ATELLOFEMORAL-PAIN syndrome (PFPS) is a common knee disorder in young athletes participating in running, jumping, and cutting sports. The prevalence is approximately 25% among adolescent athletes. Clinically, females demonstrate a greater incidence of PFPS than their male cohorts. When exacerbated, PFPS forces young athletes to restrict or stop regular physical activity. Physical activity has been advocated as a significant component in maintaining positive health behaviors during childhood and adolescence. Sports-medicine practitioners must reduce impairments and functional limitations in young athletes with PFPS in order for them to maintain active lifestyles.

Currently, there is no consensus on the conservative management of young athletes with PFPS. Conservative management of PFPS has been considered an enigma in orthopedics and sports medicine. The inability to uncover the etiology and pathomechanics of PFPS has perpetuated the use of anecdotal assessment and interventions among sports-medicine practitioners. The purposes of this three-part column are to (a) present previous theories of PFPS, (b) introduce a neuromechanical theory for PFPS, and (c) illustrate a biomechanical and motor-control approach to assessing and treating young athletes with PFPS.

Theories of Patellofemoral Pain

The exact etiology and pathomechanics of PFPS are undetermined, but investigators have suggested several theories for the underlying cause of PFPS, including chondromalacia, pathology of the lateral retinaculum, peripatellar synovitis, excessive lateral patellar pressure, vastus medialis dysplasia, patella malalignment, and limited contractile-tissue flexibility. Articular-cartilage damage to the patellofemoral joint surfaces was one of the first accepted theories for patellofemoral-pain disorders. From this theory evolved the classic terminology chondromalacia patellae (CMP), which refers to a softening of the retropatellar surface. Over time, clinicians began to diagnose all cases of anterior knee pain as CMP, including many patellar disorders without signs of articular-cartilage pathology. The theory is significantly weakened by the argument that articular cartilage lacks nociceptive output through substance-P fibers like other articular tissues at the knee. Because of such flaws in the CMP theory, other theories evolved to describe the pathomechanics and etiology of PFPS.

Dye and Vaupel proposed that the etiology of pain in most patients with patellofemoral pain is a result of pathophysiological processes such as peripatellar synovitis and increased intraosseous pressure. The investigators argue that significant changes in the mechanical and chemical environment at the knee trigger nociceptive activity that leads to patellofemoral pain. They suggest that nociceptive input is presented secondary to load shifting to the innervated subchondral bone at the knee. The threshold of pain for subchondral bone could be exceeded with abnormal stress, normal stress in an abnormal direction, or normal stress over an abnormal period of time. Support of this theory has led subsequent investigators in attempts to discover the biomechanical faults that lead to abnormal load distribution at the patellofemoral joint.
The prevailing mechanical theory to explain the onset of PFPS is the concept of vastus medialis obliquus (VMO) atrophy. Many sports-medicine practitioners describe the VMO as the primary stabilizer of the patella during terminal knee extension. Hence, VMO dysplasia or atrophy causes the patella to track excessively to the lateral aspect of the joint. An inability to selectively activate the VMO during functional activities such as walking, squatting, or stair climbing is believed to lead to increased risk of excessive lateral-pressure syndrome and lateral patellar instability. The importance of a selectively activated VMO during functional activities has been refuted, however, in the current patellofemoral literature. Instead of alterations in VMO activation, it is suggested that changes in neuromuscular activity throughout the lower extremity might be associated with PFPS.

Sports-medicine practitioners have also attempted to explain the pathomechanics of PFPS through a malalignment theory. Historically, malalignment has been described in two different models. First, it has been described as an imbalance of the extensor mechanism that results in a translated or tilted patella. Clinical use of patellofemoral taping and bracing evolved from this speculation on the etiology of PFPS. In theory, pain improves because external forces produced by the tape or brace reposition the malaligned patella. Contrary to this early theory, there is little biomechanical evidence that supports the hypothesis that translation or tilt of the patella alone is responsible for PFPS. It is suggested, however, that compressive forces produced by taping or bracing increase the contact area at the patellofemoral joint. Theoretically, this increase in contact area decreases the stress on sensitive patellofemoral structures.

The second malalignment model describes the etiology of PFPS originating from numerous anatomical sites in the lower quarter. The lumbopelvic, pelvofemoral, tibiofemoral, and subtalar joints are believed to affect the mechanics of the patellofemoral joint through the lower kinetic chain linkage. Factors such as hip anteversion, quadriceps (Q) angle, genu valgum, and subtalar- joint position are believed to lead to a “miserable malalignment” that produces patellofemoral pain. Recent literature, however, does not demonstrate a strong correlation between these static measures and the onset of PFPS in young athletes involved in dynamic activities.

Neuromechanical Theory of PFPS

The kinematics and kinetics of the patellofemoral joint are influenced by multiple factors. To completely understand the etiology and pathomechanics of PFPS in young athletes, sports-medicine practitioners must consider the neuromechanical factors influencing the patellofemoral joint during running, jumping, and cutting activities. Factors such as muscle strength and neuromuscular control of the lower kinetic chain are critical for dynamic knee stability during sports maneuvers. Grabiner and associates described neuromechanics in reference to the patellofemoral joint as a “consideration of the principles of mechanics . . . various components of the neuromusculoskeletal system . . . and an examination of the interaction of the biological model with its surroundings.” Poor functional strength, proprioception, and neuromotor control during sports maneuvers can adversely affect the biomechanics of articular tissues about the patellofemoral joint.

Optimal neuromotor control at the knee demonstrates signaling of neural input from mechanoreceptors at the patellofemoral and tibiofemoral joints to the central nervous system. After signals are processed in the central nervous system, efferent signals are projected to lower kinetic chain muscles for regulation of reflexes and motor control. Failure within this neuromotor loop to provide dynamic knee stability can lead to excessive stress on articular or extra-articular tissues.

The literature on altered proprioception and neuromotor control in patients with PFPS is sparse. Baker and colleagues found that there is an abnormal joint-position sense in individuals with PFPS. Although these deficits were found in patients with PFPS, the investigators could not determine whether they preceded or followed the onset of PFPS. Observing athletes without knee injury, Hewett and colleagues introduced several neuromuscular imbalances found in adolescent athletes, including ligament, quadriceps, and leg dominance. In particular, ligament dominance refers to the absence of muscle control in the coronal and transverse planes that leads to high valgus knee torques and ground-reaction forces. Although the primary purpose of their study was to demonstrate how dynamic neuromuscular imbalances correlate