Patellar Tendinosis in a Distance Runner

Author: Corey R. Duvall, DC

Chronic tendon pain is a fairly common symptom in distance runners but for a long time was misdiagnosed. Acute pain that comes on suddenly and disappears within two weeks is called tendinitis. It usually occurs secondary to a contusion or mechanical irritation. Tissue damage causes a normal, healing, inflammatory immune response with associated pain. The pain is best helped with anti-inflammatory pain medication, ice, and rest. Chronic pain, however, is a different problem with a different cause. This pain occurs at the beginning of an activity, "loosens up" and subsides, only to return hours later. If the condition is more severe the pain will return towards the end of the activity. The diagnosis for this is tendinosis.

A tendinosis is created by an imbalance in the forces going through a tendon. Connective tissues (tendon, ligament, cartilage, muscle, and bone) are living tissue; they respond according to the forces placed on them. When we develop in the womb our genetic make-up determines the basic structure and orientation, but then events in life slowly alter this based upon the need. Fibroblasts are the cells that produce collagen and fluid in a tendon. Chondroblasts are the cell that create the collagen and fluid in cartilage. Tendinosis is a lack of mechanical tension in a portion of the tendon causing the fibroblasts to behave more like chondroblasts.

Under normal conditions fibroblasts produce relatively more collagen and less fluid than a chondroblast. A process known as the piezoelectric effect determines both the relative amounts of collagen and fluid, as well as the direction of the collagen fibers. When a tissue is stressed a protein outside the cell is distorted and exposes an electric charge that was buried in the protein. This electric charge stimulates the fibroblast or chondroblast to function as well as directs the alignment of the collagen fibers. In a tendon, the stress is longitudinal and creates dense, parallel collagen fibers. This ensures that the tendon is strong when the muscle pulls it to move the bone. Cartilage absorbs a compressive force that dissipates in random directions through the tissue, and thus the collagen fibers are randomly oriented. This ensures that the tissue can absorb a non-direct force and dissipate the load.

When chronically painful tendons are examined under the electron microscope we find that portions do not appear as tendons, but more closely resemble cartilage. The collagen fibers are more randomly distributed and there is a relative increase in the fluid. This tells us that chronically painful tendons are receiving compressive forces and not longitudinal forces. In patellar tendinosis it is found that the deeper part of the tendon is the part that more closely resembles cartilage.

Biomechanical stress/strain studies have shown that tension through a tendon changes based upon the joint angle that the tendon is loaded. For the patellar tendon knee flexion and extension load the tendon differently. In an extended knee, the more distal and superficial parts of the tendon are stressed longitudinally. The stress of the distal part of the tendon then creates a compressive or pinching force upon the more proximal and deep portion of the tendon (Figure 1). Deep flexion of the knee allows a longitudinal stress of the proximal, deep portion of the tendon and creates a more normal tissue architecture (Figure 2). In a chronically painful tendon it is this proximal and deep portion that degenerates and appears more like cartilage.

What has been shown with scientific research is:

- Normal healthy tendons have dense parallel collagen fibers, very little fluid, and no neurovascular bundles.
- Acutely painful tendons have dense parallel collagen fibers, very little fluid, many acute inflammatory cells in the surrounding sheath, and no neurovascular bundles.
- Chronically painful tendons have loose, irregular collagen fibers, a good deal of fluid, few inflammatory cells, and the presence of neurovascular bundles in the edges of the tissue.

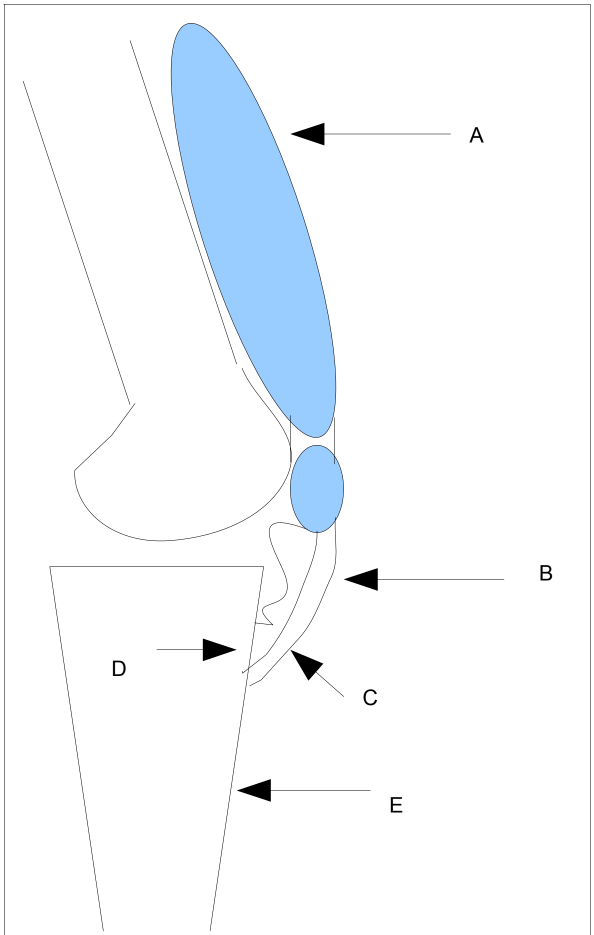

Figure 1: In an extended knee, when the quadriceps (A) contracts the patellar tendon (B) is loaded irregularly. The distal portion (C) is longitudinally stressed while the proximal portion (D) is "pinched" against the tibia (E) creating a compression.

The current theory as to why there is pain has to do with the presence of these abnormally located neurovascular bundles, though this has not been proven. The idea is that the change in architecture allows the infiltration of nerves and their blood supply to the area, but then stress to the tissue creates pain. A normal healthy tendon does not have those pain receptors but an unhealthy one does.

What does this mean for a distance runner?

Patellar tendinosis is caused by an imbalance in the compressive and longitudinal stresses on the patellar tendon. When there is a greater load while the knee is extended there is a greater compressive force on the proximal patellar tendon, creating a change in tissue architecture. This is affected two ways during the running gait:

- Amount of heel lift immediately after toe-off will determine the joint angle of

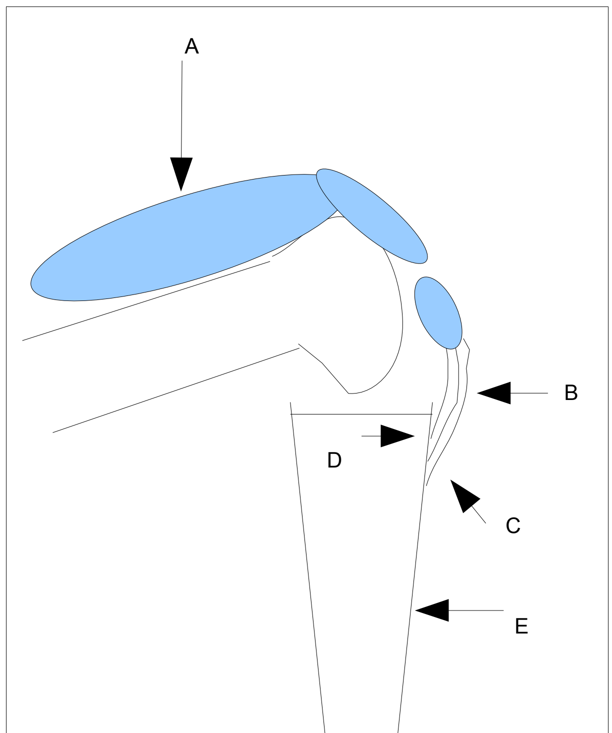

Figure 2: In a flexed knee, when the quadriceps (A) contracts the patellar tendon (B) is loaded regularly. Both the distal (C) and proximal (D) portions are stressed longitudinally.

the knee. Excessive tension in the rectus femoris will limit both hip extension and knee flexion during this phase, essentially shielding the deep tendon from longitudinal stress.
- A more forward-reaching foot strike will increase the anterior femoral shear and the knee flexion moment will be larger. Both increase the need for quadriceps contraction in an extended knee.

When the foot strike is below the center of gravity the force vector at the knee is downward. As the foot strikes anterior to the center of gravity the force vector is both downward and anterior. The anterior portion of the force vector will cause both anterior femoral translation and knee flexion. The quadriceps must increase the contraction to prevent the translation and flexion. The knee is relatively extended at this point and increased compressive force on the abnormal section of tendon occurs. Changes in gait caused by treadmill training, elliptical training, and the modern shoe predispose one to this type of problem.

Two things are necessary for balancing the loads of compression and longitudinal stress:
- Increase longitudinal stress by increasing knee flexion range.
  - Load the tendon with the knee flexed and hip extended in a heel to gluteal stretch.
- Decrease compression by decreasing quadriceps load in an extended knee.
  - Improve the foot strike location.

Your body can correct this problem if a longitudinal stress is applied to the deep tendon in balance with the amount of compression placed upon it. Longitudinal stress on the deep tendon will allow a gradual recovery of the original tissue architecture.

Dr. Corey R. Duvall is co-owner of The Stay Active Clinic. He is a Chiropractic physician and Crossfit certified strength and conditioning coach. The Stay Active Clinic follows a rational and evidence-based approach to provide the best musculoskeletal care, whether you are a weekend warrior or an elite athlete. Contact him at www.StayActiveClinic.com or StayActiveChiro@gmail.com.

References:

- "Biomechanics and pathophysiology of overuse tendon injuries: ideas on insertional tendinopathy." Sports Med. 2004;34(14):1005-17.
- "Biomechanics of the Achilles Tendon" *Disabil Rehabil.* 2008;30(20):1542-7.
- "Chondral metaplasia in calcific insertional tendinopathy of the Achilles tendon." *Clin J Sport Med.* 2006 Jul;16(4):329-34.
- "Compression etiology in tendinopathy." *Clin Sports Med.* 2003 Oct;22(4):703-10.
- "Inflammatory cells do not decrease the ultimate tensile strength of intact tendons in vivo and in vitro: protective role of mechanical loading." *J Appl Physiol.* 2007 Jan;102(1):11-7.
- "Loss of homeostatic tension induces apoptosis in tendon cells: an in vitro study." *Clin Orthop Relat Res.* 2008 Jul;466(7):1562-8.
- "Running Shoe Technology" Brian Mackenzie. Crossfit Running and Endurance Seminar. http://media.crossfit.com/cf-video/CrossFitAgainFaster_BMac_ShoeTechnology.wmv Posted December 13, 2008.
- "Stress-shielding as a cause of insertional tendinopathy: the operative technique of limited adductor tenotomy supports this theory." *J Sci Med Sport.* 2004 Dec;7(4):424-8.
- "The mechanobiological aetiopathogenesis of tendinopathy: is it the over-stimulation or the under-stimulation of tendon cells?" *Int J Exp Pathol.* 2007 Aug;88(4):217-26.