Tendinopathies of the Elbow, Wrist, and Hand: Histopathology and Clinical Considerations

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ABSTRACT: This article reviews the current opinion of the histopathological findings of common elbow, wrist, and hand tendinopathies. Implications for client management including examination, diagnosis, prognosis, intervention, and outcomes are addressed. Concepts for further research regarding common therapeutic interventions are discussed.


Tendinopathy is a term used to describe a painful tendon condition without confirmation of histopathological findings. In the worst case scenario, it may be characterized as a long-standing tendon condition with localized activity-induced pain that fails to respond to nonoperative therapeutic interventions. Conversely, an acute tendinopathy represents a painful tendon condition aggravated by activity, but the source of pain mediation is not known. For example, it may be because of inflammation of the connective tissues that surround the tendon or it may be a symptomatic degenerative tendon, or both. Clinical observation alone cannot determine if pain from a tendinopathy of insidious onset is because of inflammation and/or the first sign of a symptomatic degenerative tendon. Chosen treatment options could potentially be quite different if the therapist knew the histopathology involved.

Acute tendinopathies or tenosynovitis may develop in hand and wrist tendons as a result of rheumatologic disorders or bacteria infections. The latter is called pyogenic flexor tenosynovitis and it typically occurs from a puncture wound from a rose thorn or fishing hook. Kanavel’s four cardinal signs is the typical clinical presentation of acute tenosynovitis of the finger flexor tendons (Box 1).

Elbow, wrist, and hand tendinopathies are often associated with repetitive movement, including active muscle contractions and stretching over bony surfaces, with and without force. The prevalence of these tendinopathies increases with age and the amount of exposure to forceful repetitive movement. Overuse may occur in the workplace and in sport-related activities.

This article reviews tendon structure and histopathological findings of common elbow, wrist, and hand tendinopathies. Implications for client management and further research in the areas of examination, diagnosis, prognosis, intervention, and outcomes will be discussed. Although any tendon in the upper extremity may develop a tendinopathy, the clinical considerations presented in this article will focus on the three tendon conditions commonly treated by hand therapists: tennis elbow, deQuervain’s disease, and trigger finger.

TENDON STRUCTURE

Tendons provide the interface to transmit muscle force to bone to create joint movement. The composition of tendon is primarily collagen, ground substance, and tenocytes. A network of thin reticular connective tissue known as the endotenon surrounds and binds collagen fibers together to form the primary, secondary, and tertiary bundles that compose the tendon (Figure 1). In addition to binding collagen fibrils together, the endotenon surrounds each of the collagen bundles. Tendon bundles are surrounded by two connective tissue layers called the epitenon and the paratenon. Together, these two layers are known as the peritendon. The paratenon, a layer of loose areolar connective tissue, is the outermost layer and serves as an elastic sleeve to allow gliding of the tendon within the surrounding tissues. It is composed of...
type I and type III collagen fibrils, elastic fibrils, and synovial cells that line the inner surface of the paratenon that interfaces with the endotenon. The epitenon is sandwiched between the paratenon and the tendon and consists of a dense network of collagen fibrils. These fibrils are oriented in various directions to withstand applied loads.\(^3,4\)

Tendons in the wrist and hand are typically enclosed within a fibrous sheath with a synovial lining.\(^3,5,6\) The flexor tendons are surrounded by a complex pulley system and the extensor tendons are surrounded by a retinaculum at the wrist. These structures are involved in the composition of the fibrocartilaginous tunnels that may hinder tendon gliding at the volar metacarpal-phalangeal joints of the digits with trigger finger and the tendons in the first dorsal compartment (extensor pollicis brevis and abductor pollicis longus). Fibrocartilage is found in wrist and hand tendons and it develops in response to compression and shear forces that occur during tendon gliding. The amount and location of the fibrocartilage varies depending on the direction of gliding and the presence of bony and fibrous pulleys.\(^5,7\)

Tendons receive innervation, primarily sensory, from surrounding nerve fibers in the muscle or skin. The peritenon tissues are richly innervated with free nerve endings that function as pain receptors. Other nerve fibers penetrate through the connective tissue sheaths to the surface of the tendon and terminate on sensory nerve endings. The sensory end organs are thought to play a role in coordination, motor control, and pain mediation.\(^3\)

The vascularity of tendons arises from three distinct locations including the myotendinous junction, osseoteninous junction, and the paratenon. Tendons enclosed in a sheath have a more distinct vascular supply that arises from the vincula and mesotenon. In general, the vascularity of a mature tendon is poor and even absent in some regions of the tendon. This may contribute to the poor healing potential of tendinopathies.\(^3,8\) Neovascularization is present with tendon grafts, acute tendon injuries,\(^8,9\) and chronic tendinopathies.\(^10–12\) Although the increased capillary infiltration of the chronic tendinopathy is not associated with tissue repair, it is not clear what role this vascularity may play in the degenerative tendinopathy, but it does contribute to pain mediation.\(^8\)

**HISTOPATHOLOGY OF TENDINOPATHIES: TENDINOSIS**

Although the etiology of the degeneration is not well understood, surgical excision of the involved tendon from patients with chronic tendinopathies has allowed extensive analysis of the histopathology.\(^11,13–18\) The term tendinosis has been used to describe the histopathological findings identified in

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**Box 1. Kanavel’s four cardinal signs\(^1\)**

1. Semiflexed posture of the involved finger(s).
2. Symmetrical involvement of the entire finger(s).
3. Excessive tenderness to palpation along the course of the flexor tendon sheath.
4. Excessive pain with passive extension of the involved finger(s) localized to the flexor tendon sheath.

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**FIGURE 1.** Tendon structure with connective tissue. The collagen fibril is the smallest unit of tendon that can be mechanically tested. An aggregate of collagen fibrils form a collagen fiber. Collagen fibers are bound together by the endotenon to form the primary, secondary, and tertiary bundles. The epitenon and paratenon are the outermost layers of connective tissue and together are known as the peritenon.
a chronic tendinopathy. These findings include absence of inflammatory infiltrates; tenocyte or fibroblast hyperplasia and morphology; endothelial cell hyperplasia; microvascular thrombosis; hyaline, fatty, mucoid, calcified, fibrous infiltrates within the tendon substance; and cell necrosis. Because surgery for acute tendinopathy is rare, it is not known if similar pathological findings would be observed in these patients. With this lack of research, it is no surprise that staging patients’ tendinopathy via clinical examination remains a challenge. However, Nirschl and colleagues described four stages of tendinosis that may assist the therapist in determining what type of intervention to provide the patient. Stage 1 is described as a peritendinous inflammation, which may cause palpable crepitus with tendon movement. This stage is actually what most clinicians refer to as tendinosis. Stages 2, 3, and 4 refer to the presence of angiofibroblastic degeneration, with stage 4 being the most severe. Because of fibrosis, stage 3 may lead to tendon rupture and stage 4 to calcification. Tendovaginitis refers to the entrapment of a tendon within its fibrous sheath. Because of thickening of the tendon sheath walls, the diameter decreases or becomes stenotic, trapping the tendon. Tendon entrapment may be a more suitable name to this type of tendinopathy and trigger finger or digit and deQuervain’s tendinopathy are the most common. Degenerative changes (tendinosis) within the tendon are the same as what has been described for the extrasynovial tendons. The thickening of the tendon sheath is because of the presence of fibrocartilage as an adaptation to compression and shear placed on the tendon during gliding.

NEUROGENIC INFLAMMATION

Despite the absence of prostaglandin inflammation, patients with tendinopathies present with pain, particularly with abusive or aggravating activity. Tissue studies have identified the presence of neurochemicals within the tendons of patients with chronic tendinopathies. Significant levels of substance P, calcitonin gene-related peptide (CGRP), and glutamate were reported within the extensor carpi radialis brevis (ECRB) tendon in patients with chronic tennis elbow. No significant differences were noted in prostaglandin levels in these same specimens. The presence of significant levels of glutamate, substance P, and CGRP in tendinosis provides an alternative mechanism for pain mediation in chronic tendinopathies. To date, the presence of neurogenic inflammation has not been investigated in tendon or tendon sheath specimens from patients with wrist or hand tendinopathies.

NEOVASCULARIZATION

It is not known if this neurochemical response is present in a tendinopathy with symptom duration of six months or less and if there are concurrent inflammation or degenerative changes within the tendon because the human subject studies were only performed on tendons of patients with chronic tendinopathies at the time of surgery. Animal models must be used to determine if there is an early neurochemical response associated with acute tendinopathies. A chemically induced experimental model of tennis elbow in Sprague-Dawley rats revealed that substance P is abundant during an acute inflammatory response in the ECRB tendon compared with similar tissue samples in the control group. Messner et al. examined immunoreactivity for substance P in the endotenon and paratenon tissues in the hindlimb triceps muscle after repetitive eccentric muscle contractions in a controlled kicking rat model. Neurofilament labeling was evident within the epiotenon and paratenon of the trained tendons, but only sparsely apparent in the control tendons. Immunoreactivity for substance P was intensive in the experimental limbs of the trained animals and sparse in the contralateral limbs of the trained animals and control animal limbs. Substance P immunoreactivity was determined using bioquantification techniques in a volitional rat model of repetitive forceful motion. Substance P increases in peritendon tissue in forelimb flexor tendons (just proximal to the carpal tunnel) that have been exposed to highly repetitive and forceful tasks. The presence of substance P was also dependent on task exposure, with the greatest response at 12 weeks. Degenerative changes to the tendon tissue were also observed. These animal studies suggest that at least substance P is present in acute overuse tendon conditions.
The vascularity of the ECRB is compromised on its volar surface in the zone between the enthesis, or osseoustendinous junction, and the musculotendinous junction, making this area more vulnerable to degenerative changes.\textsuperscript{21,25,31} Tennis elbow is really an enthesiopathy\textsuperscript{32} and may respond differently to treatment compared with a tendinopathy that occurs within the midsubstance of the tendon or at the musculotendinous junction.

MODELS OF TENDINOPATHY

After more than 25 years of study, it is generally agreed that an injury to a tendon is the inciting event that leads to the development of a tendinopathy.\textsuperscript{1,13,21} The inciting event may be a traumatic injury, an injury because of forceful repetition, a chemically induced injury such as fluoroquinolone toxicity, or a pathogen that causes a pyogenic or purulent response. As with any connective tissue injury, cell-mediated inflammation is the initial hallmark response to healing. The problem is that the tendon fails to heal and remains in a state of stagnant fibroplasia. The current dilemma is that what initiates and maintains the “failed healing” stage remains unclear.\textsuperscript{30,31}

Current theories present an array of cytokines and growth factors that are either absent or present in quantities that upset the equilibrium of the healing process during fibroplasia and do not allow the healing continuum to achieve remodeling.\textsuperscript{30,31} This is the basis for using guided ultrasound injections of autologous whole blood or platelet-rich plasma to restore cytokine/growth factor equilibrium into the area of tendinosis.\textsuperscript{30,33,34}

As a result, the clinical presentation is twofold: 1) morphologic changes to the tendon leading to decreased tensile strength, which may lead to tendon rupture and 2) increased pain mediated by cytokines and vascular ingrowth containing high levels of potent pain mediators such as glutamate and substance P. The latter is the foundation of another novel approach to the management of tendinopathies. Sclerosing agents such as polidocanol may be injected into the area of tendinosis and vascular ingrowth using guided ultrasound with color Doppler to destroy the neovascularization and reduce pain.\textsuperscript{28,30,33}

Additionally, changes in the pain and motor systems of the brain and spinal cord (neuroplasticity) may occur as a result of afferent input from tendon sensorimotor end organs and the presence of neurogenic inflammation.\textsuperscript{35} As a result, pain may become more widespread because of central sensitization, and motor impairments may occur because of abnormalities in sensory feedback, impaired strength, and altered motor control.

CLINICAL EXAMINATION

A thorough examination of the patient with suspected tendinopathy begins with a comprehensive patient history to help determine whether the tissue is chiefly characterized by inflammation or degeneration. The index of suspicion is directed toward a degenerative condition if the patient 1) is older than 40 years; 2) has a duration of symptoms greater than three months; 3) has had more than one episode of the same tendinopathy in the same extremity; 4) has exposure to physical risk factors (repetitive, forceful, awkward movements); and 5) has comorbidities with a high prevalence of tendinopathies, such as rheumatologic disorders, diabetes mellitus, and amyloidosis.\textsuperscript{1,19,20}

Current medications should be reviewed and with the patient, but it is also important to ask the patient about any recent history of taking antibiotics, specifically fluoroquinolones.\textsuperscript{36,37} There is a known risk of inducing tendinopathies in patients taking this class of antibiotics. Risk appears to be greatest in patients older than 60 years, active in regular exercise or sports, or receiving concomitant corticosteroids.\textsuperscript{36}

A differential diagnosis should be performed as part of a thorough clinical examination. Proximal sources of pain such as cervical radiculopathy, proximal neurovascular entrapment must be ruled out using patient interview, upper quarter screen, cervical examination, shoulder examination, and neurodynamic assessment.\textsuperscript{38–41} Once proximal sources of pain are ruled out, local sources may be addressed. Radiographs may be used to rule out osseous conditions at the elbow or wrist.\textsuperscript{42–44}

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severity of symptoms to improve communication among therapists and identify which interventions have the best outcomes.

IMAGING

Imaging procedures such as magnetic resonance imaging and diagnostic ultrasound (sonography) may contribute to the clinical assessment of tissue status in chronic tendinopathies without the need to biopsy or remove the tendon tissue. However, the results are not always discriminatory. Imaging can illustrate overt morphologic changes to the tendon (such as a tear or thickening of the tendon or its sheath) and detect the presence of fluid or increased metabolic activity. However, these tests cannot show the composition of the tendon tissues or identify histopathological findings such as identify the cellularity, type of inflammation (neurogenic vs. prostaglandin), or the presence of cytokines that may facilitate or impede tendon healing. Each of these components of pain mediation or healing potential determines the severity of symptoms identified by the patient and prognosis. Imaging is best for determining structural changes to the tendon, but not the histopathological findings. For this reason, and because of the high costs associated with imaging, it is typically not ordered until the patient with tendinopathy has failed at least one episode of care in nonoperative management, which may or may not include hand therapy.

THERAPIST’S MANAGEMENT: USING THE USUAL SUSPECTS

Physical Agents

Physical agents are frequently used in the nonoperative management of elbow, wrist, and hand tendinopathies to modulate pain or to facilitate tissue healing based on the theory of failed tendon healing. There is no consensus in the literature as to their effectiveness. Recent reviews demonstrate there is some evidence for the use of ultrasound, iontophoresis, and low-level light therapy in patients with tennis elbow, but there are no studies that have determined the effectiveness of these same agents on patients with trigger finger or deQuervain’s tendinopathy.

Most studies involving physical agents incorporated other therapeutic interventions into the plan of care such as patient education and exercise. In fact, the Nirschl et al.’s study demonstrated that the iontophoresis control group (saline delivered) had similar outcomes to the iontophoresis treatment group (dexamethasone delivered) at one-month follow-up compared with group differences observed after six visits (two-week follow-up). These results suggest that the other therapy interventions were helpful to both groups two weeks after iontophoresis treatment was completed. Physical agents should not be used in isolation and may only provide short-term benefits for pain modulation and tissue healing.

There is anecdotal evidence for the use of noxious level stimulation, a form of electroanalgesia, for pain modulation in tendinopathies. After adequate skin preparation, two small electrodes are applied to the focal area of tendon pain to create a high current density and are secured by tape to prevent migration within interelectrode distance (Figure 2). The painful area is identified by palpation and painful provocative movement such as a Finkelstein’s test for deQuervain’s tendinopathy. To determine treatment effectiveness, pain scores using either a verbal or visual rating scale should be used pre- and posttreatment. Using a burst modulated alternating current (Russian Stimulation), the parameters include a medium carrier frequency (2,500 Hz) delivering 50 bursts/sec; 50% duty cycle, 12 seconds on and 8 seconds off for 10–15-minute treatment duration. The sensation is intense tingling, not burning, and is increased during treatment if tolerated. The expected outcome is immediate pain relief documented by lower pain scores for palpation and the provocative maneuver.

Immediate pain relief may be because of interelectrode analgesia from gating peripheral nociceptive fiber (Aδ) input at the treatment area and a more long-term analgesic response by stimulation of periventricular gray areas of the brain that release enkephalins and/or endorphins to activate the endogenous descending pain system. Although the mechanisms for pain modulation require further

FIGURE 2. Noxious-level stimulation electrode placement for tennis elbow. After proper skin preparation to the target area, small electrodes are placed over the focal area of pain parallel to the direction of the musculotendinous unit (common extensor tendon). As a precaution, electrodes are secured with tape to prevent migration if the patient moves or the surrounding muscle contracts.
investigation, Defrin et al. \textsuperscript{65} demonstrated greater pain relief using noxious level stimulation parameters in patients with chronic knee osteoarthritids than with sensory level stimulation parameters. Clinical investigations using noxious level parameters for chronic tendinopathies may yield similar results.

**Transverse Friction Massage**

Cyriax\textsuperscript{66} advocated the use of transverse friction massage for soft-tissue conditions such as tendinopathies to reduce pain and to promote tissue healing. He theorized that friction massage would promote collagen alignment and fiber lengthening, which would reduce scar formation within the common extensor tendon. He also believed that blood flow would increase in the area of massage as a result of a reactive hyperemia from the firm pressure applied.\textsuperscript{66,67} A recent systematic review of transverse friction massage reported that no conclusions could be drawn from the current literature because of lack of controlled clinical trials, adequate sample size, and comparable methods.\textsuperscript{68} Clinical observation reveals that many patients do not tolerate the required firm pressure because of exquisite point tenderness associated with their tendinopathy. Additional research is necessary to determine the clinical efficacy of massage and soft-tissue mobilization, with and without therapist-assisted instruments, in the outcome of therapist’s management of elbow, wrist, and hand tendinopathies.

Additional systematic reviews of the same clinical studies are not needed. Instead, future clinical investigations should focus on the application of physical agents, transverse friction massage, and soft tissue mobilization based on patient classification categories that still need to be developed for each of these patient populations: tennis elbow, deQuervain’s, and trigger finger.

**Orthotic Intervention**

Rigid orthoses are frequently used to abate acute pain symptoms associated with tennis elbow, deQuervain’s tendinopathy, and trigger digits.\textsuperscript{19,49,69} As acute symptoms resolve, flexible orthoses, braces, or taping may be used until support is no longer indicated. The orthoses are designed to prevent the movement most likely to aggravate the condition; therefore, patients wearing these orthoses should limit functional use of the involved hand. It may not be beneficial for the patient to wear a rigid orthosis during their normal functional activities as they may adopt abnormal movement patterns from “fighting against the orthosis.” This may lead to no reduction in current pain symptoms or create new areas of discomfort. Orthotic intervention requires careful patient selection and patient education to ensure proper use of the orthosis.

There are limited clinical trials that have evaluated the effectiveness of rigid and flexible orthoses alone, especially without corticosteroid injections. Recent systematic reviews for the use of orthoses with tennis elbow provide limited evidence because of low-quality clinical studies.\textsuperscript{70,71} Future clinical investigations will require more rigid research design and study populations with greater numbers and homogeneity.

**Eccentric Exercise**

In recent years, painful or high-load eccentric exercise has been advocated to resolve pain associated with chronic lower extremity tendinopathies.\textsuperscript{28,29,72} It is theorized that painful eccentric exercise reduces neovascularization (which is implicated as a source of pain mediation), within the tendon. Eccentric strengthening exercises may result in adaptive tissue remodeling at the musculotendinous junction that include hypertrophy and increased fibroblast activity, which leads to increases in collagen production.\textsuperscript{73} Therefore, this form of exercise may facilitate healing and increases in tensile strength in chronic tendinopathies, which would result in a resolution of pain and increased tolerance for eccentric loading during functional activities.

Eccentric exercises are not commonly used with wrist and hand tendinopathies. The use of eccentric exercises for tennis elbow has not shown superior results to symptom resolution when compared with other types exercises, or are inconclusive\textsuperscript{74–77} to the outcomes observed in the chronic lower extremity tendinopathies. Several factors specific to the involved tendons need to be considered when trying to compare the results of these different tendinopathy groups. These factors vary so greatly, that attempts to manage all tendinopathies in a similar manner using eccentric exercise may not be appropriate.

Based on the alignment with the respective muscle and structures that surround the tendons, the Achilles tendon and the patellar tendon would be considered traction tendons. The common extensor tendon at the elbow would also be a traction tendon. In traction tendons, the muscle and tendon are aligned and the tendon does not make a significant change in direction. Gliding tendons are those tendons that do divert their direction away from the alignment of the muscle to move over or around bony prominences or fibrous pulleys. The tendons in the wrist and hand (enclosed within a fibrous tendon sheath and secured by pulleys to bone) would all be considered gliding tendons.\textsuperscript{78} Gliding tendons have fibrocartilaginous zones in areas exposed to compression or shear because of the bony prominences or pulleys.\textsuperscript{7,23} Tension loads lead to degenerative changes.
in traction tendons and compression or shear forces lead to degenerative changes in gliding tendons, especially in areas of the tendons with poor vascularity. Eccentric strengthening exercises mimic tension loading of traction tendons.

The location of tendinosis within the tendon may impact the benefits of eccentric strengthening exercises. The histopathological findings of the lower extremity tendinopathies are typically found within the tendon substance away from the insertion of the tendon. In the Achilles tendon, tendinosis, neo-vascularization, and spontaneous ruptures occur 3–6 cm from the insertion of the tendon on the calcaneus. In tennis elbow, the degenerative changes to the tendon including tendinosis and neo-vascularization, occur at the insertion or enthesis of the tendon onto the lateral epicondyle of the humerus. The composition of the tendon varies from midsubstance to enthesis. Fibrocartilage blends with bone at the enthesis. Adaptive tissue remodeling would be different for these different zones within the tendon and the enthesis may not respond favorably to the eccentric tendon loading.

The application of eccentric loading is different between the lower and upper extremity tendons. Eccentric loading of the Achilles and patellar tendons occur naturally with functional use of the lower limbs such as during gait and descending stairs. Identifying functional activities in the upper extremity that use eccentric load is a bit more challenging. Gripping or accepting a load with the elbow completely straight would cause eccentric loading of the common extensor tendon. However, most people would quickly extend their wrist and bend the elbow to carry the load. The tendons in the first dorsal extensor compartment are subjected to an eccentric load when pouring a beverage from a plastic jug, but the tilting angle and emptying of the jug would reduce the eccentric load. These functional differences between the lower and upper extremities may render eccentric strengthening exercises less important for the upper extremity tendons and provide some explanation for why they may not benefit or tolerate eccentric loading when a tendinopathy is present.

Because functional eccentric loading is common to the lower extremity tendons, it is easier to load these tendons for strengthening by using body weight with or without the use of additional weights supported on the trunk. Conversely, some type of external load such as a dumbbell, kettle bell, or flexible resistance bar is applied to create eccentric loading at the elbow and wrist. This type of exercise may seem “less natural” to the patient and it is more difficult to maintain or progress the load. This is particularly true when resistance bands are used to apply the external load. Flexible resistance bars, a novel way to apply an external eccentric load, should be further investigated for both tennis elbow and deQuervain’s tendinopathy using a more rigorous study design (e.g., examiner blinding) and large sample sizes to enhance statistical power.

Another concept related to load application is the amount of force required to make the eccentric exercise effective in producing adaptive changes. Most studies describe performing exercise that is “painful” but do not operationally define it (e.g., pain scale). Some studies do not indicate if the exercise was done in a painful manner. Pilot data regarding the use of eccentric strengthening exercises suggest that hand therapists do not have patients perform the exercise in a painful manner. Classification of patients with elbow and wrist tendinopathies using symptom or pain phase stratification would allow us to examine the outcomes of eccentric loading with and without pain.

Relative Rest

Tennis elbow is considered the most prevalent work-related musculoskeletal disorder at the elbow and sufficient evidence exists for a strong association between its prevalence and a combination of physical risk factors including force, repetition, and posture. Similar findings have been observed for deQuervain’s tendinopathy and trigger finger in the workplace. During the patient history, information is gathered about work duties and avocational activities to determine potential causes of tendinopathies. Management of these causes or risk factors, through education and jobsite or tool modifications, may be more important to the worker’s prognosis than medical or therapy intervention.

Elbow, wrist, and hand tendinopathies are also prevalent in sports such as tennis, golf, and rock climbing. Prevalence increases with risk factors such as poor skills in the sport, inadequate neuromuscular conditioning, and increased time spent performing the sport. Management of these risk factors through patient education and skills acquisition from the proper coach should improve treatment outcomes.

Interventions such as ergonomic counseling, sports modifications, and other lifestyle changes to reduce aggravating activity during the current episode and prevent recurrent episodes are key components of patient education. In a recent survey, hand therapists identified patient education and activity modification as essential components of therapy for tennis elbow. However, evidence to support patient education does not exist. In a frequently cited paper on tennis elbow, one of the study groups received a brochure on the concepts of relative rest. This group was not viewed as a treatment group and was labeled the “wait and see” group. During the initial six-week follow-up, this group did not do as well as the treatment groups that either received a cortisone injection.
or therapy. However, at the one-year follow-up, the outcomes for the “wait and see” group were significantly better than the cortisone injection group, but not quite as well as the therapy group. The main conclusion was that relative rest was almost as good as therapy. A study that combined the concepts of relative rest and standard therapy interventions may yield even better outcomes. Further clinical investigations are needed to determine the optimal methods of delivery and content involved in patient education.

**CONCLUSION**

The varied clinical presentation of chronic tendinopathy certainly explains the long list of interventions that might be used to offer symptom relief and restore function to patients. As science continues to unfold the mysteries of the failed healing response, hand therapists need to use a sound clinical reasoning approach to determine the status of the involved tendon tissue to predict which therapeutic interventions will most likely achieve the best possible outcome. Hand therapists should be encouraged to take a fresh look at all treatment interventions, including novel techniques, and engage in high-quality interdisciplinary clinical research. The long list of systematic reviews showing inconclusive results because of poor quality studies indicates that clinicians have yet to discover the “magic bullet” that will resolve the most recalcitrant tendinopathies of the elbow, wrist, and hand.

**REFERENCES**


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#1. In a tendinopathy patient, clinical observation
   a. can confirm that it is an acute inflammatory process
   b. can confirm that it is an insidious degenerative process
   c. cannot discriminate between an acute inflammatory process and an insidious degenerative process
   d. is of little or no value

#2. Peritendon is composed of
   a. epitenon and paratenon
   b. collagen bundles surrounded by vascular tissue
   c. perpendicularly oriented collagen fibers
   d. amorphous loosely packed immature tissue

#3. As a general statement the blood supply to tendons is
   a. rich
   b. pervasive
   c. absent
   d. tenuous

#4. Chronic tendinopathy is considered to be
   a. neither a tendinitis nor a tendinosis
   b. both a tendinitis and a tendinosis
   c. a tendinosis rather than a tendinitis
   d. a tendinitis rather than a tendinosis

#5. The term tendinopathy implies a specific histopathology
   a. true
   b. false

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