



SOFT TISSUE / TRIGGER POINTS

Why Chronic Ankle Instability Can Affect a Proximal Joint

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A patient presents with two-month knee pain for no apparent reason. The patient also has noticed minimal hip pain on the same side for one month. History reveals that the patient suffered a severe ankle sprain five years ago. On rare occasions, the ankle feels "tight," but is not really a problem. There is no trauma or history of previous pain to the knee or hip.

This case study is fictitious for the purpose of discussion, but it does represent a rather common scenario. We can consider all orthopedic and neurological tests of the knee and hip as normal. Particular muscles such as hamstrings, quadriceps, soleus, and fibularis may test weak. Gait analysis may reveal some minor pathology. Palpation around the ankle and leg reveal some tender, nodular-type areas.

MRI of the ankle five years ago showed abnormal retinacula with thickness and adhesion to the subcutaneous layers and uneven appearance of the retinacula. There was also talofibular ligament rupture with bone marrow edema. (MRI findings taken from Stecco study [reference #8 below]). The above history is described in order to explain why a chronic ankle instability can be responsible for proximal joint (i.e., knee and/or hip) pain.

Arthrogenic Muscle Response Due to Chronic Ankle Instability

One of many studies¹ evaluating arthrogenic muscle response to a unilateral chronic ankle instability reported inhibition of the hamstring muscles bilaterally and facilitation of the quadriceps on the painful side. The study authors mentioned these findings may be present long after the acute injury has resolved.



Other studies also have found arthrogenic muscle inhibition due to chronic ankle instability in the [fibularis and soleus](#) muscles,² concluding that due to a previous ankle sprain, facilitation of the quadriceps motor neuron pool may be related to inhibition of the hamstring motor pool. A motor neuron pool represents all the individual motor neurons that innervate a single muscle.

"These motor neuron pool responses are likely due to the residual effects of chronic ankle instability that stem from a loss of, or change in, sensory information emanating from the injured ankle joint."¹ They are not specific about the location of the sensory information. Most of the sensory information in these joints resides in what are known as the retinacula.

McVey, et al.,² state that an ankle injury results in deafferentation affecting afferent capacity and thus altering signals to the central nervous system. And according to Kandel, et al., "Joint receptors play little if any role in postural sensations of joint angle. Perception of the angle of proximal joints such as the elbow or knee depends on afferent signals from muscle spindle receptors and efferent motor commands."³

Treatment Options and the Influence of the Retinacula

Many soft-tissue methods including instrument-assisted soft-tissue mobilization, friction massage and other pressure techniques seem to help painful areas, especially around the elbow, wrist, knee and ankle. One of the best explanations for working around these joints and obtaining results has to do with the effect of treatment on the retinacula, rather than associated muscles and ligaments.

Retinacula are usually defined as bands around tendons that hold them in place and function mainly to stabilize a tendon. As Carla Stecco (2015) explains in her new text,⁴ the retinacula are very thin and flexible, and have a minimal effect on the mechanical stability of the joint.

Retinacula are more reinforcements of deep fascia than separate structures. For example, it was found that retinacula of the ankle are thickenings of the deep fascia of the leg and foot, not autonomous structures.⁵

Retinacula are located around all the joints of the limbs and are composed of multiple layers of collagen inserting into bones, muscles and tendons. Of all the fascial tissue in the body, the retinacula are the most innervated, containing free nerve endings, Ruffini, Pacinian and Golgi-Mazzoni corpuscles. Ruffini receptors are midrange afferents that give information concerning joint angles and limb movements; while Pacinian corpuscles are very sensitive to acceleration and deceleration, and are used as proprioceptive feedback for movement control (kinesthesia).³

Inability of these receptors to report information back to the central nervous system (CNS), just as the spindle cell dysfunction in the fascia surrounding muscles, results in joint incoordination. Due to the connection of retinacula to specific muscles and bony areas, retinacula sense bone movement and muscular contraction. Retinacula as part of the deep fascia are now considered to have an integrative role in peripheral control of articular motility.

As Carla Stecco explains,³ there is normal stretching (for example, of the ankle retinaculum during pronation and supination), but if there is abnormal gait, trauma or misalignment of the bony structures over time, abnormal retinacula traction may occur. Eventually, abnormal and accessory fibrous bundles may develop in the retinacula tissue. Limited motion and increased tension in this tissue will therefore affect the proprioceptive function of the tissue.

Retinacula are made up of multiple layers that normally slide on each other. Klein, et al., (1999)⁶ described three separate layers of retinaculum in the wrist and ankle. The inner layer contained hyaluronic acid, whose main function is to allow gliding between layers of connective tissue and muscles. Due to lack of gliding between fascia and muscles, receptors (proprioceptors, mechanoreceptors) within the fascia are prevented from being deformed or stretched properly to provide normal feedback to the CNS.

Ankle retinacula damage diminishes normal proprioception at the ankle and along the deep fascia of the leg. As Stecco states: "This can alter the activation of the muscle fibers that insert within the ankle retinaculum, eventually resulting in new retinacula/fascial bony insertions. This may result in a change in the distribution of forces within the deep fascia, affecting ankle joint function and even extending to the knee area." Abu-Hijleh and Harris (2007)⁷ found retinacular "structures" in regions of deep fascia that were never before designated as "retinacula."

So, here we go again, back to the myofascial kinetic chain, with another example of someone entering our office with unexplained knee pain. The case history may reveal a previous ankle trauma that occurred years ago. Invariably in these situations, palpation of the ankle retinacula will reveal areas of densification and pain. The non-traumatized painful knee, or even the hip or lower back, is compensating for a previous injury. This scenario can occur throughout the body.

In an MRI study,⁸ investigators compared treatment of two groups of patients with ankle sprains: one (group A) with ankle retinacula damage only and another (group B) with retinacula damage and anterior talofibular ligament rupture or bone marrow edema. Of the 21 patients treated, MRI revealed specific alterations of the ankle retinacula, including edema and interruption of continuity, thickening or adhesion to the subcutaneous layers, partial or full-thickness gap of one retinaculum. Nine had edema around at least one retinaculum; two showed abnormal retinaculum thickening with uneven appearance; seven had anterior talofibular ligament rupture and/or bone

marrow edema.

Fascial manipulation treatment was administered to the foot and leg three times: one visit per week for three weeks. Patients were evaluated at one, three and six months. Both groups showed similar improvements in VAS scale, ROM, pain during walking, distance without fatigue, stabilometric platform results (sway path, sway area, maximum swing with eyes open and closed).

The anatomical continuity between ankle retinacula and the deep fascia helps explain the reported alterations of motor neuron pool excitability in muscles acting on joints proximal to the ankle with chronic ankle instability. Some other references pertaining to this subject are Bullock-Saxton, et al.⁹ (1994) and McKeon (2008),¹⁰ among others. The authors concluded, "Damage to the ankle retinacula may alter the lines of forces inside the deep fascia of the whole lower limb and thus the capacity for contraction of the underlying muscles."

A practitioner recently told me he relieved a [post-surgical ACL](#) using friction massage around the knee. Treating the retinacula may explain this result.

An important tip in treating restricted fascial areas is to restore gliding of the underlying tissue. For normal function of proprioceptors found in the fascia, it is essential that they are free to glide - in this case, within the retinacula. This requires the palpatory skill of feeling the densified, restricted, usually painful area to be treated. Treatment of an area based on patient pain can never be as significant as treating a palpated restriction.

And most important, a practitioner must be able to palpate a difference after treatment, i.e., return to a normal gliding sensation. Often if there is hamstring weakness, for example, or other positive functional test within the area, if you have normalized an altered location, retesting the hamstring or other related dysfunction will show improvement. Many of these fascial points run in particular sequences (similar to acupuncture meridians), and treating the correct sequence often solves the problem. (By the way, the acupuncture system is within the fascia.)

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