group in our study.<sup>13</sup> The malalignment theory also does not explain the variability of patellofemoral symptoms in the same patient at different times, including the presence of sharp pain on occasion and then dull aching pain on another, as well as the possible absence of pain. The so-called movie sign (or deep patellar aching) with prolonged flexion also is not explained by the malalignment theory.

What can provide a better explanation? During the past nearly 20 years, our research group in San Francisco

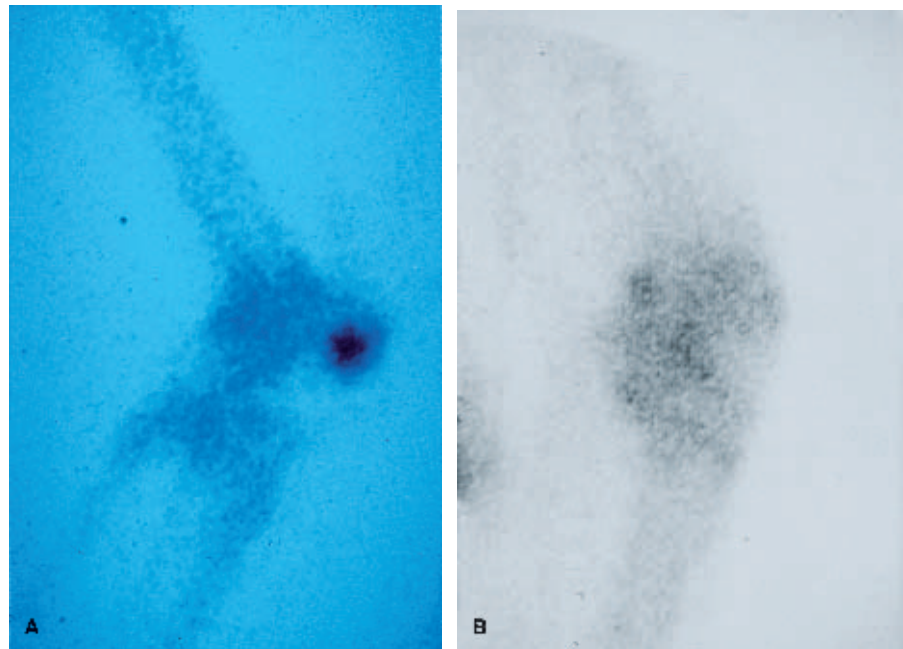
has been attempting to address these questions. One of the confusing variables regarding patellofemoral pain is that some patients have clinically significant malalignment that responds to malalignment-oriented treatment, including a lateral release. However, in my experience, the numbers of these patients are relatively few. An alternative perspective to the malalignment (and chondromalacia) theory must address several issues, including what is the source of the patellofemoral pain; what tissues are involved; what are the pathophysiologic factors relative to the nociceptive neurologic output; why do some patients have patellofemoral pain with no observable structural abnormalities; why do unilateral symptoms exist in patients with similar structural characteristics of both knees; what accounts for the variability of symptoms in the same individual from sharp to dull to absence of pain; and what accounts for the presence of the movie sign?

What are the potential sources of pain? This author has experienced sharp lancinating pain secondary to transiently increased intraosseous pressure experimentally produced within the right patella through a 15-gauge Jamshidi needle (placed painlessly within the medial facet under local anesthesia).<sup>16,17</sup> In a separate study, designed to provide a neurosensory map of the internal structures of the knee by direct palpation without intra-articular anesthesia, this author noted that even light touch of unanesthetized peripatellar synovium was quite painful<sup>8</sup> (Fig. 2). Histologic observation reveals the distribution of nerves in all peripatellar tissues, with the



**FIG. 2.** Schematic representation of a neurosensory map of the human knee obtained by palpation of intraarticular structures of the author's knees without anesthesia. Perceived pain ranged from 0 (no sensation) to 4 (severe pain), and either (a) accurate spatial localization or (b) poor spatial localization. Palpation of synovium elicited severe localized pain (4a) and similar palpation of patellar articular cartilage was completely without sensation. (A) Coronal representation. (B) Sagittal representation. (Reprinted with permission.)

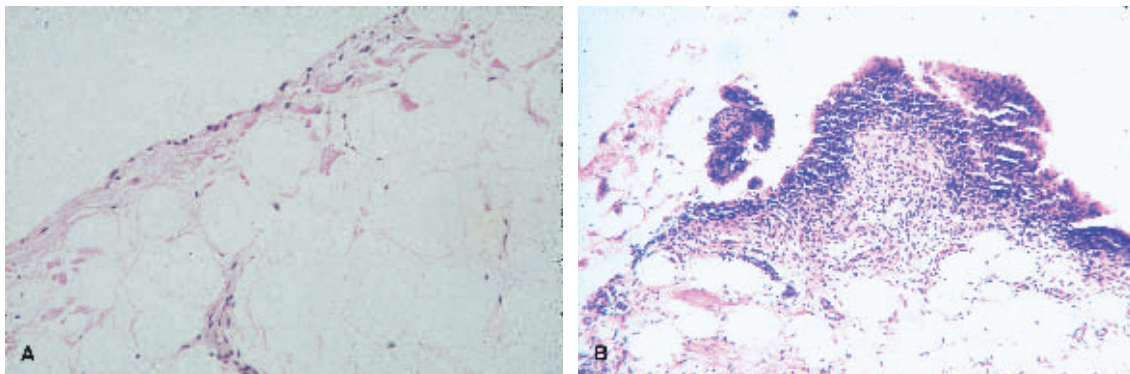
**FIG. 3.** **A.** Positive patellar bone scan of a 32-year-old woman with patellofemoral pain and no evidence of malalignment, representing loss of osseous homeostasis. **B.** Repeat bone scan of the same patient 4 months later, at the time of symptom resolution, with a conservative therapeutic program showing restoration of osseous homeostasis.



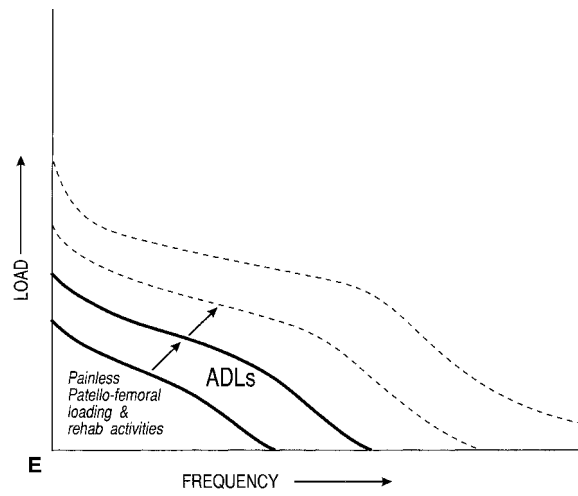
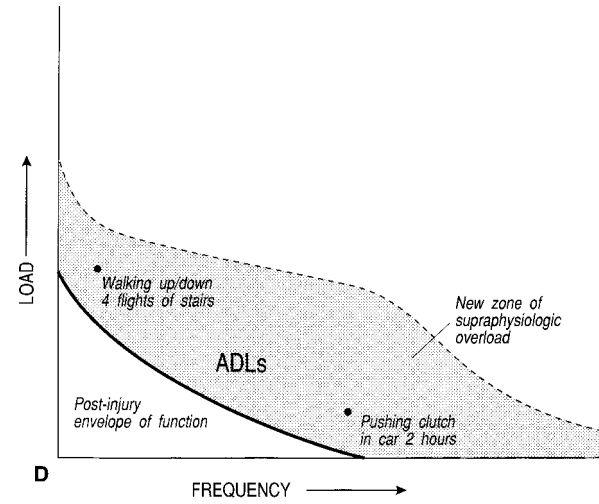
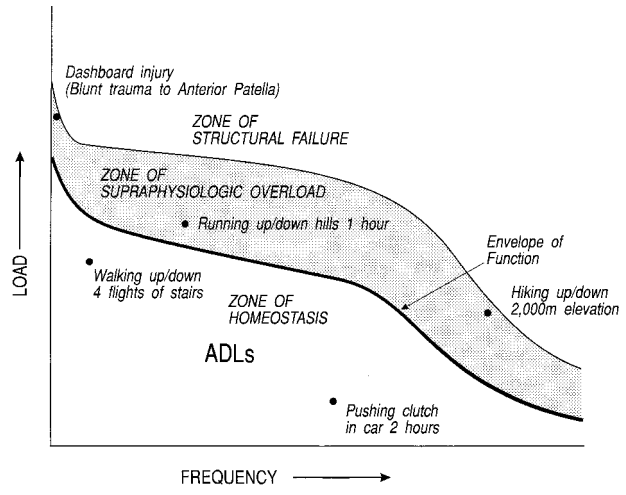
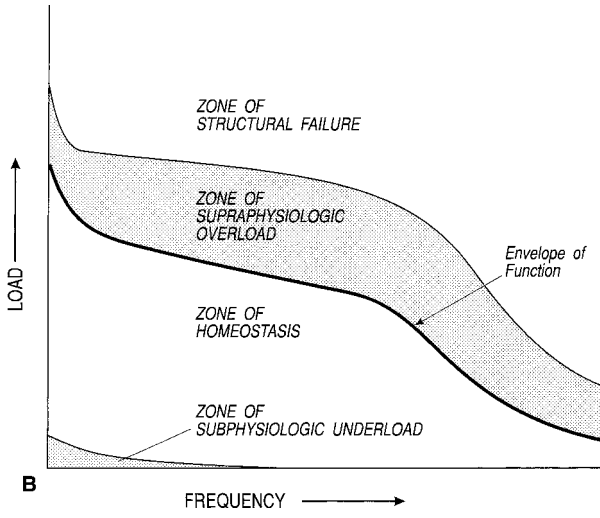
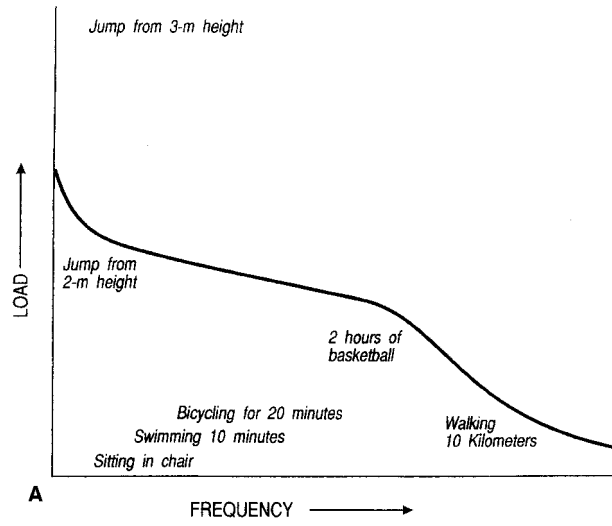
exception of articular cartilage.<sup>18</sup> An article relative to this topic by Biedert and Kernen is provided in this symposium. In addition, Wojtys et al.<sup>19</sup> have shown the presence of the neuroactive peptide associated with pain perception (substance-P) within the nerves of symptomatic peripatellar synovium. Through these findings, one can logically assume that the perception of patellofemoral pain, in most instances, is a function of nociceptive neurologic output of any combination of innervated patellar and peripatellar tissues. The most likely candidates for the genesis of nociceptive output resulting in the perception of patellofemoral pain, in this author's view, are peripatellar synovium and related soft tissues and the intraosseous environment of the patella. It also is important to know that perceived anterior knee pain can arise

from nonpatellofemoral sources, such as referred pain from an injury to the saphenous nerve or degenerative arthrosis of the ipsilateral hip. Andrish has made the astute observation that in some teenage female patients the perception of pain may represent somatization secondary to possible physical or sexual abuse (Andrish JT. Personal communication. Cleveland Clinic, Cleveland, OH, 2001).

Pain denotes nerve irritation, either mechanically, as through pinched synovium and increased intraosseous pressure, or chemically, as through the presence and production of cytokine enzymes. Pain connotes loss of tissue homeostasis.<sup>20</sup> Normal asymptomatic living structures (e.g., bone, ligaments) can be described as having the characteristic of tissue homeostasis, with constant main-



**FIG. 4.** **A:** Biopsy of normal peripatellar synovium and fat pad showing a thin layer of synovial cells and deeper oval fat cells. **B:** Biopsy of peripatellar synovium of a patient with patellofemoral pain showing thickening, inflammation, and lymphocyte infiltration of the synovium.



**FIG. 5.** **A:** Graph representing the envelope of function for an athletically active young adult. The letters represent loads associated with different activities. All of the loading examples, except *B*, are within the envelope for this particular knee. The shape of the envelope of function represented here is an idealized theoretical model. The actual loads transmitted across an individual knee under these different conditions are variable and attributable to multiple complex factors, including the dynamic center of gravity, the rate of load application and the angles of flexion and rotation. The limits of the envelope of function for the joint of an actual patient probably are more complex. (Reprinted with permission.) **B:** Graph showing the four different zones of loading across a joint. The area within the envelope of function is the zone of homeostasis. The region of loading greater than that within the envelope of function but insufficient to cause macrostructural damage is the zone of suprathysiologic overload. The region of loading great enough to cause macrostructural damage is the zone of structural failure. The region of decreased loading over time resulting in a loss of tissue homeostasis is the zone of subphysiologic underload. (Reprinted with permission.) **C:** Subphysiologic loads outside the envelope: a dashboard injury, running up hill 1 hour, and hiking downhill 2000 meters. (Reprinted with permission.) **D:** Diminished envelope of function after suprathysiologic patellofemoral loading showing that activities of daily living and activities such as climbing four flights of stairs and pushing a clutch in a vehicle for 2 hours have become suprathysiologic loads, leading to recurrent loss of tissue homeostasis and continuance of peripatellar symptoms. (Reprinted with permission.) **E:** Incremental expansion of the diminished envelope of function by restricting patellofemoral loading to within the envelope. (Reprinted with permission.)

tenance of normal physiologic processes at the cellular and molecular level, as well as restoration of normal physiologic processes after perturbation or injury (i.e., healing).<sup>21-23</sup> This is perhaps best manifested in living bone tissue through the use of technetium scintigraphy.<sup>16,17</sup>

The perception of pain clearly evolved throughout hundreds of millions of years of evolution as a negative feedback loop system designed to detect the presence of dangerous conditions and noxious stimuli to alert the central nervous system of these conditions. Posttraumatic inflammation after tissue overload (e.g., strain, contusion) produces a complex biochemical cascade, including the production of cytokines that irritate (stimulate) nerve endings, resulting in the perception of pain. Without this negative feedback loop system, musculoskeletal components are at constant risk of excessive (suprathysiologic) overload that could lead to eventual permanent structural damage. Just such a case is exemplified by patients born with congenital insensitivity to pain, who often experience advanced degenerative changes of their joints by a young age.<sup>24</sup> The destructive changes of Charcot joints after certain conditions that affect the neurologic system in adults also reflect failure of the negative feedback loop system by diminishing the perception of pain.<sup>25</sup>

The addition of a method to manifest metabolic characteristics of living bone (technetium 99m methylene diphosphonate scintigraphy) to our clinical and radiographic evaluation of patients with patellofemoral pain led to the concept of the loss of tissue homeostasis as an important, yet covert, factor in the genesis of symptoms. We noted that about one-half of patients with patellofemoral pain demonstrated increased patellar uptake, compared with only 4% of the control subjects ( $p > 0.001$ ).<sup>17</sup> The increased osseous metabolic activity of the patella, detected by the bone scan, was biopsy proven to represent increased remodeling activity of bone compared with controls, without evidence of tumor or infec-

tion. Many with intensely positive scintigraphic activity were histologically identical to the findings of an early stage of a stress fracture, manifesting cutting cones and Howship's lacunae.<sup>16</sup> Patients with anterior knee pain and a positive bone scan were treated conservatively and followed up clinically. When these patients underwent follow-up imaging, it was noted that many who experienced resolution of painful symptoms also demonstrated resolution of the bone scan to normal activity<sup>26</sup> (Fig. 3). Thus, the findings of a positive patellar bone scan came to be interpreted as representing a loss of osseous tissue homeostasis and a subsequent normal bone scan as demonstrating restoration of osseous homeostasis. The use of technetium scintigraphy was viewed as a method to sensitively manifest the presence or absence of tissue homeostasis of the osseous aspect of the patella (and trochlea), which often matched well with the presence of pain and its resolution.<sup>16</sup> The bone scan illuminated well the osseous metabolic tile of the mosaic of possible pathophysiologic processes accounting for the genesis of patellofemoral pain. Because only one-half of the patients with patellofemoral pain manifested loss of osseous homeostasis, it was clear that other factors were present to account for the genesis of symptoms in many patients in addition to loss of osseous homeostasis.<sup>17</sup> The innervated peripatellar soft tissues were the obvious nonosseous potential sources of nociceptive output, which is supported clinically by the presence of tenderness to palpation of, for example, the patellar tendon, retinaculum, or synovium/capsule. It also was logical to interpret the presence of pain and tenderness of the peripatellar soft tissues as representing the symptomatic loss of homeostasis of these tissues, which we later proved in synovium with biopsy specimens compared to those of control subjects<sup>21</sup> (Fig. 4). The tissue homeostasis perspective appeared to explain, with much greater clarity, the often variable nature of patellofemoral pain from patient to patient that is obviously lacking in the malalignment theory. The variable nature of a given patient's symp-

toms on a given day can be seen as, in essence, a function of the variable mosaic of loss of tissue homeostasis of innervated patellar and peripatellar tissues.<sup>21</sup> I believe that sharp pain most often represents mechanical pinching of peripatellar synovium—a tissue that is documented to be well innervated.<sup>8</sup> Such mechanical pinching episodes occur frequently in knees without any evidence of patellofemoral malalignment. The presence of an effusion also is consistent with synovitis as part of the mosaic of pathophysiologic processes that can account for patellofemoral pain. The dull aching that one experiences after exercise is understandable as overload and subsequent irritation of innervated tissues. The absence of pain reflects loading that is nonirritating to those innervated tissues. The movie sign theoretically is best explained by possible transient increases in intraosseous pressure as a function of slight venous outflow obstruction that resolves rapidly with extension or ambulation.

One must also be aware of seemingly confounding variables not directly related to patellofemoral loading, such as the perception of patellar pain secondary to changes of barometric pressure; this phenomenon may be attributable to barosensitivity of the intraosseous environment of the sensitized patella.<sup>20</sup> In addition, the presence of painful neuromas, as demonstrated by Fulkerson and Hungerford<sup>2</sup> and Sanchis-Alfonso and colleagues<sup>27,28</sup> in the retinacula and other peripatellar tissues, also can play a role in the genesis of patellofemoral symptoms, representing a direct neural pathophysiologic source of nociception. Neuromas are unimaginable by any current technique, including MRI, and are diagnosed clinically by tenderness to palpation and a positive Tinel's sign and ultimately by histologic analysis. The possible pathologic factors that are likely to induce patellofemoral pain are listed in Table 1. Patellofemoral

alignment can be a factor in the genesis of patellofemoral symptoms representing, in essence, an internal load shifting. However, its importance is properly put into perspective in a more diminished role than is currently espoused.

### KNEE AS TRANSMISSION

Because the primary goal of orthopedic treatment is restoration of joint or musculoskeletal function, what is the function of the knee? The knee can be thought of as a type of biologic transmission whose purpose is to accept, redirect, and ultimately dissipate biomechanical loads.<sup>22</sup> The patellofemoral joint can be visualized as a large slide bearing within this living, self-maintaining, and self-repairing transmission system. Ligaments can be viewed as sensate, adaptive linkages, with the menisci as mobile, sensate bearings. The muscles in this analogy act as living cellular engines, which in concentric contraction provide motive forces across the knee (transmission) and in eccentric contraction act to absorb and dissipate loads.

The functional capacity of a joint to accept and transfer a range of loads and yet maintain tissue homeostasis can be represented by a load/frequency distribution termed the "envelope of function" (or envelope of load acceptance)<sup>(21,22,29)</sup> (Fig. 5A). If too little load is placed across a joint for an extended period of time, as exemplified by prolonged bed rest, loss of tissue homeostasis can ensue, manifested by muscle atrophy and disuse osteopenia (Fig. 5B). This region of diminished loading is termed the zone of subphysiologic underload. If excessive loads are placed across a joint beyond the range of acceptable limits but insufficient to cause macrostructural damage, loss of tissue homeostasis can occur, manifested in bone by a positive technetium scintigraph before radiographic changes, as exemplified by a stress fracture of the tibia in a long-distance runner. This region of excessive loading is termed the zone of supraphysiologic overload. If sufficiently great loads are placed across a joint or musculoskeletal system, overt macrostructural damage can occur, exemplified by a fracture of bone or a rupture of a ligament. This region of excessive loading resulting in overt structural damage is termed the zone of macrostructural failure.

In the current author's opinion, the most common cause of loss of tissue homeostasis that is the source of patellofemoral pain is a patient with normal patellofemoral alignment who sustains loading into the region of supraphysiologic overload, either through a single event, (e.g., direct blow, such as a dashboard injury) or repetitive loading (e.g., excessive stair climbing)<sup>(20,21)</sup> (Fig. 5C). The tissues of the patellofemoral joint sustain the

**TABLE 1.** *Factors inducing patellofemoral nociceptive output\**

Mechanical environment
Direct patellofemoral trauma
Excessive intrinsic compressive and tensile forces
Normal alignment
Malalignment (load shifting)
Impingement of intraarticular structures
Increased intraosseous pressure
Barometric pressure changes
Chemical environment
Presence of cytokines
Altered pH of damaged tissues
Localized peripheral neuropathy
Painful neuroma
Nonpatellofemoral sources
Referred pain (such as hip osteoarthritis)
Phantom limb pain in above-the-knee amputee

\* From Dye and Vampel. Table 1 is reproduced with permission.

highest loading of any human joint and often function at or near the limits of biologic load acceptance and transference capacity.<sup>30</sup> Thus, these tissues often are the first about the knee to be loaded to the point of physiologic failure, leading to symptomatic loss of tissue homeostasis indicated by the perception of patellofemoral pain. The envelope of function, or the safe range of painless loading, frequently diminishes after an episode of injury to the level where many activities of daily living that previously were well tolerated become symptomatic (out of the envelope of function), leading to the prolongation of symptoms (Fig. 5D). Restricting loading to within the newly diminished envelope of function allows normal tissue healing processes to proceed to homeostasis most rapidly without recurrent subversion (Fig. 5E). This restriction of loading to pain-free levels is the clear purpose of the evolutionarily designed negative feedback loop system and also corresponds to common sense. Recurrent painful loading out of the envelope, by participating in previously well tolerated activities such as stair climbing and arising from or sitting in chairs, only subverts normal healing processes and is a hallmark of chronic patellofemoral pain in many, if not most, patients.<sup>21</sup>

The therapeutic implications of a tissue homeostasis perspective are presented in a companion article in this issue of *Sports Medicine and Arthroscopy Review*. In general, the goal of therapy from a tissue homeostasis perspective is to maximize the envelope of function for a given joint or musculoskeletal system as safely and predictably as possible. The treatment approach to patients with patellofemoral pain will be seen to be rational and inherently safer than those based on correcting observable structural and biomechanical factors. Included within this treatment approach will be correction of pathokinematics, including load restriction, anti-inflammatory therapy, rehabilitation, and possible carefully designed and gently performed surgery.

The new concepts presented in this symposium, taken together, represent an alternative perspective of patellofemoral pain that can substantially diminish the mystery of this orthopedic enigma. However, much remains to be discovered by future research. In particular, new methods of sensitively and geographically manifesting loss of soft tissue homeostasis, including dynamic cytokine and substance-P mapping techniques, need to be developed.

## REFERENCES

- Hillsgrove DC, Paulos Le. Complications of patellofemoral surgery. In: *The Patella*. Berlin: Springer-Verlag, 1995:277–290.
- Fulkerson JP, Hungerford DS. *Disorders of the Patellofemoral Joint*. 2nd ed. Baltimore: Williams & Wilkins, 1990.
- Grelsamer RP, McConnell J. *The Patella. A Team Approach*. Gaithersburg, MD: Aspen Publishers, 1998.
- Bentley G. Chondromalacia patellae. *J Bone Joint Surg [Am]* 1970;52:221–232.
- Bentley G, Dowd G. Current concepts of etiology and treatment chondromalacia patellae. *Clin Orthop* 1984;189:209–228.
- Insall J, Falvo KA, Wise DW. Chondromalacia patellae. A prospective study. *J Bone Joint Surg [Am]* 1976;58:1–8.
- DeHaven KE, Collins HR. Diagnosis of internal derangements of the knee. The role of arthroscopy. *J Bone Joint Surg [Am]* 1975; 57:802–810.
- Dye SF, Vaupel GL, Dye CC. Conscious neurosensory mapping of the internal structures of the human knee without intra-articular anesthesia. *Am J Sports Med* 1998;26:773–777.
- McGinty JB, McCarthy JC. Endoscopic lateral retinacular release: A preliminary report. *Clin Orthop* 1981;158:120–125.
- Radin EL. A rational approach to the treatment of patellofemoral pain. *Clin Orthop* 1979;144:107–109.
- Kimball ES. *Cytokines and inflammation*. Boca Raton, FL: CRC Press, 1991.
- Grelsamer RP. Patellar malalignment. *J Bone Joint Surg [Am]* 2000;82:1639–1650.
- Dye SF, Boll DH, Dunigan PE, et al. An analysis of objective measurements including radionuclide imaging in young patients with patellofemoral pain. *Am J Sports Med* 1985;13:432.
- Thomee R, Renstrom P, Karlsson J, Grimby G. Patellofemoral pain syndrome in young women: I. A clinical analysis of alignment, pain parameters, common symptoms, and functional activity level. *Scand J Med Sci Sports* 1995;5:237–244.
- Stäubli HU, Durrenmatt U, Porcellini B, Rauschnig W. Anatomy and surface geometry of the patellofemoral joint in the axial plane. *J Bone Joint Surg [Br]* 1999;81:452–458.
- Dye SF, Chew MH. The use of scintigraphy to detect increased osseous metabolic activity about the knee. *J Bone Joint Surg [Am]* 1993;75:1388–1406.
- Dye SF, Boll DA. Radionuclide imaging of the patellofemoral joint in young adults with anterior knee pain. *Orthop Clin North Am* 1986;17:249–262.
- Biedert RM, Stauffer E, Friederich NF. Occurrence of free nerve endings in the soft tissue of the knee joint. A histological investigation. *Am J Sports Med* 1992;20:430–433.
- Wojtys EM, Beaman DN, Guner RA, Janda D. Innervation of the human knee joint by substance-P fibers. *Arthroscopy* 1990;6:254–263.
- Dye SF, Vaupel GL. The pathophysiology of patellofemoral pain. *Sports Medicine and Arthrosc Rev* 1994;2:203–210.
- Dye SF, Stäubli HU, Biedert RM, Vaupel GL. The mosaic of pathophysiology causing patellofemoral pain: Therapeutic implications. *Operative Techniques in Sports Medicine* 1999;7:46–54.
- Dye SF. The knee as a biologic transmission with an envelope of function. *Clin Orthop* 1996;325:10–18.
- Guyton AC. *Textbook of Medical Physiology. Functional Organization of the Human Body and Control of the "Internal Environment."* Philadelphia, PA: WB Saunders, 1986:3.
- Hirsch E, Moye D, Dimon J III. Congenital indifference to pain: Long term follow-up of two cases. *South Med J* 1995;88:851–857.
- Koshino T. Stage classifications, types of joint destruction, and bone scintigraphy in Charcot joint disease. *Bull Hosp Jt Dis Orthop Inst* 1991;51:205–217.
- Dye SF, Peartree PK. Sequential radionuclide imaging of the pa-



- tellofemoral joint in symptomatic young adults. *Am J Sports Med* 1989;17:727.
27. Sanchis-Alfonso V, Rosello-Sastre E, Monteagudo-Castro C, Esquerdo J. Quantitative analysis of nerve changes in the lateral retinaculum in patients with isolated symptomatic patellofemoral malalignment. *Am J Sports Med* 1998;26:703-709.
  28. Sanchis-Alfonso V, Rosello-Sastre E. Immunohistochemical analysis for neural markers of the lateral retinaculum in patients with isolated symptomatic patellofemoral malalignment. A neuro-anatomical basis for anterior knee pain in the active young patient. *Am J Sports Med* 2000;28:725-731.
  29. Dye SF, Wojtys EM, Fu FH, Fithian DC, Gillquist J. Factors contributing to function of the knee joint after injury or reconstruction of the anterior cruciate ligament. *J Bone Joint Surg [Am]* 1998;80:1380-1393.
  30. Dye SF. Functional anatomy and biomechanics of the patellofemoral joint. In: Scott WN, ed. *The Knee*. St. Louis: Mosby, 1994:381-389.