Effects of Neuromobilization on Tendinopathy: Part I

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Tendon injuries account for 30–45% of injuries in sports, with the most common sites affected being the Achilles tendon, patellar tendon, and the extensor carpi radialis brevis (ECRB). Previously, patients presenting with pain at the tendon were commonly diagnosed with tendinitis, but recent histological analysis of tendons demonstrated a lack of inflammatory markers in many injuries classified as tendinitis. Evidence of suspended healing or tendon degeneration led to an increased use of the term tendinosis and the eventual clinical acceptance of the term tendinopathy to classify the general presentation of pain at a tendon. The potential issue with these terms is the attachment to a local pathoanatomical pathology as the root cause of the patient’s pain and dysfunction. As this model does not acknowledge the complexity of these patient presentations, others have suggested the terms reactive tendinopathy or tendinalgia. Use of these terms shifts the clinical focus to the other related causes that may result in a patient presenting with pain at a tendon. The risk of using a term that focuses on a model of a local inflammatory state, or suspended state, is that it may lead to treatments that are not optimal for a specific patient or situation. Thus, it is imperative for the clinician to consider all of the local, as well as regionally interdependent, sources of dysfunction that may result in tendon pain.

Currently, there are three main theories proposed for the local etiological origin of tendon pain: mechanical, vascular, and neural. The first two theories are more accepted and many common treatments are aimed at targeting these causative factors. The neural component, however, is commonly overlooked, which may explain some of the poor outcomes associated with the treatment of patients traditionally classified with a tendinopathy. The utilization of neurodynamics, from an evaluative and treatment perspective, may be important for improving outcomes in patients who present with tendon pain that has a neural component. The purpose of Part I of this manuscript is to review the potential neural involvement in patients with tendon pain and discuss the role of neurodynamics in treating tendon pain.

**Key Points**

- Researchers have not come to a conclusion on the origins of tendinopathy pain.
- Comprehension of tendinopathy theories can help guide a clinician in their patient treatment.
- Neurodynamic tests can help differentiate between a neurodynamic dysfunction and musculoskeletal dysfunction.

**Tendinopathy Theories**

Most clinicians diagnose tendinopathy based on patient signs and symptoms present during physical exam, with the primary complaint of pain over a tendon structure as key diagnostic criteria. Due to the varying
patient presentations and injury states, it is often difficult to identify the origin of tendon pain. While multiple pathology classifications, injury states, and outcome timelines have been suggested, researchers have not come to a definite conclusion on a definitive process for the origin of the pathology or the mechanism of recovery. Due to the lack of consistent diagnostic criteria, it is important to understand the three primary theories explaining the sources of tendon pain.

The mechanical theory is based on the idea of mechanical overload of the tendon resulting in damage to the collagen and other cellular matrix. Tendons that receive high strain loads, such as the Achilles, are often loaded during movement and have been suggested to sustain physiological strain loads of up to 6–8%. Repetitive high strain loads is the theorized causative factor for tendon degeneration. The chronic overload of the tendon may occur over time, causing the tendon to present in a degenerative state without inflammatory markers. The mechanical theory may account for tendon degeneration; however, it does not explain why certain areas of the tendon are more prone to degeneration and does not account for all examples of pain or recovery.

The vascular theory is based on the concept that tendons may experience vascular compromise and neovascularization. Due to the lack of vascularity, the tendon would not be capable of healing because of repetitive high strain loads. The natural disposition of the vascular system may predispose specific portions of the tendons to degeneration. For instance, a mature Achilles tendon has poor vascular supply to the distal portion of the tendon (i.e., two to six centimeters above the insertion). Neovascularization is the proliferation of blood vessels to the tendon, not associated with tendon repair. Researchers have theorized that neovascularization of the paratenon leads to a disturbance in blood supply to the tendon, causing ischemic pain during exercise.

Without appropriate testing, a clinician cannot diagnose a pathological tendon with neovascularization based on patient symptoms. For example, patients that present with pain and stiffness in a tendon are likely to have a diagnosis of tendinopathy without diagnostic testing being performed; therefore, a clinical diagnosis of neovascularization could not be determined. Knowledge of the vascularity in the tendon could help the clinician determine which treatment would best benefit the specific patient as certain interventions (e.g., eccentric exercise) appear to have the potential to reverse the neovascularization. The vascular theory may account for potential causative factors for degeneration of a tendon but fails to address other tendinopathy issues, such as why adolescents would be predisposed to neovascularization in a growing tendon despite an abundance of cellular and metabolic activity.

The neural theory includes several components that may lead to tendon pathology. Increased stresses and tension on nerves will result in altered or ceased blood flow to the nerve, which will influence blood supply to the tendon. The strain forces have been demonstrated to alter nerve conduction if an 8% strain is sustained for an hour and microscopic degeneration of a tendon has been noted at 6–8% of stretch in tendons. Axoplasm, the cytoplasm for peripheral nerves, is five times thicker than water. The decreased movement of the nerve may result in the axoplasm developing into a thick, gel-like substance and, if the axoplasm is interrupted, may lead to dysfunctional neurons. The dysfunctional neurons may affect ion channels, which are channels made up of proteins that are bound in the membrane of the neuron.

Ion channels are gated to open and close based on several mechanisms. While channels are open, ions are allowed to flow through based on the lock and key principle, such as chemicals (ligand gated), electrical current (voltage gated), or stretch and pressure (mechanically gated). The ion channels are generated in the dorsal root ganglion and sent to unmyelinated sections of nerves via axoplasm to the axolemma, usually unevenly distributed. Compression may cause demyelination, introducing more area for ion channels, which can cause up-regulation of ion channels resulting in an increase in sensitivity. Clusters of ion channels, due to demyelination, may result in abnormal impulse generating sites (AIGS) along the nerve, resulting in pain. Chemical mediators being released, such as substance P and glutamate, may result from innervated tendons. Increases in sensitivity and pain, in patients with pathological tendons, may be due to an up-regulation of ion channels in innervating nerves and excitatory chemicals associated with pain (i.e., glutamate).

**Treating Using the Neural Paradigm**

Increases in sensitivity of the nervous system can be attributed to a decrease in neural movement and ability to tolerate strain. Butler suggests movement of the nervous system should reduce mechanical constraints