Acute and Chronic Tendon Injuries: Factors Affecting the Healing Response and Treatment

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Objectives: Tendons have biomechanical properties based on collaborative remodeling of all their cells through normal lysis and synthesis. This review assesses factors that affect the healing response and presents solutions for rehabilitating acute and chronic tendon injuries. Data Sources: MEDLINE (1970–2002) and SPORTDiscus (1970–2002). Key words searched were tendon, tendinitis, tendinosis, tendinopathy, rehabilitation, ultrasound, NSAIDs, exercise, mobilization, aging, immobilization, and healing. Data Synthesis: The biomechanical roles tendons play change throughout one’s lifetime and are influenced by maturation and aging, injury and healing, immobilization, exercise, medications, and therapeutic modalities. Suggestions from animal, case, and clinical studies are varied but provide solutions in the treatment of acute and chronic tendon injuries. Conclusions and Recommendations: All factors that affect the tendon structure should be considered in a rehabilitation program. Therapeutic exercise, medications, or therapeutic modalities should never be used as a stand-alone therapy. Key Words: inflammation, tendinitis, tendinosis, aging, rehabilitation


Tendons have biomechanical properties that are highly adaptive to the complex functions these structures must perform. These properties are based on collaborative remodeling of all the cells in the tendon structure through normal lysis and synthesis to produce an adult tissue capable of performing complex biomechanical roles. Complex biomechanical roles related to the tendon’s response to loading modes such as tensile stress, frictional forces, and repetitive movement change throughout one’s lifetime and are influenced by maturation and aging, exercise, immobilization, medications, and injury.

Various studies, case studies, and articles are published every year describing protocol variations. Despite the information available for various musculoskeletal injuries, little information on the treatment of chronic tendon injuries can be found. This might be related to the fact that little was
known until recently\textsuperscript{15-19} about the changes a tendon structure undergoes during chronic injury. One can only assume that the repair process is similar to acute tendon healing, based on microscopic and biomechanical testing from animal studies, and propose hypotheses for the healing response.\textsuperscript{7}

Another consideration is the fact that classification of chronic tendon injury might reflect what is happening to the tendon and tissues surrounding the tendons. Inflammatory disorders of the tendon sheath or tendon are classified as tenosynovitis, peritendinitis, and tendinitis. A degenerative process of the tendon, as opposed to an inflammatory disease, is described as a tendinosis.\textsuperscript{15,19,20}

The purpose of this review is to present factors that affect the biomechanical properties of tendons, such as maturation and aging, injury and healing, immobilization, exercise, medications, and therapeutic modalities. An additional purpose is to present solutions for the rehabilitation of acute and chronic tendon injuries. With this information, athletic trainers will be able to design appropriate rehabilitation programs before decreases in tensile strength and range of motion (ROM) become irreversible.

**Factors That Affect the Biomechanical Properties of Tendons**

The tendon transmits force from the muscle to the bone to allow movement. Collagen, which is produced by a precursor molecule tropocollagen, and elastin allow the tendon to carry out this function. Tensile movement is the loading mode that this wavy, crimplike structure can handle because of intramolecular cross-links with other collagen fibers. As is evident with in vitro animal and cadaver studies, any loading mode within the elastic zone is dealt with well. Once the load places the structure in the plastic zone, the tendon, even with its viscoelastic behavior, will rupture. The rupture site of the weakened tendon might be at the midsubstance area, the myotendinous junction, or the connection to the bone. Other loading modes such as frictional forces and repetitive movement lead to chronic tendon injuries, which result in inflammation, degeneration, and rupture if excessive force is placed on the tendon.

**Maturation and Aging**

During maturation, there is a change in the number and quality of the cross-links within and between collagen molecules. These elements influence the response of the tissue to stress, the amount of deformation possible, the ability of the tissue to return to its original length after deformation, and the method of force transfer within the tissue.\textsuperscript{8} The number and quality of the intramolecular cross-links between the tropocollagen molecules increase, resulting in greater tensile strength of the tendon.\textsuperscript{1} Collagen-fibril diameter and total collagen content further increase, adding to the stability of the
collagen fibril. Even elastin shows an increase in the number of cross-links, but the total number of elastin fibers decreases.

After maturation and with the progression of aging, a plateau is reached in regard to mechanical properties and joint ROM. Eventually, collagen and elastin begin to show a decline in their mechanical properties. This results in a decrease of tensile strength and stiffness. In addition, the crimp structure of the collagen changes. With an increase in the wavelength comes a corresponding decrease in wave-crimping angle, which causes the collagen fibrils to reach the linear zone of the load-deformation curve sooner. This might be the reason, aside from degeneration, that Achilles-tendon overuse injuries occur at a higher rate in older athletes than most other typical overuse injuries. Decrease in ground substance reduces the gel:fiber ratio. The space between the collagen fibers diminishes, with more binding occurring between these structures. This in turn leads to more stiffness and eventually affects joint ROM.

**Injury and Healing**

After acute injury, the tendon undergoes a process similar to what is noted in the healing of incised skin wounds. Because of the lack of human biopsy studies, the information has been obtained from studies on acute healing performed on horses, rabbits, and rats. The healing process is divided into 3 stages. The first is called the inflammatory cycle. During the inflammatory cycle, inflammation, phagocytosis, and neovascularization occur to remove debris, provide circulation and oxygen, and prepare the site for connection of the torn fibers. The second stage is known as the fibroblastic/proliferative phase, in which the fibroblasts secrete tropocollagen, which aggregates, polymerizes, and cross-links in the extracellular space as type III collagen. The difference between tendon healing in this stage and wound healing is that epithelialization and wound contracture do not occur. Myofibroblasts are present, but only with a paratenon repair. The last stage is known as collagen remodeling. During this stage, type III collagen is converted to type I collagen. It is important to note that these phases are not absolute and do overlap. Chronic-tendon-injury healing does occur over time, but the stages are not as well defined as in acute-injury healing. Treatment of injury and healing are discussed later in the article. The stress–strain curve when referring to the tensile strength of types III and I collagen is discussed elsewhere.
to the injury site is facilitated by chemicals released from disrupted cell membranes and platelets deposited at the site of injury as a result of bleeding. These chemical mediators increase cell-membrane permeability and circulation to the area to bring oxygen and nutrients to aid in the healing process.

To continue phagocytosis, 2 days later chemical mediators attract monocytes and later macrophages and lymphocytes to the site of injury. Macrophages might play the most important role because they promote migration of fibroblasts to the site of injury and stimulate proliferation. How this happens has been a matter of speculation, but the macrophages produce several different growth factors, which are related to other growth factors and chemotactic agents such as fibroblast growth factor, leukotriene B₄, platelet-derived growth factor, transforming growth factor, and insulin growth factor I. To bring oxygen and nutrients to the area, new vascular buds (neovascularization) develop from capillaries in the vicinity of the rupture. As the buds project into the wound, they anastomose to develop a rich vasculature. Several prostaglandins, macrophageal growth factors, neutrophils, T lymphocytes, platelet-derived growth factor, fibroblast growth factor, and epidermal growth factor initiate development of blood vessels. Once the area is clear of debris, the fibroblasts start to secrete tropocollagen molecules. At the end of this stage, the gap initially filled by a hematoma is invaded by proliferating fibroblasts from the epi-
tendon, endotendon, and surrounding connective tissue. These fibroblasts become active at day 7 and start to initiate the process of scar development that brings the torn fibers together. If the debris and edema are not removed before scar development, there will be a wider gap between the torn fibers that will result in laxity and scar tissue that will reach peak stress-to-load deformation more quickly.

The second stage of acute-injury healing is known as the fibroblastic/proliferation stage, which is massive and prolonged in acute tendon injury. During this stage, redevelopment of the extracellular matrix is important for structural integrity of the tendon. The extracellular matrix, which encompasses collagen, supplies the tendon with both structural support and a medium for the transport of essential nutrients. The ground substance (proteoglycan, glycosaminoglycan, and water) provides structural support for collagen fibers and regulates the extracellular assembly of procollagen into mature collagen. Fibroblasts lay down the components of a new extracellular matrix by synthesizing tropocollagen molecules, elastin, and ground substance. As the fibroblasts proliferate, large masses of cytoplasm with networks of roughened endoplasmic reticulum, several electron lucent vesicles, some dark granules, numerous ribosomes, and a few undefined Golgi complexes are developed. The abundance of cytoplasm suggests that secretory proteins (tropocollagen) are being synthesized, as is evident by the collagen fibrils in the extracellular matrix. These collagen fibrils are mostly in disarray, but an occasional longitudinally arranged fibril is present with proteoglycan ground material in the extracellular matrix.
Type III collagen is the predominating collagen in the early stages. Collagen chains formed are disorganized in a gelatinous fluid and are very fragile until cross-linking occurs. This structure replaces the hematoma that developed earlier in an attempt to bind the torn tendon stump together. Fibroblasts that appeared during the first week continue to proliferate, becoming longitudinally oriented on the surface of the tendon at the same time that they are being progressively grouped into bundles. Cross-links begin to develop among the collagen, but these fibers are randomly distributed throughout the tendon. Specialized cells called myofibroblasts, which have smooth-muscle-contraction ability, are present if the tear is in the paratenon structure. They pull the torn parts closer together but can produce adhesions if mobilization is not attempted. These cells are very active in the healing of incised wounds.

Toward the end of the fibroblastic phase, the maximum mass of collagen is present with cross-linking and interlacing but only has 15% of available tensile strength. This is enough to permit controlled movement and allow healing to progress to the next phase. Healing can also regress back to the previous phase, though, if motion of the tendon exceeds 15% of available tensile strength, known as bidirectional movement.

A problem occurs when there is a large enough gap between the torn ends to prevent close approximation of the fibers. Surgery is one option to bring the torn fibers together during this stage. Patient selection and a complete diagnostic analysis at the time of trauma can help in deciding the treatment in each individual case. After surgery for an Achilles-tendon rupture, about 70% to 90% of athletes successfully return to sport. In contrast, Achilles-tendon surgery in rats did not offer any advantage over nonoperative treatment, whereas in another study initial functional and biomechanical impairment improved consistently throughout the study.

The last stage of acute-injury healing is referred to as collagen remodeling and takes place 6–12 months postinjury. The initial scar formed in the preceding stage is very fragile, with disoriented collagen fibers. In response to internal and external influences, the scar differentiates to become quasi-tissue-specific. Synthesis and lysis occur to change the appearance of the scar to fairly flat and rosy. To do this, the rate of collagen turnover increases at an accelerated rate at the injured site, as well as in surrounding uninjured tissue. Randomly deposited scar tissue, which is type III collagen, must undergo a process to rearrange or reorient into the usual collagen-fiber orientation. During this process, a linear pattern emerges with organized cross-links to produce a strong but mobile scar of type I collagen with intramolecular bonds between the fibers. Collagen reorientation and cross-linking contribute to a continued slow increase in tensile strength, reaching 70% of the initial strength in 6 months to 2 years. Eventually the rate of collagen and scar-tissue production decreases, indicating a minimal histological difference from the normal tendon.

The strong, dense, parallel-fibered collagenous structure of tendons and
ligaments is difficult if not impossible to restore. The scar tissue in a remodeled tendon almost always differs from the original connective tissue in having fewer cells, fewer blood vessels, and a large number of loose and disorganized collagen fibers. Scar tissue also affects the physical properties of collagen and the musculotendinous unit. It creeps more than the normal tissue structure. The musculotendinous unit then operates at a disadvantage, making it less capable of storing energy in stretch for recovery during a subsequent rebound.

**Chronic Injury.** Little was known until recently about the changes a tendon undergoes during chronic injury. One can only assume that the repair process is similar to acute-injury tendon healing, based on microscopic and biomechanical testing from animal studies. Another consideration is that classification of a chronic tendon injury might reflect what is happening to the tendon and tissues surrounding it. Inflammatory disorders of the tendon sheath or tendon are classified as tenosynovitis, peritendinitis, and tendinitis. A degenerative process of the tendon without inflammation is described as a tendinosis. If one is not sure whether to classify as either tendinitis or tendinosis, *tendinopathy* is used. In some cases, the clinician can rely on clinical signs and symptoms during the clinical evaluation, such as swelling and crepitus in a tenosynovitis injury. Signs of tendinosis include tendon thickening, thickened nodules, and a soft feeling in the tendon when palpated as compared with the other limb. In most cases, however, diagnostic tools such as ultrasonography and MRI or histology to analyze a biopsied tendon after tendon rupture appear to be more specific and sensitive to this pathology-based classification system.

In the chronic stage, the healing tendon is weaker than the uninjured tissue secondary to tissue defects and poorly aligned immature collagen fibrils. In tendinitis there might be a longer period of tissue inflammation and cellular proliferation. This begins with an initial injury and is maintained by a repetitive activity that does not facilitate maturation of injured tissue because of the resulting breakdown. Symptoms (ie, pain) might cause the individual to rest the injury, providing a period of immobilization. This provides a time for the injured tendon to continue the repair process. When symptoms subside, however, activity often resumes again before the healing process is complete, putting stress on the poorly organized tendon fibers. Thickened nodules found in the tendon support the proposed model of repetitive injury contributing to immature tendon scars.

The pathophysiology of these injuries might be related to continual leakage of plasma proteins, which can lead to the gross appearance of chronic tendinitis as dull gray, friable, and often edematous. The chronically inflamed paratendineal tissue, as is noted in Achilles tendinitis, does not seem to have enough capacity to form mature connective tissue. Normally ordered tendon fibers are disrupted by an invasion of fibroblasts and vascular, atypical, granulationlike tissue. There is increased catabolism, decreased oxygenation in the inflamed paratenon, massive formation of
connective tissue, fat necrosis, vascular proliferation, and marked vascular degenerative alterations.\textsuperscript{15,19,21,48}

In the acute inflammatory stage of tendinitis, tenosynovitis, or peritendinitis, there is a mild sprinkling of chronic inflammatory cells in supportive fibroadipose connective and skeletal-muscle tissue, along with edema and exudation.\textsuperscript{15,50} If the acute stage becomes chronic, new or organizing granulation tissue, lymphocytes, and early to late scar tissue appear, as well as pain. Kvist et al\textsuperscript{50} noted that typical morning pain and stiffness result from disturbances of blood supply during night rest and the impairment of the gliding function of the chronically inflamed tendon.\textsuperscript{15} If the condition becomes pathological, secondary fibrosis, exostoses, tendon thickening, decreased vascularity, tendon calcification, cellular atrophy, and cell death will predominate.\textsuperscript{15,19,49-51}

Ultrasound examination of tendons provides a good picture of the actual inflammatory and degenerative process.\textsuperscript{52} In acute tendinitis the tendon volume increases, echogenicity decreases, and the contours are blurred. In chronic tendinitis the tendon is thickened, it is inhomogeneous and hypoechoic, the contour is altered, and intratendinous calcification is noted.\textsuperscript{53,54} Although MRI\textsuperscript{55} can be performed, ultrasound provides a structural analysis and a dynamic interactive examination.\textsuperscript{54} In addition, ultrasound is fast, noninvasive, inexpensive, and readily available.

In degenerative tendinosis, pathomechanics are related to aging and mechanical stress. Inflammation is usually not present.\textsuperscript{40} There appears to be disintegration of collagen fibers\textsuperscript{22} with accumulation of granulation tissue and myofibroblasts that might contribute to joint contractures and impaired circulation.\textsuperscript{15,19} The number of fibroblasts and metabolic activity increase, as indicated by the pronounced roughened endoplasmic reticulum.\textsuperscript{15-17} Degenerative changes are noted in the matrix, as well as inside the collagen fibers, with the occurrence of unequal and irregular crimping and degenerated type I collagen, along with the accumulation of lipid cells.\textsuperscript{15,18} Pain evolves from excessive cyclic loading, which results in matrix molecular damage, loss of tissue strength, resultant increased deformation with loading, and stimulation of mechanoreceptors.\textsuperscript{15,19,45}

The injured structures undergo a repair process after injury, as evident by inflammatory cells and fibroblast proliferation. These reparative attempts, however, perhaps based on impaired circulation\textsuperscript{14} or reinitiation of injury by overuse, do not result in true healing or regeneration of the degenerated tissue. During the reparative process, loose granulation tissue later turns into scar tissue, making the pathological changes permanent.\textsuperscript{19} This pathological condition also causes local and distant changes in the area. Local changes involve flexibility and weakness in the involved muscle–tendon group. In distant changes, there are alterations in strength and biomechanics in other parts of the kinetic chain in which the affected tendon participates. This in turn can lead an individual to substitute activities to compensate for the injury.\textsuperscript{56}
Chronic injuries might benefit from surgery. Guidelines to indicate surgery are inadequate relief from the exercise sequence or chronic pain responsiveness for 6 or more months. During surgery, degenerative tissue is excised from the tendon structure. Afterward, the tendon is sutured or left to heal in a lengthened position. Other methods have included percutaneous longitudinal incisions and tenotomy in the Achilles tendon and ultrasound-guided percutaneous longitudinal tenotomy in the patellar tendon. Frequency of surgery increases with age, duration of symptoms, and tendon pathology. Twenty percent of surgeries for Achilles-tendon overuse injuries require a reoperation, and 3% to 5% of these patients do not return to their previous levels of activity. With patellar tendons, there is a 60% to 80% likelihood of return to sport. Immobilization is required, so this might add to other problems and increase the time to recovery by up to 8 months.

**Immobilization**

Non-weight-bearing or immobilization causes many changes on both the tissue and the cellular level. Ground substance and collagen, as well as muscle and bone, are affected. Reductions in the mechanical properties of tendons, decrease in tendon strength, and increase in collagen turnover are evident. Prolonged immobilization diminishes joint lubrication, contributing to fracture, disease, atrophy of cartilage, capsular contraction, chronic circulatory disturbances, and joint stiffening. When the synovium has been damaged, treatment should aim at early restoration of movement in order to keep adhesions and joint stiffness to a minimum. Unfortunately, rigid immobilization from internal or external fixation is not required for these changes to occur. If a joint is not moved through its full normal ROM or periarticular connective tissue and muscle are not stretched to the fullest, stiffness will result.

Increased turnover rate from immobilization, along with the synthesis and lysis of normal turnover, is observed in collagen. Ligament and periarticular structures, however, react differently. Periarticular tissue stiffens, whereas ligaments weaken and lose stiffness. This in turn produces a ligament that is less able to tolerate stress. Ground substance also affects the histology of the collagen structure. A significant decrease is found in the water content and glycosaminoglycans, causing a change in gel:fiber ratio and a decrease in tissue viscosity. This in turn leads to a decrease in lubrication and the distance between the fibers. The loss of glycosaminoglycan buffer between collagen fibrils facilitates the synthesis of increased cross-links at strategic points between adjacent collagen fibrils. Even though these cross-links provide strength, they limit ROM, leading to hypomobility and, in turn, contracture.

Immobilization causes a weakening of the musculotendinous unit by inducing resorption of collagen at the tendoperiosteal interface, as well as within the tendon and muscle themselves. There is a loss of contractility,
viscoelasticity, and strength of the muscle after immobilization. These losses are especially evident when activity is resumed. Further immobilization of the foot and ankle triggers a cascade of negative effects on skeletal muscle. These include muscle atrophy, loss of myofibrillar and sarcomal protein, and adaptive shortening of muscle and periarticular tissue (contracture). Along with a decrease in muscle activity comes diminished motor-discharge frequency or recruitment and synchronization, with a shift to a phasic pattern of firing. Circulatory changes are also effected, with evidence of diminished blood flow.

Exercise and Therapeutic Exercise

Greater collagenous strength and hypertrophy would help prevent injury in all tissues, provided that normal ROM is maintained. Increased tension loading can make cells in fibrous tissue structures increase the amount of collagen in their cross-sections in adults and in children. Adding collagen to a structure’s cross-section correspondingly increases its thickness, stiffness, and strength. The point beyond which stressing repairing tissues results in further damage rather than an adaptive response of functionally stronger connective tissue has not been defined. It should be noted that too aggressive a move to begin vigorous sports that one is not accustomed to could result in a spontaneous rupture of a tendon. In this case, microdamage occurs more rapidly than the relatively sluggish but presumably normal biologic repair processes. Clinical features are relied on to watch for reinjury, and any signs or symptoms that suggest a worsening of the injury are a clear indication to modify the rehabilitation program.

Results of exercise programs reported in the literature are variable. Studies on manual and resistive exercises to prevent tendon and muscle atrophy, promote intrinsic healing, and encourage longitudinal reorientation of adhesions associated with extrinsic healing have been reported. Most dealt with early mobilization and ROM after injury in the laboratory, physical-therapy, and athletic-training clinical settings.

Mechanical exercising profoundly influences the mature intact tendon or ligament. Woo et al noted in a study on healing flexor tendons in dogs that strength after immobilization was 21% of that of the intact tendon, whereas passive motion resulted in an increase of up to 33%. The application of tension as noted in other studies caused an increase in tensile strength during healing. Hypertrophic scars and keloids found at the injury site are the result of normal synthesis and inhibited lysis if early controlled motion does not begin in uninvolved joints. Scar remodeling is most successful when tension is applied in low loads and long durations with a gentle stretching technique. Controlled mobilization has been found to be superior for revascularization, muscle regeneration, metabolic processes, orientation of muscle fibers and tensile properties, and prevention of scar formation.
Manual massage of a scar facilitates fiber alignment and might increase tensile strength. Hardy\textsuperscript{13} noted that tendon repairs left immobile over bony fractures would ultimately resolve into bony adhesions encasing the nongliding tendon. Fascia, skin, tendon, ligament, cartilage, capsule, and bone have all been shown to lose tensile strength and normal collagen array with immobilization and stress deprivation. The recovery curves for tissue experimentally immobilized for 2–4 weeks reveals that reversal requires months to complete and often is never successful. Striving to maintain the flexibility of the scar during the healing process helps by applying the correct stress to the correct tissue at the correct time.\textsuperscript{13}

In the beginning stages of an acute injury, immobilization might be necessary to decrease swelling and lessen stress on a weakened structure.\textsuperscript{13} When a tendon is completely ruptured (grade III), surgery and immobilization occur within a time frame based on the surgical procedure and the tendon involved. If, however, the tendon is slightly stretched (grade I), all that is necessary is to begin a static stretching program, active ROM, and resistive exercises of an isotonic nature after modalities. Somewhere in between (grade II) indicates to use passive ROM first so as not to put stress on a weakened tendon and to allow for edema reduction. Static stretching and active ROM then follow with the addition of proprioceptive-neuromuscular-facilitation static-contraction activity. Eventually resistive exercises are added with the use of surgical tubing, dumbbells, or ankle weights, based on the body part injured. Whether the injury shows slight or complete tearing, early mobilization should be performed. This is not only to decrease edema but also to provide the stimulus for collagen-fiber realignment and maturation.\textsuperscript{4,7,38,39}

A low load over a long duration should be applied to maintain flexibility and prevent the formation of adhesions on normal tissue in stage 2 of healing.\textsuperscript{13} Leadbetter\textsuperscript{45} noted that the accumulation of repetitive scar adhesions, degenerative change, and adverse effects in chronic microtrauma imply that recovery will be slower. Again, a period of vulnerability to reinjury results, which is increased when conventional anti-inflammatory measures and reduction of pain are applied without regard for the lack of adequate structural integrity.

In the chronic stage of pathoanatomic tendon classification, rest or modification of activity might be the first concern. A second concern is that the athlete resume normal activity during the healing process and not after, as is the case with macrotrauma injuries.\textsuperscript{66} The last concern is that realistic goal setting should be used to prevent patient frustration that will occur at various points during rehabilitation.\textsuperscript{67}

Tendon maturation and alignment are important because the structure already demonstrates immature collagen. The objective is therefore to regain or improve strength, ROM, and endurance.\textsuperscript{66} To assist with ROM, Cyriax massage, augmented soft-tissue mobilization (ASTM),\textsuperscript{16,17,68,69} and active ROM are included. Studies of ASTM in rats\textsuperscript{6,17} have indicated an increase in fibroblast recruitment and healing in chemically induced tendinitis. In
the treatment for patellar tendinitis, after 6 weeks of using ASTM, 100% of pain and functional impairment resolved. In contrast, symptoms in the traditional therapy group (not using ASTM) resolved by only 60%. When the remaining 40% crossed over to the ASTM group, only 50% of that group had resolved.

For strength improvement, progressive eccentric exercises have been advocated since the 1980s for acute and chronic tendon injuries, but the efficacy of this exercise was not studied in clinical and experimental research before a few years ago. The success of eccentric exercise appears to be based on strengthening the muscle–tendon unit to withstand the higher force experienced during eccentric exercise. This results in decreased strain with joint movement and increased tensile strength of the tendon. The focus is to train individuals to activate muscle at the appropriate time to decelerate body segments, thus decreasing the magnitude of the force of the decelerating limb and thereby decreasing the average force transmitted through the tendon during movement. Functional eccentric-contraction exercises might decrease the force experienced during repetitive activity and decrease the risk of reinjury. Despite its effectiveness, there has been no consensus as to when eccentric exercise should be incorporated in a rehabilitation program. Eccentric training is started when there is no disabling pain evident during treatment and continues for a period of 3–6 months.

Nonsteroidal Anti-Inflammatory Drugs and Therapeutic Agents

Nonsteroidal anti-inflammatory drugs (NSAIDs) are used in the treatment of acute and chronic injuries. Examples of these drugs are aspirin and ibuprofen. In the acute inflammatory phase, NSAIDs prevent the release of specific chemical mediators that lead to the signs and symptoms of inflammation, as well as increased circulation and permeability of the cell membrane. In chronic musculoskeletal injuries NSAIDs are used to help control pain and inflammation that might result from therapeutic exercise. Limited studies on NSAIDs have been done with chronic conditions. The effect these drugs have on collagen healing has been studied in some of the research cited in this section.

Carlstedt reported that indomethacin treatment increased the tensile strength in developing and healing plantaris longus tendon in the rabbit. The mechanism for this increase was probably an increased cross-linkage of collagen molecules and accelerated maturation of collagen. Viidik came to the same conclusion regarding healing of skin wounds, and similar benefits were noted by Dahmers et al in a study on ligament healing. Caution was noted, however, against translating these results to humans because of the half-life differences of drugs with studies done on small animals.

A negative claim about the use of NSAIDs is that if taken during the acute
inflammatory phase, they might delay healing because of their effect on the chemical mediators and other structures.\textsuperscript{10,11,20} Chemical mediators are needed to increase cell-membrane permeability and circulation, to bring white blood cells to the area to help remove debris, and to help reduce excessive edema. If this edema is not removed, it can lead to a poor connection between the torn ligaments, which will result in a scar with low tensile strength and early failure in the load-deformation curve.\textsuperscript{37} When NSAIDs were administered in the early stages to decrease pain and edema and increase ROM for 7–14 days, however, healing was slightly more rapid and inflammation was slightly decreased in the treated patients, with return to practice occasionally faster than in the placebo-treated group.\textsuperscript{74}

Studies on animals and humans vary in regard to findings on delays in the healing process. Results from these studies\textsuperscript{10,11,20} are inconclusive as to whether NSAIDs should be given during the first 12–24 hours after an injury. The risk of long-term compromise of structure and function of the injured tissue, however, might outweigh the short-term benefits.\textsuperscript{12} Future studies should include randomized double-blind and placebo-controlled clinical trials within the first 24 hours of injury.\textsuperscript{12} From clinical observations, NSAIDs have not produced any serious short-term side effects except for gastric irritation and slightly increased body temperature. The dosage and the type of NSAIDs should be prescribed on an individual basis in conjunction with other therapy procedures.\textsuperscript{11,12,45}

In the case of tendinitis and other overuse injuries, corticosteroids are also used. Results from animal studies tend to vary because dosage, duration of therapy, location of injection, and type of drug are not always similar.\textsuperscript{2} More side effects appear to be evident with the use of corticosteroids. Tensile strength of the tendon is decreased, and production of collagen and ground substance is reduced.\textsuperscript{4} Degenerative changes are noted in the tendon and paratenon with a slowing of blood flow, which can be detrimental because of the already reduced blood supply to the area. If an injection is given, it should not be into the tendon, which would lead to rupture,\textsuperscript{75,77,78} but rather in the tendon sheath. Short-term effects of an injection in the peroneal tendon of rats for 24 days, however, included an increase in tensile strength and maximal load and stiffness.\textsuperscript{75} Prednisone injections in knee joints for 24 days resulted in decreased strength of the bone–ligament interface with reduced tensile strength, even though diminished adhesions were evident. The long-term effects indicated that within the first 2 weeks there was a rapid increase in stability of collagenous tissue. Nonetheless, inhibition of protein synthesis leads to a progressive thinning of the collagen later on.\textsuperscript{2}

NSAIDs, when taken on a chronic basis, should be used to provide some relief from pain and inflammation resulting from therapeutic exercise used during the treatment phase.\textsuperscript{4,7,20} Because of cellular and vascular fragility and lack of oxygen, any decrease in swelling would put less stress on the injured area. Improvement in the healing process has not been studied, however, and in some cases inflammation is not present.\textsuperscript{40} Chronic tendon
injuries can take several months to heal, and most NSAID studies have a short follow-up period. At no time should steroid injections be given on a short- or long-term basis, because they would result in a reduction of collagen and ground substance. In addition, because the tendon becomes thicker and nodules appear where there is microtearing, the decrease of pain and symptoms might encourage an athlete to continue the injurious activity. This could result in eventual rupture or further microtearing. In conclusion, the decision to use NSAIDs or corticosteroids should be based on the individual case and involve a physician.

**Therapeutic Modalities**

Several treatment modalities are available to allied health-care providers to treat musculoskeletal injuries, but despite these, many injuries can leave an individual with persistent pain and dysfunction that can prevent a return to full activity. In some cases, surgery might not be an effective or reasonable option. Studies on tendinitis and other soft-tissue injuries in humans have been limited and are based solely on results from MRI, ultrasonography, and return to competition after treatments with anti-inflammatory drugs, ultrasound, phonophoresis, cross-friction massage, ASTM, and eccentric exercise. Studies on rats and rabbits have provided interesting results. An increase in fibroblasts in rats with chemically induced tendinitis was seen after ASTM. Multitherapy treatments such as combined laser and electrical stimulation on a ruptured Achilles tendon and combined ultrasound, electrical stimulation, and laser effected biochemical and biomechanical changes, but the treatments tend to work best individually. Gross noted that galvanic current has been used in animals whose tendons have been transected and sutured. It resulted in increased ultimate strength of the tendon in both in vitro and in vivo studies. Nessler looked at regenerating rabbit flexor tendons cultured in a cell-free medium. A stainless-steel electrode was used to provide a high electric-current medium. It was noted that the experimental group showed positive effects as early as 14 days. Those effects were increased cellularity, change in cell shape, epitenon proliferation and bridging of the gap, appearance of delicate new collagen fibrils, and capping of the free ends of the tendon. The author concluded that the application to humans should await further optimal stimulation patterns. Placing electrodes directly over the tendon stump revealed that the anodal stimulation created stronger tendons than did cathodal or no treatment when measured 14 days after tenotomy and suture. Study results conflict in regard to positive and negative effects of ultrasound. Ultrasound has been used in animal models with reported results of an increase in ultimate strength and energy absorption in healing tissues. In some studies, however, the breaking strength was less than 1% of normal. In others there was an increased rate of collagen synthesis,
more fibroblasts evident during a 2- to 3-week treatment protocol, more morphological changes, and increased breaking strengths. Indirect ultrasound using a stationary sound head at 1.5 W/cm² increased the rate of repair of injured Achilles tendons, as indicated by increased breaking strength. The authors concluded that the increased collagen synthesis might be involved in the mechanism when tissue repair is enhanced by ultrasound treatment.

Phonophoresis and iontophoresis are used in addition to other suggested treatments. Klaiman et al compared the effects of ultrasound and phonophoresis and concluded that ultrasound could be used to reduce pain and pressure sensitivity in their study of patients with tendinitis. Adding a corticosteroid to the ultrasound gel did not change the efficacy of the treatment. Gross used phonophoresis to administer anti-inflammatory agents locally instead of systemically. Iontophoresis is another way to administer medication locally. In this case, ions of medication are driven into the body by using an electrical current. Multiple treatments of ultrasound, electrical stimulation, and laser indicate positive results in that they enhance collagen synthesis, but they only present a trend toward improvement in the gross biomechanical properties. The beneficial effects of ultrasound and laser photostimulation, however, might counteract one another when applied together.

An alternative treatment for tendinitis that is appearing in the literature is the use of electromagnetic fields (EMFs). EMFs have been used to facilitate bone healing for a number of years. Only recently has EMF therapy been considered for use in the treatment of tendinitis and other soft-tissue injuries. Electromagnetic stimulation was shown to increase relative histologic maturity, restore stiffness and failure strength earlier, and return collagen content toward normal unoperated values sooner in the healing ligaments. Lee et al also indicated a positive effect in the early stages of healing with the use of EMF fields for Achilles tendinitis. Sandrey et al used pulsed EMFs with tendinitis and reported a trend evident with the EBI field (proprietary bone-healing field) in comparison with a geofield (Earth’s magnetic field), 60 Hz sinusoidal and sham sinusoidal (control, no field), as a viable alternative for accelerating healing of chemically induced Achilles tendinitis in rats. Comparisons of types I and III collagen were made at 14, 21, and 28 days. Greenough, however, concluded that the configuration of pulsed EMF used in their study had no influence on adhesion formation or repaired strength of healing tendons in the rabbit.

Riuenburgh stated that there are 3 facts to consider when using physical modalities to treat tendon injuries in humans. First, there are a variety of techniques to obtain desired effects. Second, timing is an important factor in intervention and use of physical modalities to evoke an appropriate response. And third, tissue response is an unknown factor because of a lack of research and inconclusive results, forcing us to decide on an individual basis.
During the inflammatory stage of an acute injury, modalities of choice are cryotherapy, electrical muscle stimulation, and intermittent compression\textsuperscript{38,39,66} to control edema formation and relieve muscle spasm and pain. These treatments can be used more than once a day for at least 20 minutes at a time. Both electrical muscle stimulation and an intermittent compression bladder, using an alternating current, create a pseudo muscle pump to involve the lymphatic system in the drainage of edema.\textsuperscript{37} To assist in edema reduction, elevation with light circular massage in the area should be performed because of the fragility of collagen. Hardy,\textsuperscript{13} Matthews,\textsuperscript{35} and Wilkerson\textsuperscript{37} have alluded to the fact that if edema is not removed from the area, the torn fiber ends will not be aligned and an even larger scar will form.

During the second and third stages of healing, continued use of ice prevents inflammation after rehabilitation sessions. Deeper massage and electrical muscle stimulation are introduced to work with muscle strengthening. Thermal therapy to relax tendons and increase circulation is introduced in the form of contrast baths, warm whirlpools, and hydrocollator packs. This last technique should be introduced gradually so as to not cause further swelling or put stress on an already weakened vascular structure. To maintain scar-tissue flexibility, thermal ultrasound is introduced during late stage 2 and early stage 3. A direct technique is used with the sound head moving at an intensity of 1.0–1.5 W/cm\textsuperscript{2}. Gieck and Saliba\textsuperscript{66} suggested that 1.5–3.0 W/cm\textsuperscript{2} be used depending on the depth of the tissue being treated. Chan et al,\textsuperscript{91} however, felt that the effective radiating area was more important than dosage and intensity and should be related to superficiality of the structure whether using a frequency of 1 or 3 MHz. Adhesions between tendon and adjacent tissue can be the source of continuous tissue irritation and inflammation. Therefore, Cyriax massage is used after ultrasound treatments to release collagenous adhesions to adjacent tissue and to facilitate increased blood flow.\textsuperscript{4,7,38,39}

Chronic tendon injuries might not respond to treatment as well as do acute injuries. Inflammation, limited healing, and fiber degeneration all present an unlikely prognosis for complete repair. In addition, if there is a decrease in water content, the ground substance will not be able to separate the collagen fibers, thus allowing adhesions to form. The tendon is usually weaker with secondary tissue defects, a poor blood supply, lack of oxygen, and edema. This could lead to rupture if excessive force is placed on the tendon.

When the chronic tendon injury is first evaluated, ice should be used before and after treatment, then only posttreatment in the later stages. Eventually, heat is introduced at the beginning of treatment sessions to relax the tendon and increase circulation to the area. Gross\textsuperscript{7} suggested the use of phonophoresis to administer an anti-inflammatory agent, whereas Gieck and Saliba\textsuperscript{66} and Hunter and Poole\textsuperscript{92} suggested iontophoresis and
phono-phoresis. Both provide a local effect, as opposed to the systemic effect of oral medication. Electromagnetic fields might be a viable alternative treatment as soon as the FDA approves them. To further prevent adhesions and inflexible scar tissue, ultrasound, ASTM, \cite{16,17,68,69} and Cyriax massage \cite{66} are used. Gross' suggested 3 times per week after the first 1–2 weeks for Cyriax massage, with the technique being performed in a transverse direction. If the chronic tendon injury does not improve with rest or modified activities and therapy, surgery might be an alternative. \cite{4,7,45}

**Future Directions**

This review presented the factors that affect the biomechanical properties of tendons, such as maturation and aging, injury and healing, immobilization, exercise, medications, and therapeutic modalities. It also presented solutions for the rehabilitation of acute and chronic tendon injuries. As can be seen from the literature and in the clinical setting, acute tendon-injury rehabilitation is well represented and accepted. In chronic tendon injuries the efficacy of treatment does not uniformly support the premise that currently used treatment methods significantly change the natural history of these problems. \cite{20,40} Perhaps this is related to which pathoanatomic tendon classification is used and what is actually happening on the cellular level. If a true “tendinitis” is not present, \cite{22,40} how can clinicians differentiate between tendinitis and tendinosis and, in turn, determine the treatment? There is a need for additional research on the pathogenesis of tendinosis and other chronic tendon disorders. Imaging modalities might help in the differentiation process, as well as clinical rating scales, but there is a need for guidelines and criteria. In addition, clinical research is needed to determine the management options for tendinosis. These should include but are not limited to therapeutic modalities, NSAIDs, eccentric strengthening protocols, and surgical options.

**Conclusions**

In conclusion, early treatment of acute tendon injuries should begin on day 1 with cryotherapy to decrease inflammation. Deep thermal modalities should be added in stages 2 and 3 of healing to help reduce edema and scar-tissue adhesions. Exercise and mobilization should be limited in the first stage of healing, with gradual progression in the second and third to create a strong and viable collagen fiber with intramolecular cross-linking.

Treatment protocols for chronic tendon injury are not as easy to determine. If inflammation is not present, the focus of treatment should be changed. Therapeutic modalities and exercise should be incorporated to enhance type I collagen alignment and intermolecular binding of collagen fibers. Mobilization should begin immediately, whether using Cyriax or
ASTM, as tolerated by the individual. Flexibility and eccentric strengthening exercises should be added on an individual basis, avoiding placing stress on the healing tendon. At no time should NSAIDs or other therapeutic agents be the sole treatment plan.

References


