

THE NATURE OF TENDONS AND LIGAMENTS

A range of information in this chapter provides insight for the entire topic. Material here on tendons/ligaments is vital for understanding injury, recovery, and the therapy I am using. The story of the dancer, Julia L., in Chapter 1 refers to my work with adhesions and laxity of her injured tendon and the alignment pattern of its fibers—and a steady recovery process. These physical changes are given a foundation in this chapter.

The material here reveals much promise for the potential of these connective tissues to heal. The account starts with basic structure and function. Following are standard medical views and then the perspective of innovative theory and research. This introductory preview and a chapter summary will acquaint less technically-oriented readers with the essentials of the material.

Grounding in the basic structure begins with a view of collagen fibers, cells (fibroblasts), and ground substance—building blocks of tendons and ligaments. This proceeds to their essential properties such as visco-elasticity and tensile strength. The passive structures seen in the conventional model do not produce movement; when stretched past a certain point the tissue cannot regain its normal resting length and will remain lax. When the process of injury becomes serious it results in major tearing of fibers, adhesions, tissue degeneration, and loss of function. Portrayal of the central, primary elements of inflam-

mation and chronic injury syndrome can inform us as to how they can be reversed.

Following is a section featuring findings from recent scientific research enriching the standard perspective. Intriguing clues in this material lead to avenues through which manual therapy can benefit the injured structures. Normal connective tissue (e.g., tendons and ligaments) continuously remodels itself for regular healthy maintenance. There is increasing evidence that these tissues are equipped to repair themselves from injury by manufacturing new microfibers and large amounts of collagen and ground substance.

Through new information the traditionally held view of the neural function of ligaments is expanded to a more active one; they are involved in complex neuromuscular feedback circuits. Data indicates that the richly innervated, responsive tendons/ligaments play a substantial role in neural control of movement and posture. Research in electromagnetism points to its important bodily influence on internal reporting of and response to injury, along with growth for repair. Connective tissue appears to react readily to this form of energy, which is also linked to the acupuncture system. This has intriguing implications for its healing potential and for treatment strategies, as does research on the properties of cells.

Studies in cell biology yield evidence for contractile capacity of non-muscle cells, including the fibroblasts of tendon and ligament tissue. Cellular contraction and capacity for locomotion indicates strong possibilities of the tissue to generate forces from within itself. Such activity could increase tissue tone when it is weak and also assist reversal of laxity; this lengthened condition of chronically overstretched ligaments is usually thought to be irreversible without invasive procedures.

Other potentially valuable cellular aspects of connective tissue health are found in research on satellite (stem) cells. These relatively simple cells are active in regeneration of damaged muscle. A cellular cycle of despecialization into stem cells, leading to their respecialization

in order to form healthy tissue, may be operative for injury reversal in all body tissues. The conditions that promote regeneration are described as the “plastic state.” This state is a component of a basic bodily healing response, seen in the chapter summary.

This chapter is a backdrop for the cases of the people reported in Chapter 4, and defines the whole topic. The latter part of the material here includes the insightful theories of some pioneering researcher/clinicians in the field. Their views on tendons/ligaments round out the chapter’s picture of relatively dynamic, interactive structures. From my standpoint, there is some fascination with the scientific evidence of vitality and sophistication embodied in these tissues.

Structure, Composition

Tendons and ligaments are dense, regular connective tissue structures (bands, cords, or straps) with mostly parallel fiber arrangement. Connective tissue has much variety, but in general it is characterized by the presence of a large extracellular matrix and a wide dispersion of cells.^{1,2} The *cellular* portion of tendons and ligaments are the fibroblasts that synthesize and maintain the fibers and the ground substance.

The *extracellular* matrix consists of ground substance and fibers. The ground substance is the nonfibrous component of the matrix and is made up of glycosaminoglycans and proteoglycans. These long-chain molecules are linked to the collagen fibers to help form connective tissue. The ground substance facilitates tissue metabolism and provides support, shock absorption, and resiliency. It also decreases friction and attracts and binds water. Water occupies the largest percentage of weight (and space) in all tissues (60% to 80%).³ The varying percentage of water-binding protein contained in the ground substance affects the hydration level of the tissue.

The fibrous component of the matrix is primarily collagen fibers, with some elastin. This is the support framework and comprises 75% to 80% of the dry weight of tendons and ligaments. Collagen is the most abundant protein in the human body; its basic building blocks are

amino acids, primarily proline, glycine, and lysine. Its amino acid chains assemble into long triple helix molecules by hydrogen bonding to oxygen radicals along the chains.

The collagen molecules line up side-by-side to attach by intermolecular bonding, forming the hollow collagen fibers. These fibers are the individual units that also attach by molecular bonding to form fascicles (bundles) that comprise the overall structure of the tendon or ligament.^{4,5} Cross-linkages between molecules and fibrils are electro-chemical bonds important for structural strength of the tissue.

Collagen fibers have a tensile strength approaching that of steel. The parallel arrangement of the fibers is somewhat wavy in the relaxed state, and straight when under a tension pull. Dense connective tissue has more collagen fibers and less ground substance than other connective tissue. Elastin is an extensible substance that forms a small component of tendons and most ligaments. A low percentage of specialized ligaments contains a large component of elastin, which gives them increased extensibility.

Tendons and ligaments are surrounded by a sheath (called a paratenon, and unnamed for ligaments) of loose areolar connective tissue, which facilitates gliding on contiguous structures. In locations involving high-friction forces, particularly in the wrist and hand,

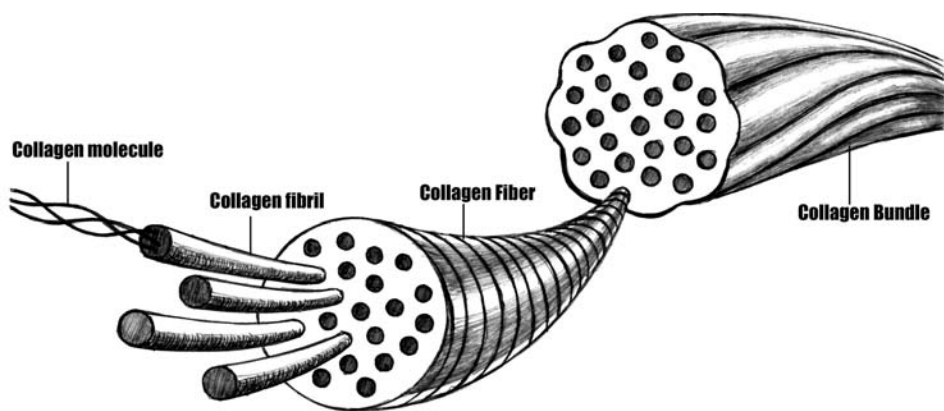
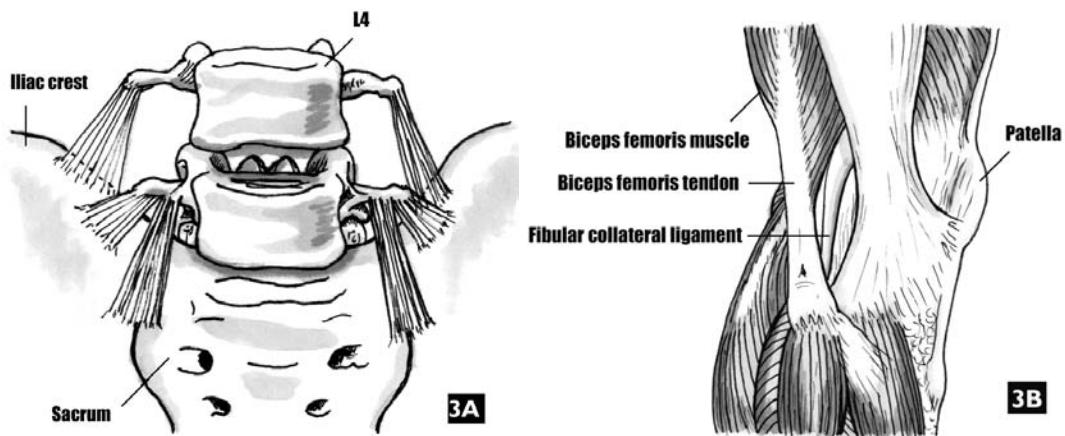


FIGURE 2
Collagen structures in tendon or ligament.

tendons are surrounded by an additional sheath beneath the more fibrous paratenon. This is the epitenon and is a synovial tissue layer that produces synovial fluid for lubrication.^{1,6}

Tendon tissue blends with muscle at one end, and at the other (similar to both ends of a ligament) meets with its bony attachment by blending through the fibrocartilage covering, and then into the cortical bone itself.⁷ The musculotendinous junction and tendon-bone, ligament-bone junctions are often the location of injury. Ligaments are extra-capsular, intra-capsular, or are thickenings of the joint capsule. Both structures are richly innervated and have relatively little direct blood supply.



FIGURES 3A AND 3B

Ligaments and tendons in location. The iliolumbar ligaments (3A) contribute to stability of the lumbosacral area. Therapy for a biceps femoris tendon (3B) injury (Julia L.) was described in Chapter 1.

Properties, Function

The following description of the properties and function of tendons/ligaments represents the accepted view in standard medical theory. Some aspects of this view are altered in the perspective of recent theory and research. Ligaments are described as passive structures whose main function is to stabilize joints, guide their movement, and prevent excessive joint motion.⁷ Tendons are also described in the standard

literature as passive structures. Their main function is to transmit forces from muscle to bone and fascia.

Collagen structures are described as anisotropic, as they don't have equal mechanical properties when loaded in different directions. They exhibit properties (strength and elasticity) that vary according to their orientation in space when a constant force is applied.^{1,8} Due to their parallel fiber arrangement, tendons and ligaments are particularly adapted to resist tensile loads.

Ligaments and tendons exhibit the property of visco-elasticity. Similar to purely elastic materials, they can regain their original shape following deformation, after removal of the deforming load. When an elastic material is stretched it has work done on it, which increases its energy. It stores this energy and keeps it available, which enables the material to recoil back to its original shape. Due to the additional presence of viscosity, visco-elastic materials exhibit time-dependent properties of recoverability. Connective tissues also are temperature sensitive, which affects their rate of creep (slow elongation). To most effectively stretch out (elongate) this tissue, it should be heated and subjected to a large load over a long time period, to produce creep.^{1,6,7}

When tendons or ligaments are subjected to sudden, prolonged, or excessive forces, the elastic limits of the tissue may be exceeded and the tissue enters the plastic range. In the plastic range the tissue is permanently deformed and is no longer able to return to its original state following removal of the deforming force. Ligaments or tendons that are thought to be incapable of returning to their original length after elongation are described as lax, or permanently elongated.^{1,7} After the plastic range is exceeded, with continued loading, the structure reaches the point of failure as the fibers rupture.

The term "load" refers to an external force applied to a structure. Two main factors determine the strength response of a ligament or tendon under loading: their size and shape, and the speed of loading. The greater the number of fibers that are oriented in the direction of loading, and the wider and thicker those fibers are, the stronger the

ligament or tendon. These structures exhibit increased strength and stiffness (resistance to movement) with an increased speed of loading.^{1,7,8}

Tendons and most ligaments have a fairly balanced combination of brittleness (resistance to force without having a plastic range) and ductility (capacity for deformation without failure). They have some brittleness and some ductility. Differing values for failure point are given in many references, but Frankel's and Adams' figures are fairly standard when they state that collagen fiber bundles reach a failure point after exceeding an elongation of either 6% to 8%⁷ or 10% to 15%.⁹ This is typical for tissue with a very high percentage of parallel collagen fibers (tendons and most ligaments). Tendons and ligaments have a high degree of resilience—the capacity to absorb and store energy within the elastic range, and then readily return to the original dimension and release that energy.⁵ Their normal response to intermittent tension (application and release of a tensile force) that is not excessive is an increase in thickness and strength.¹

Connective tissue ground substance can vary from a watery sol-state to a viscous gel-state. It has the characteristic of becoming more fluid when it is stirred up, and more of a solid gel when it is colder and sits without being disturbed. With a higher metabolic rate, motion, and warmth, the energy level of the tissue is raised and the ground substance is more fluid and ductile. With a lower energy level from reduced metabolism, lower temperature, and inactivity, the ground substance is more of a gel and the tissue is less able to soften and stretch.⁵

When tendons and ligaments are subjected to prolonged immobilization, they show disorganization of their parallel fiber arrangement and a decrease in their water and proteoglycan content.^{3,7,10} The tendon or ligament insertion into the bone becomes weakened due to osteoclastic activity destroying their fibers. In some tests, ligaments that had been immobilized for eight weeks showed decreases in strength and measurements of load-to-failure of 35% to 40%.^{3,10,11}

Standard Medical View of Properties, Capacity for Healing

The standard medical model of the properties and healing capacity of ligaments and tendons is that which is most prevalent for orthopedists, sports medicine physicians, and some physical therapists. In this perspective these structures are seen as passive structures that don't inherently produce movement. Their tension is dependent on their length. Ligaments are described as "fixed-length stays."^{5, 6, 7, 12}

Viscoelastic properties are recognized, and when stretched so that their deformation is increased to the point of failure they will not be able to return to their original dimension (length). There is little or no discussion of any other property that would reverse this laxity. Paris states that overstretched ligaments cannot regain tone, making it difficult to correct poor posture.¹³ Subotnick's view is that fibrocytes (mature fibroblasts) of adult tendons are inactive, so that healing is dependent on metaplasia of surrounding fat cells.¹⁴ The standard view is that once an injury is chronic, laxity cannot be counteracted by conservative care and can only be reversed surgically.

A characteristic opinion is expressed by the surgeon who states that "once a ligament is torn it is never going to come back all the way" (even with surgery).¹⁵ Ligamentous fiber rupture is seen as difficult to heal, and is usually treated by surgery. Likewise for a tendon, the greater the extent to which it is torn, the higher the probability that surgery will be recommended.^{6, 7, 8, 13, 14, 15} The medical perspective is that if tendinitis becomes chronic, the tendon gives way to scar and degenerative tissue and can require surgery to clean out the area.

Substantial rupture of the Achilles tendon will most often need surgical repair. Seeking to downplay unrealistic expectations of the possible results of surgery, Garrick states that "there are injuries that just don't get better" (even with surgery).¹⁵ There are variations as to when an injury is defined as chronic. The orthopedist Keene defines a chronic tendon injury as one with over six to eight weeks' duration.⁸ Definitions of chronic injury extend to being over six months' duration, with three or four months as fairly common.

The standard medical view of tendons and ligaments and their injuries determines their treatment in most instances. The clients in my case studies had exhausted the standard care options and had then chosen not to undergo surgery.

Neural Involvement: Standard View and Recent Research Findings

The traditional view of the innervation and resultant function of tendons and ligaments has been altered by information from some research findings since the 1960s. Ligaments are intimately related structurally with the joint capsules and have similar neural receptors. In most of the cutaneous sense organs and joint areas, the receptors are specialized, histologically modified ends of sensory nerve fibers.² They convert energy into action potentials. The four cutaneous senses are touch-pressure, cold, warmth, and pain. Traditionally, the receptors identified by anatomists in joint areas are the Ruffini, Pacinian, Meissner's, Merkel's, and more recently, free nerve endings. All these are mechanoreceptors that respond to tactile stimuli. The tendons contain receptors known as Golgi tendon organs, which have close correlates found in ligaments.²

The traditional view was that ligaments had some of the same receptors contained in joints, and that these articular receptors function to provide some proprioception (sense of body position in space) and a protective role of supplying signals in a reflex loop with neighboring muscles. When the ligament or joint reaches the end-point of movement range it reports through paths to the spine, which in turn effect muscle contraction through stimulating the alpha motorneurons in the muscle—thereby providing a splinting action preventing further joint movement.

Recent research findings lead to a new perspective. Ligaments and joint receptors are now also seen as part of a more complex neuromuscular control system, which also operates through the smaller gamma motorneurons, located in neuromuscular spindles (stretch receptors) embedded in muscles. When the gamma neurons receive appropriate

signals related to position and movement range and rate, they effect contraction of the muscle that they supply.

The gamma loop is a length-regulating reflex mechanism for each muscle that is protective against excessive stretch; it is important for production of accurate, well-coordinated movement and postural control. Its functions have been brought to light relatively recently. Ligament and joint receptor signals have considerable effects on this feedback system.^{16, 17, 18, 19} They affect it throughout the entire range of joint motion, and include rate of movement as another reporting variable in addition to position.^{16, 19, 20}

Swedish neurophysiologists Johanssen and Sjolander report on research findings showing that joint and (particularly) ligament receptors are able to report joint angle throughout the full range of movement.²⁰ The presence of mid-range receptors expands on the older

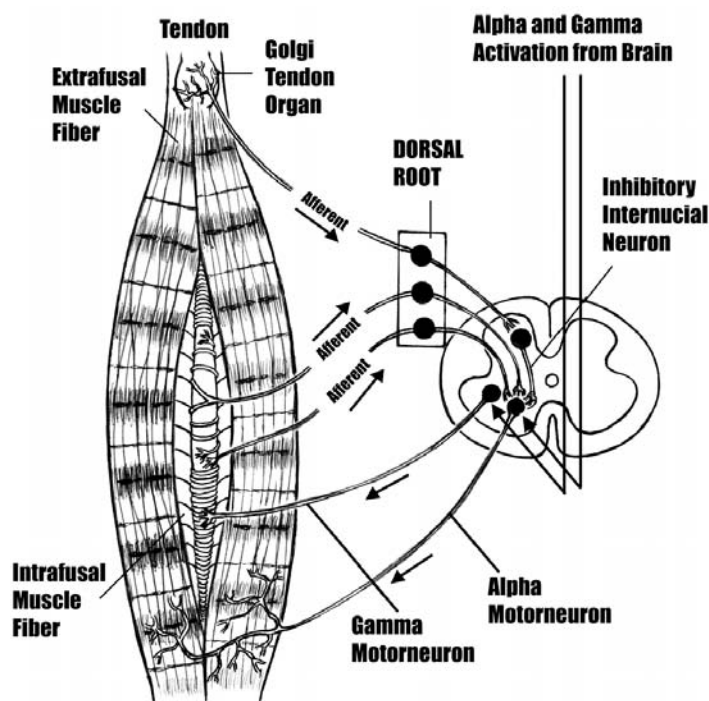


FIGURE 4

Muscle spindle and gamma neuromuscular loop. This illustrates the schema of the gamma nerve fibers supplying the intrafusal fibers of the muscle spindle, alpha nerve fibers to the main (extrafusal) body of the muscle, and their connections with the central nervous system. The drawing indicates the influence of muscle spindle activity on the main muscle fibers.

model that restricted signaling to the endpoints of range, and shows that articular receptors contribute more substantially to movement and position sense than was previously recognized. It has also been shown recently that the complexity and diversity of articular receptors are equal to that of cutaneous receptors.¹⁷ In addition there is now strong evidence of rich

innervation of ligaments with the same nerve types as those in joint capsules, to provide varied receptor flows through the whole range of joint movement.

Nerves are now seen to fulfill an important trophic function of providing delivery of nutrients to peripheral structures such as tendons and ligaments. Korr and other investigators have elaborated this mechanism.

Neurotrophic activity, the delivery of proteins by axoplasmic flow from the nerve cell body along the axon to its target tissue (both anterograde and retrograde), varies in the rate of flow and quality of the axoplasm.^{16, 39} It can be compromised by structural pressures in the nerve root, along the course of the axon, and in the target tissue; it has major effects on the health of tendons and ligaments, which are so richly innervated.^{16, 54} Freedom from excessive structural pressures is necessary for optimal trophic function.

These recent research findings indicate an expanded neural role for ligaments and tendons. Ligament and tendon receptors function to assist smooth coordination of movement and muscular contraction for stability, as well as contributing to position and equilibrium maintenance through signaling to the brainstem and supplying higher centers with input related to more conscious body-image.²¹ Much of the nerve control of posture is located in tendons and ligaments.^{17, 19} In many ways they are highly sensitive structures that play a more important neural role than is portrayed in the traditional view. This role is more responsive and complex.

Injury Process and Result, Tissue Changes

Depiction here of injury and inflammation primarily has conventional acceptance although some of the information is recent (Korr, Simkin, Radin, Viidik). It is a foundation for expansion and alteration of the standard view that resumes in the section on tissue remodeling, through the end of the chapter.

There are some common features and characteristics of the process and result of most tendon and ligament injuries. Injuries are most likely to result from a sudden trauma, or to be more gradual and then be classified as some type of overuse injury. There are also systemically caused injuries from disease such as rheumatoid arthritis or diabetes, endocrine imbalances, or from infection.⁷ A tendon injury is termed tendinitis, a ligament injury usually called a sprain. Inflammation occurs, causing abnormal and impaired function of the tendon or ligament. There is some degree of rupture to the fibers and blood vessels of the structure, and some swelling. Tendinitis can often involve damage to the tendon sheath.

Establishing a diagnosis includes examination for instability of the joint where the tendon or ligament is located, and examination for loss of function.⁸ Radiographic findings can be utilized. Injuries are often classified into three grades reflecting degree of rupture and instability.^{6, 7, 8} In Grade 1 injury the ligament recovers its resting length after trauma and sustains microfailure of some fibers. In Grade 2 the structure doesn't completely recover its length, and there is some instability and a greater degree of fiber rupture. In the Grade 3 injury the tendon or ligament has a region of gross disruption of its fibers, and considerable instability. The serious injury thus consists of significant fiber tearing and loss of function.

Traumatic injury to a tendon often occurs when there is sudden unanticipated stretching of an already contracted muscle, resulting in a tear of its tendon. Likewise, an unanticipated force applied to a ligament incapacitates its afferent signaling to limit joint motion via muscle antagonist contraction. Thus, the joint may undergo a substantial, sudden movement and the adjacent ligament can rupture.²²

Gradual onset (overuse) tendinitis is caused when microfailure of fibers occurs from excessive repetitive loads on a tendon, or when the rest period between loads is inadequate to enable the tendon fibers to regain their resting length. Predisposing influences are temperature extremes, excessive vibration, repetitive tasks, tissue degeneration, or

usage in unusual postures.⁶ Chronic injury processes and the degenerative tendon changes pictured in the “What My Hands Told Me” section of this chapter are important in overuse/repetitive strain problems.

Tendinitis (or ligament sprain) often starts as minute single-fiber tears. When the fibers tear they also swell. This causes them to rub against the tendon sheath, which becomes irritated and can also swell. The capacity for tendon gliding in the sheath is reduced as more and more adhesions are formed. Progression of the problem will result in a greater degree of tearing. It is common to find nodules, fatty mucoid deposits, and marked thickening in the ligament, along with a chronic low-grade inflammatory process⁴ (see Figure 5, “Main Elements of Tendon/ligament Injury”).

Inflammation in the Injury Process

Inflammation is an important aspect of tendon or ligament injury. It is a term used for a large group of normal processes provoked by damage or alien material.^{2, 23} Inflammation is in part a bodily response to injurious agents in order to remove toxic or foreign material. Fiber and blood vessel rupture in injury leads to infiltration of the region with white blood cells, platelets, fibrin, histamines, blood, and tissue fluids.^{24, 25, 26}

Inflammation manifests as swelling, heat, redness, and pain. The acute inflammatory phase lasts from three to seven days after injury. Following this acute phase the result is either tissue repair or adhesion and scarring (see upcoming section on remodeling process).

When the inflammatory process is prolonged, it causes problems for the ligament or tendon that is healing.²⁶ Continued inflammation leads to excessive adhesion, fibrosis, and scarring. There will be inappropriate cross-linking of the fibers which contributes to dense, inflexible tissue. The developing fiber orientation is often random and scattered (not parallel).²³

When lymphatic drainage is inadequate, the continued edema from excess fluid results in decreased circulation from excess pressure.

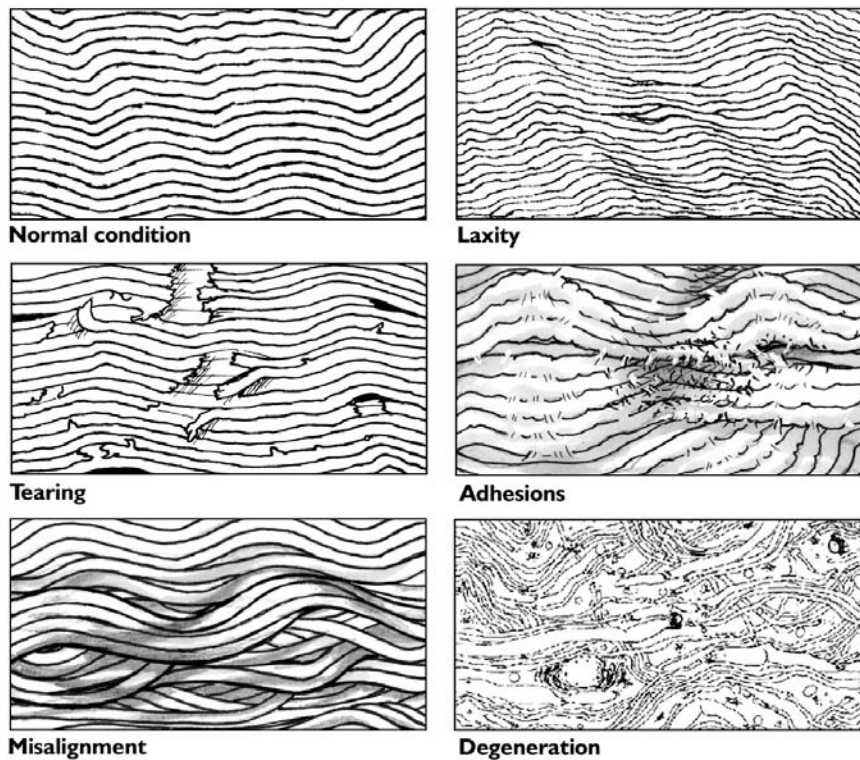
Edema in the joint area disrupts alignment and contact of the bone surfaces that leads to joint instability and consequent increase of ligamentous laxity. The rheumatologist Simkin reports that continued joint inflammation can compromise the capacity for lymphatic drainage, which further compounds the problem of instability.^{24, 27} False strain reflexes caused by long-term inflammation result from excessive afferent nerve input to the CNS. The excessive input is partially caused by musculoskeletal tension imbalances, and also acts to maintain them (see section on neural involvement).²⁸

When a region remains swollen the structures are bathed in serofibrinous exudate. Fibrin, a protein needed in blood clotting, is deposited between tissue layers in tendons and ligaments, and between them and surrounding structures such as sheaths. The fibrin seals these structures in a shorter, less mobile, adhered condition.^{2, 29}

The purpose of post-injury vascular events as part of the inflammatory process is to mobilize and transport the defense components of the blood (leukocytes) to the injury area, and to secure their passage through the vessel walls into the tissue spaces. When this post-injury defense mode is prolonged excessively, it interferes with the normal healing process of the tissue. A self-perpetuating cycle of irritation and inflammatory response leading to further irritation becomes a component of the chronic injury pattern.

Chronic Injury

In chronic tendon and ligament injury, the normal healing process of connective tissue is disrupted. Normal tissue remodeling is prevented by stresses (such as prolonged inflammation, inactivity) and hypovascularity.^{8, 10, 25, 26} Connective tissue tends to become shorter and denser as it heals, unless it has beneficial conditions.³⁰ This contracture and thickening partially results from the increased interfiber bonding in a shortened state. The individual fibers lose gliding capacity and mobility relative to each other as do bundles of fibers, and whole tendons and ligaments relative to their surrounding structures (joints, capsules, sheaths).

**FIGURE 5**

Main elements of tendon/ligament injury, disruption of fibers. A combination of two or more of these patterns would be found in a chronic injury.

The adhesion process of fibers and layers of tissue leads to reduced extensibility and mobility.^{1,5} With poor circulation, hypoxia, and continued tearing, tissue regrowth takes the form of scar tissue as tears coalesce in portions of the ligament.^{10,26} Scar tissue is weaker than the normal type. The water content and amount of ground substance (proteoglycan content) of the tissue is reduced; there is a lessening of its normal ratio of ground substance to collagen fibers, which is another factor causing a denser, less pliant structural situation and impaired metabolism of the tissue. By-products of tissue degeneration and breakdown can form deposits in the ligament, disrupting its function.

In chronic injury the normal parallel fiber arrangement is disorganized, which reduces extensibility and strength. Prolonged low-grade inflammation is maintained along with disturbed lymphatic and

vascular flow. This also causes abnormal metabolic function in the tissue.^{8, 26} Ligaments and tendons can exhibit both hypo- and hypermobility. Often, both these conditions exist in different portions of the same structure. There is a disruption of balanced neural activity, as the tendon and ligament receptors report inappropriate strain signals caused by the irritation of the tissue.^{16, 31} This sets up an ongoing stress on the ligament due to muscular tension, uncoordinated movement, and joint instability; all these factors contribute to general neurotrophic disturbance and impairment of circulation in the tissues.^{16, 20, 31} An in-depth look at degeneration change in an injury process is seen ahead in the “What My Hands ...” section.

Remodeling Process of Connective Tissue

In normal nonpathologic functioning, all connective tissue is capable of healthy remodeling. Rolf asserts that it is in a continuous state of structural reorganization.³⁰ This self-renovation is exemplified by the response of tendons or ligaments to increased stress levels. Tissues respond to fatigue stresses (such as athletic training) by increasing the rate of tissue production (collagen fibers, ground substance). When there is proper vascular function and tissue metabolism, and sufficient rest between loading, the body can adapt adequately.^{1, 7, 25, 32}

It is important for healthy remodeling of a ligament or tendon that the fibers are laid down in proper alignment. Non-excessive, well-directed loads on the structure with adequate rest encourage the healthy alignment of the fibers. The result is a structure that is stronger and also properly deformable, with adequate inter-fiber mobility. Good extensibility is also facilitated by remodeling with a normal ratio of collagen to ground substance. (It is important to have enough ground substance in the proportion.)^{6, 23, 25} Production of ground substance is readily stimulated by movement after a ligament has been immobilized.⁸²

The acute inflammatory phase immediately following an injury (described earlier) is the first of a three-phase post-injury healing process. Acute inflammation lasts from three to seven days and is followed by a proliferative/repair phase.

The proliferative (second) phase includes ingrowth of capillary buds and the appearance of fibroblasts that start to manufacture ground substance and collagen. The combination of new blood vessels, fibroblasts, and extracellular matrix is termed granulation tissue. The proliferation phase lasts until about the 21st day after injury (although it can be longer as the three post-injury phases overlap each other).

The third phase of the post-injury process is maturation/remodeling. It usually starts at 14 to 21 days and can extend a year or more. Collagen production continues and now is greater than that of ground substance. Remodeling includes the chemical cross-linking of the fibers which enables them to gain fuller strength. Type 3 collagen formed in the proliferation phase is replaced by Type 1 collagen which forms stronger fibers.^{23, 26, 103, 105}

There is now increasing evidence that connective tissues are equipped to repair themselves by manufacturing and remodeling large amounts of collagen and proteoglycans. These components are continuously, although slowly, remodeled in the tissue in its normal healthy condition. When an injury occurs, production of connective tissue (along with collagen and proteoglycans) can double or triple. Synthesis of collagen by the fibroblasts is given a high priority (for nutrient supply in the body) during the healing process. Fibroblasts are responsive to extra-cellular influences such as cytokines, growth factors, and inflammatory mediators that allow fibroblasts to maintain and repair connective tissues. Fibroblasts are capable of proliferation to repopulate a region.⁹⁹

Blood Supply of Tendons

In their book, *Tendinitis: Its Etiology and Treatment*, Stanish et al. synthesize research findings on the blood supply of tendons, which had previously been thought to be avascular.¹⁰² The tendon receives blood supply from three avenues, each serving about one third of the tendon:

1. Musculotendinous junction. Small blood vessels divide near the junction and send branches to both muscle and tendon. There is no

direct capillary circulation between muscle and tendon except that some vessels in the outer covering of the muscle (perimysium) traverse the junction.

2. Along the length of the tendon. The supply is either from the paratenon or the synovial sheath of the tendon. The paratenon contains many vessels.

3. Tendon–bone junction. Tendon and bone vessels do not communicate directly. There are indirect anastomoses between tendon vessels and those in the periosteum.

Internal vasculature. Vessels are primarily longitudinal in the endotenon and thus arranged around fiber bundles (fascicles). Vessels are the size of arterioles and are flanked by two veins. Capillaries connect them. The internal vessels are fed by vessels in the epitenon which enter radially.

Vasculature of the tendon or ligament is reduced in areas of friction, compression, and excessive wear. Shearing and other forces can damage the vasculature. Reduced, compromised vascular supply is a major element in tendon injury.

What My Hands Told Me: Clinical Experience and Current Research on Injury and Recovery

My perceptions in 29 years of palpation and observation are in line with the direction of much new investigation in the field. The case study reports in Chapter 4 illustrate my direct sense of damaged and healing ligaments and tendons as intricate, responsive, lively structures. In revising for this second edition, it is exciting to see that many research insights from the last four years further confirm and elaborate a more vital, complex picture of ligament and tendon metabolism, injury, and recovery.

Highlights of current research results in this section of the chapter are as follows:

- Further evidence that tendons and ligaments are more metabolically active than the traditional view. Research focuses on collagen

physiology and production, the cross-linking process of collagen fibers, and the vascular supply of tendons reported in a preceding section. (Much of the newer research here targets tendons.)

- A shift of emphasis regarding tendon dysfunction and injury. The ongoing focus on the inflammation process in tendon injury has been expanded to a shared emphasis with the role of internal degeneration of the tendon. Research now provides a more detailed picture of the changes involved in degeneration and injury.
- As evidence of recent recognition of a somewhat greater healing capacity for tendons (and the need to counteract degeneration), a program featuring exercise has received attention in injury rehabilitation. There has been more investigation into the structural integrity of tendons and tissue changes in the strengthening process.

METABOLIC ACTIVITY OF TENDONS

A recent increased awareness of tendon metabolic process is reflected by the researchers Stanish, Curwin, and Mandel which acknowledges this metabolic activity. They report that only recently has research interest been focused in this area because of long-standing beliefs that metabolic activity was negligible.¹⁰² Several areas of tendon physiology are now more illuminated.

CROSS-LINKING IN COLLAGEN STRUCTURE OF TENDONS, LIGAMENTS

Curwin writes that the vital role of healthy cross-linking for proper strength of the tendon has now been given more importance.¹⁰³ These bonds are essential for adequate tensile strength in the tendon or ligament.¹⁰⁴ Cross-links can be damaged by shearing forces which occur in injuries. The process of link formation and maintenance necessitates adequate enzyme supply to the tissues, as well as sufficient oxygen supply provided by good vascular activity.

The cross-links are enzyme-facilitated chemical rearrangements of adjacent amino acids. They start as bimolecular, and in mature structures progress to trimolecular, a process that can take months to complete. The influences of both exercise and different types of collagen on cross-linking activity are discussed below.

COLLAGEN PRODUCTION, PHYSIOLOGY

The continued metabolic activity over time of tendon/ligament cells, fibrocytes (tenocytes) is confirmed by Jozsa and Kannus.¹⁰⁵ Previously thought to be inactive, fibrocytes are the successor to the more active fibroblasts, although the reverse conversion sometimes occurs. Some major features of recent research on collagen physiology are:

- Curwin's study of the effects of exercise (jogging) shows that tendon collagen cross-linking increases with gradually increasing loads, but actually decreases with intermittent strenuous loads. She reports studies concluding that collagen tissues can be induced to form new microfibrils which can group together to form new fibrils. The new microfibrils can also be added to existing fibrils, increasing their size.¹⁰³ There is now added validation that exercise increases collagen synthesis, concentration of metabolic enzymes, and the size, number, and strength of fibers.^{102, 103, 106, 107}

- Collagen has several types, of which the primary component in normal tendons is type 1 collagen. Type 3 collagen is found abundantly in degenerated tendons and those healing after injury, and is normally less than 5% of a healthy tendon. Type 3 forms smaller diameter fibrils, and during healing converts to type 1, which promotes more cross-linking, forming larger, stronger fibers.^{103, 105, 108, 109}

- Increased load-bearing capacity in tendons results from collagen bundles sometimes following a more complex three-dimensional structure which is not solely in the straight longitudinal alignment. Studies now show spiral and diagonally-oriented bundles in portions of some tendons, which is well suited to their demands for force transmission.¹⁰⁵

- Cellular response to mechanical forces on connective tissue is the subject of some new studies. Injured rat tendons treated with mechanical pressure from solid instruments exhibited fibroblast proliferation. The cells in these tendons also showed signs of fibroblast activation and active collagen synthesis such as increased presence of rough endoplasmic reticulum, larger numbers of ribosomes, and prominent round nuclei.¹¹⁰ Mechanical stretching of fibroblasts also stimulates their proliferation.¹¹¹ While ultrasound treatment produced

regenerative response of connective tissue in some studies, others such as Almekinders' showed no sign of regenerative effect.^{105, 112}

- Ruptured fibril ends in tendons can be rejoined by synthesis of new collagen materials.¹⁰⁵

INJURY PROCESS—RECENT PERSPECTIVES

The role of degeneration is now featured more prominently as a major factor in tendon injury. The earlier parts of this chapter emphasize an inflammatory process in connective tissue dysfunction, and we currently have more insight into inflammation as it interacts with degeneration in ligaments and tendons.

Tendinosis has now joined tendinitis as a description of injury. Tendinosis refers to intratendinous degeneration that can initially be asymptomatic.⁶ It is non-inflammatory although it can interact with and overlap inflammation, and views vary as to sequence. Tendinosis is a process of atrophy-related factors such as vascular compromise, hypoxia, aging, microtrauma, overuse, tissue breakdown, and lack of adequate cell response.^{102, 103, 105, 108, 113}

The two major types of tendon and ligament problems are overuse injury and substantial rupture; degeneration is now thought to be a frequent causative factor for each one. In overuse injury it is common that tissue microtrauma and breakdown (tendinosis) precedes the perception of pain. Pain usually signifies an inflammatory condition (now called tendinitis by some authors). Jozsa and Kannus studied tendons which had spontaneously ruptured within the preceding 24 hours. They found that 97% of the specimens exhibited significant degenerative changes.¹⁰⁵

As pictured earlier in this chapter chronic dysfunction often involves a circle of inflammation, scarring, hypoxia, and failed healing response. The current view expands on earlier findings and includes a sharper focus on the elements of tendon degeneration and chronic disturbance. These elements are:

- Vascular compromise leads to hypovascularity and tissue hypoxia. The result is impaired metabolic activity and tissue nutrition.

Studies show compression from stasis of extracellular fluid and narrowing of the lumina of the arterioles. Deposits of fibrin and thrombus formation are found. Damage to the microvasculature can occur in localized regions of the tendon. Vascular compromise and insufficient oxygenation (tissue hypoxia) receive the highest emphasis as a central factor in tendon degeneration.^{105, 107, 114}

- Fatty deposits can be intracellular, in tenocytes, disrupting cellular oxidation. Lipid deposition also occurs between fibers leading to disrupted alignment and fiber separation and thinning. Unhealthy accumulations of mucoid ground substance deposits lead to similar problematic effects as from the lipid deposits. Portions of the tendon can exhibit a soft, mushy consistency.^{105, 113}

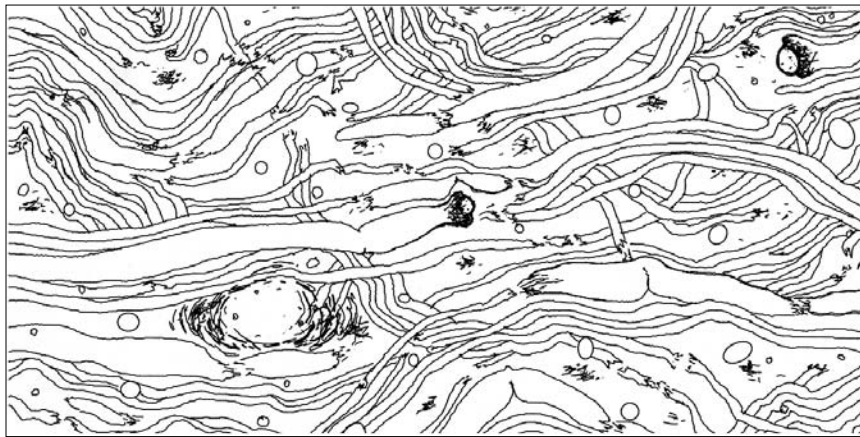


Figure 5A

Degeneration, failed healing tissue. This is a composite showing:

- | | |
|--|--|
| 1. "bubbles" at center and upper right | 6. angulation. Loss of continuity without systematic orientation |
| 2. mucoïd vacuole at lower left | 7. bulbous amianthoid fibers |
| 3. fibrin granular material | 8. variations in thickness |
| 4. lipid droplets (ovals) | 9. loose structures |
| 5. disruption and fraying | |

- An incomplete, failed healing response leads to accumulation of excessive fibrin deposits. These deposits can contribute to major disorganization of the internal structure of the tendon. The immature fibrioid tissue in the tendon as well as buildup of degraded collagen, ground substance, and vascular granulation tissue is sometimes referred to as "amorphous debris."^{102, 115}

- Collagen fiber alterations in the injury process include findings of bulbous fibers as well as thinned, frayed, split, curled fibers. Fibers lose continuity as there are microruptures of tendinous bundles. Strained or ruptured tendon fibers exhibit tapered ends and knot formation, both consisting of denatured collagen. Hypoxic changes in the tendon lead to progressive increase in the dryness content of its collagen fibers, increasing fiber stiffness.¹⁰⁵ Fibrosis, adhesions, nodules, and scarring are common in tendinosis and have been covered earlier in the chapter.

- Cross-linking of fibers is impaired by inadequate oxygenation, enzyme defects, and endocrine response to stress (which may result from athletic overtraining).¹⁰³ Progressive collapse of lateral cohesion from inadequate cross-linking results in more loss of strength and vulnerability in the tendon.

- Cellular abnormalities in a degenerative process include lessened numbers. Decreased cell response capability results from tissue overload and hypovascularity. This diminished capacity of the cells to respond normally is a primary cause of degeneration. The cells (fibrocytes) exhibit altered nuclei and nucleoli, dilated vacuoles, swollen mitochondria, reduced DNA, and dilated degranulated endoplasmic reticulum.¹⁰⁵

- Calcium deposits (hydroxyapatite crystals) appear in the tendon, partly as a result of hypoxia in the tendinosis process. There is also a non-degenerative, very painful, rapid onset condition of the shoulder, termed calcific tendinitis.¹¹⁶ Wolf describes his success in calcific tendinitis with a technique of needling into the calcium deposits to treat this acute condition.¹¹⁷

- Inflammation and its central role in tendon or ligament injury is illustrated in detail earlier in this chapter. Inflammation can cause degeneration and rupture and can also result from overuse and degenerative changes. Recent studies suggest that inflammation can be initiated by the presence of calcium deposits, fibrin deposits, and degraded, denatured collagen in the tendon and its sheath.¹¹⁵ The inflammatory reaction may be an auto-immune response.¹¹⁸ Stanish et al. feel

that pain indicates ongoing inflammation of a tendon/ligament and is the most common measure of tendinitis severity.¹⁰²

OVERUSE INJURY

Overuse is now thought to account for 30% to 50% of sports injuries.¹⁰⁴ A high percentage of occupational-related patients also present with a slow, insidious onset consistent with much overuse tendon injury.¹⁰⁸ All the degenerative tendon changes listed in this section can occur in overuse conditions. Overuse tendon and ligament injury (repetitive strain injury) can be defined as a level of repetitive micro-trauma sufficient to overwhelm the tissue's ability to adapt.¹¹⁹

EXERCISE PROGRAMS FOR TENDON REHABILITATION

Exercise programs to promote tendon injury recovery are now receiving more research interest and validation. This emphasis is consistent with the view that degeneration and its reversal is important in tendon injury. Exercise can be beneficial for promoting collagen synthesis, remodeling, and tensile strength.

Eccentric exercise, where tissue tension is generated as the muscle-tendon unit lengthens (e.g., lowering a weight) is a vital component of some recent programs. Eccentric (lengthening) contraction produces a greater force in the muscle and tendon than concentric contraction. A number of authors feel that many sports injuries occur in the eccentric phase of the activity cycle. Eccentric contraction is more efficient than concentric in that the muscle uses less energy and recovers more quickly.^{120, 121, 122} A 2001 study for treating Achilles tendinosis showed superior short-term results for eccentric calf muscle training compared to concentric training.¹²³

A program featuring eccentric exercise for tendon injuries has been created by Stanish, Curwin, and Mandel at the Sports Clinic of Nova Scotia.¹⁰² This program (described more fully in Chapter 6) has reported good clinical results as has a related program used in Swedish and Danish controlled-trial research studies.^{106, 124} The subjects in both the Swedish and Canadian studies had utilized physical therapy and con-

ventional exercise programs without success. Curwin's article offers perspectives on the appropriate application of their program; her view is that all joint signs should be cleared and the subject should have a pain-free range of motion before starting the program.¹⁰³

Both Peter Edgelow, P.T., Clinical Professor of Physical Therapy at UCSF and Diana Herold, Dance Medicine Director at St. Francis Hospital Center for Sports Medicine, feel that the eccentric exercise program can be helpful for patients at a certain stage of recovery. It could be a useful component of an overall program.^{125, 126} Edgelow, an expert in treating thoracic outlet syndrome, emphasizes for rehabilitation the need to restore coordination and balance as much as strength. He feels that the eccentric phase of movement enhances control and coordination more than the concentric phase.

For areas with more complex biomechanics such as the shoulder, more guidance is beneficial for the patient to do eccentric exercise along the best line of direction. There is a need for more limited range of motion exercise in earlier and/or more severe phases of injury, that can include some eccentric training.¹²⁶ An eccentric exercise program for tendinopathy is presented in Chapter 6.

LIGAMENT INJURY REHABILITATION—RECENT DEVELOPMENTS

Much of the material in this section on research developments is primarily focused on tendons, although it is very likely that a high proportion of findings on collagen physiology and injury/degeneration also applies to ligaments. While we are still waiting for more attention to ligaments in the literature, some recent information includes:

- Reports of success for ankle sprain exercise therapy using an “unloading technique” (harness suspension, incline board) to reduce gravitational force and allow pain-free exercise.¹²⁷
- Balance, proprioception, and strength training reduced risk of re-injury after ankle sprain.¹²⁸
- Friden et al. found impaired proprioception in the knee joint after ligament injury.¹²⁹

- An engineering model study shows the anterior cruciate ligament can be injured differently at varying knee joint angles.¹³⁰
- Loosli and Oshimo's study demonstrates that a thorough exercise and bracing program had equal or greater success compared to reconstructive surgery for effective rehabilitation of anterior cruciate ligament injury in recreational athletes (tennis, skiing).¹³¹ Dancers' knee injuries responded well to specialized Pilates exercise.¹³²

HEALING POTENTIAL—RECENT PERSPECTIVES

A healing capacity of tendons and ligaments that is greater than the traditional view now has further confirmation from researchers. Stanish and Curwin discuss the current reassessment of a long-held attitude that connective tissue healing is "slow and imperfect." Their exercise program is based on acceptance of significant tissue remodeling capacity of tendons.¹⁰²

Jozsa and Kannus feel that recent evidence shows that tendon healing capacity may have previously been underestimated. They describe how denatured collagen and frayed fibril ends in tendons are readily removed by proteolytic enzymes produced by macrophages. The ruptured fibril ends may then be rejoined by the synthesis of new collagen materials.¹⁰⁵ A Scandinavian study reports the regeneration of Achilles tendons after necrosis.¹³³

The material on collagen physiology in the preceding pages includes aspects important to the recovery process. Among them are: the capacity for tendons to develop new microfibrils which form new fibrils or thicken existing ones, fibroblast activation and proliferation in response to mechanical pressure, continued long-term activity of tendon/ligament cells (fibrocytes), and validation that exercise can increase collagen cross linking and synthesis. Exercise increases production of insulin-like growth factor (IGF-1) in tendon fibroblasts. IGF-1 is known to strongly stimulate collagen synthesis and cell replication and seems to mark remodeling activity.¹¹⁴

A presentation on stem (satellite) cell activity in connective tissue appeared in this book's first edition, and is a subject which is now receiving a tremendous amount of interest for regeneration of many types of tissue. Discussion of stem/cell activity is found in the upcoming section on tendon and ligament regeneration.

Electricity, Magnetism, and Tendons/Ligaments

A number of researchers in the last three decades have found evidence demonstrating the presence and influence of electricity and magnetism in connective tissue function. This appears to have an effect on the health status of tendons and ligaments and their capacity for healing from injury.

Orthopedic surgeon and researcher Robert Becker describes a bodily Direct Current system of internal communication, located in the perineural cells that ensheath the entire nervous system including the brain.^{33, 34, 35} Becker traced the DC internal field map as it mirrors the design of the nervous system. This was confirmed by studies using the SQUID device for magnetic field detection.^{33, 36, 37} It is an analog communication system that integrates body processes and controls

the activity of the body's cells by producing DC electrical environments. The analog DC system is more primitive than the digital AC system of nerve impulse conduction, and can provide a relatively steady-state environment interacting with AC activity. Becker and others found this system to have an important role in growth and healing.^{38, 39, 137}

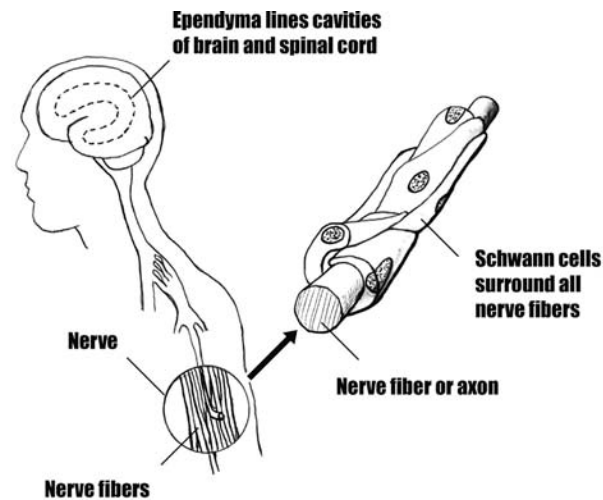


Figure 6
Direct Current conduction system in perineural structures.

The Direct Current circuit flows appear to guide formation of collagen after tissue injury.³⁴ Dr. Becker describes a “current of injury” that is a DC flow from injured tissue that serves as a reporting mechanism. This conveys information of bodily damage to the brain and can evoke a response that is vital to reparative growth and other internal controls (see Figure 8). Magnetic energy was found to be increased at the location of injury.³³ The Swedish radiologist and researcher Nordenstrom also reports evidence demonstrating an electrical current circulatory system in the body switched on by infection and injury.⁴⁰ The cell biologist Lackie’s research indicates that a wide variety of individual cells appear to have the ability to respond to DC fields.⁴¹

Williamson reports on tracing magnetic fields from the body that are produced by activity of potassium ions along cell membranes. He also asserts that ferromagnetic substances in the body can interact quite strongly with the geomagnetic field.³⁷ Becker reports on research showing the influence of the geomagnetic field on bodily growth patterns, stress responses, and biological cycles.^{33, 34}

The physiologist and researcher Valerie Hunt describes a “growing emphasis from biomagnetic field studies on the electrical nature of life.”⁴² Motoyama and Reichmanis have both researched and demonstrated specialized electrical characteristics of the acupuncture meridian system.⁴⁰ Reichmanis found it to be linked to the perineural DC system and showed that acupoints generate DC potentials.⁴³ Hunt’s research found acupuncture-like meridian energy flows in all connective tissue. She describes connective tissue as an anatomical electromagnetic circulatory system. The microtubular array of collagen in connective tissue is a structural component that facilitates this conduction.

Hunt’s instrumentation detected differing electromagnetic currents in various body tissues. Denser tissue like bone and cartilage exhibited slower moving, lower frequencies that she associates more with direct current and magnetism. (This in comparison to “lighter” tissues like nerves and glands with higher frequencies and more alternating

currents.) Tendons and ligaments as denser tissue exhibit the predominance of the magnetic part of the field.

Dr. Hunt characterizes the magnetic part of the spectrum as that which is instrumental in tissue healing.⁴² She found that the energy at the location of an injury switches from predominantly electrical to become primarily magnetic in preparation for repair and healing. The increase of the low frequency magnetic field encourages repair cells to redifferentiate and grow. She notes the similarity of the repair role of this direct current flow to that which is described by Robert Becker. Hunt asserts that a primary breakdown in the body's healing response can occur in the electromagnetic system—in the strength and range of its impulses, as well as in its coherency. This appears to have a major influence on the condition and healing capacity of connective tissue including ligaments and tendons.

Contractile Capacity, Cellular Locomotion

Information that expands the conventional model until this point has been in the areas of neural involvement, bioelectromagnetism, and self-remodeling of the tissues. Another broadening of the perspective on the properties and functional capacity of connective tissue is provided by examining research on its contractile capacity and its cellular motility.

There is evidence for the widespread existence of the structural components and the active mechanism for contractile capacity in non-muscle cells. Cell biologists and biochemists have found a system of muscle-like proteins in nonmuscle cells, including fibroblasts. These proteins are actin, myosin, tropomyosin, and actinin. Actin is one of the body's most abundant proteins, constituting between 5% to 15% of the total protein content of cells.^{2, 41, 44, 45} Actin can self-assemble into a helical polymer filament. Myosin is a hexamer and not a single protein.

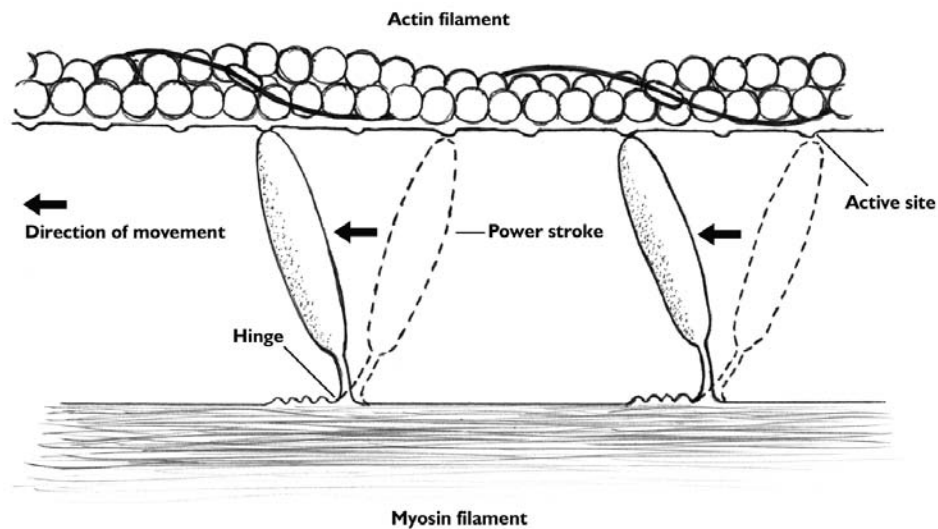


FIGURE 7
Actin-myosin motor.

The interaction between actin and myosin is basic to what is termed the *acto-myosin motor*, which is a primary contractile mechanism vital for muscle contraction. A primary component of this process is myosin filaments containing protruding heads, which are potential cross-bridges. The myosin heads fold/unfold as they attach and release by molecular bonding with various sites on the actin filament. This causes a pull that moves the actin. This contractile motor mechanism is found also in nonmuscle cells, including fibroblasts.^{41, 45, 46, 47}

Cell locomotion occurs when the cell moves its position relative to its surroundings, and is the most sophisticated of cellular movements. Studies on tissue fibroblasts and large amoebae have provided much of the evidence for the mechanism of crawling movement of cells.^{41, 44} The process includes a phase in which actin assembles into a cross-linked gel meshwork in the “front” or protrusive portion of the cell. This protruding region of the cell adheres to an anchorage beyond the present distal sites, so that the contractile machinery can pull the cell forward.

Cell locomotion thus involves two main components: 1) the actomyosin motor system to generate contractile forces, and 2) an actin gel-assembly process for protrusion of the front of the cell. The cell surface must have the capacity for attachment, de-linking, and then reattachment to a new site. Locomotion of fibroblasts is essential for wound healing. The fibroblasts move into the area and then the contraction of numbers of fibroblasts pulls the edges of the wound together.^{41, 44}

Evidence for the mechanism of cellular contractile capacity and locomotor activity of nonmuscle cells, when applied to the fibroblasts of ligaments and tendons, indicates that these structures may not be as passive as was previously thought. Tendon and ligament tissue would possess the capacity to generate motion from within. This changes the perception of them as inert, passive structures that are only moved by external forces.

The evidence would also indicate that the tissue has some capacity to reorganize itself into a different shape and to contract in ways that would facilitate the knitting together and condensing of tendons and ligaments that have been overstretched; these would have previously been considered to be permanently lax.

Possibilities for Tissue Regeneration in Tendons/Ligaments

To discover capabilities for tissue regeneration of injured tendons and ligaments, I have explored research findings for regeneration of various body structures; this provides a view into basic mechanisms of the process. There appear to be common features that are applicable to tendon and ligament healing. Extensive research in this area was done by Robert Becker in connection with his investigation of bioelectricity, but first is a look at the regeneration and repair of muscle tissue by satellite cells.

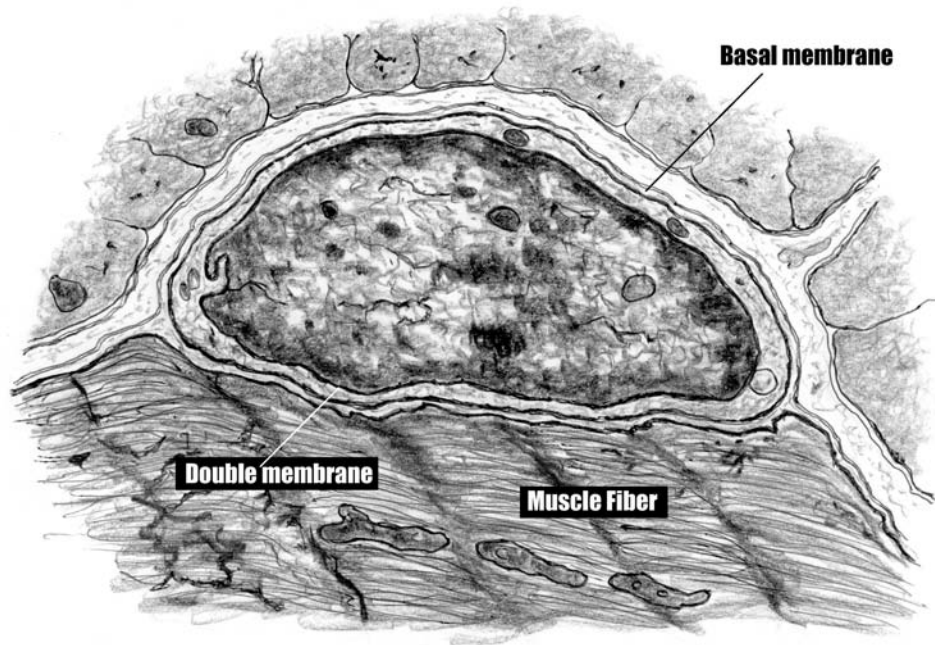


Figure 8

Satellite cell located in periphery of muscle fiber.

The satellite cell in skeletal muscles was discovered by Mauro in 1950 and is the *stem cell* (unspecialized), which, when activated, provides a new cell population for muscle regeneration in vertebrates by transforming into myoblasts.⁴⁸ They are small mononuclear cells below the basement membrane, stored in the extra-cellular matrix of the muscle fiber.^{49, 50} The Russian histology researcher Studitskii reports that “transformation of satellite cells into myoblasts is now an established fact.”⁵⁰ This is particularly distinct following trauma, and may be instrumental in normal development and self-renovation of muscle fibers.^{49, 51}

The two theories of satellite cell origin are: they are myonuclei that are pinched off from damaged muscle cells which then de-differentiate to form a population of myogenic cells, or they are an ongoing reserve of precursor cells below the basement membrane.⁵² They have the ability to synthesize DNA, divide, generate differentiating muscle cells, and give rise to new stem cells.⁵³ Under steady-state conditions stem cells are quiescent.⁵³

The result of satellite cell activation is development of daughter muscle fibers in maternal ones, and subsequent separation into new fibers. The molecular biologist and muscle researcher Richard Strohman reports that satellite cells can form new fibers or activate the thickening of existing fibers. This occurs not only after major damage, but in conditions of increased loading of the muscle where a normal repair function leads to strengthened fibers.^{48, 49} He feels that normal growth and regeneration are different degrees of response along the same continuum.

Studitskii did extensive studies on skeletal muscle transplantation and grafting and the role of satellite cells. He defines plasticity as the capability for structural change depending on change of conditions. This is a different usage of the word from the description of “plastic range” of tissue deformability described earlier in this chapter. The plastic activity of muscle tissues is manifested by structural changes of growth and differentiation; it is facilitated by a particular reactive state of the tissue that Studitskii calls the plastic state.⁵⁰ The *plastic state* appears with any condition that deprives the muscle of its working capability, including weak traumatization. Plastic activity is a restorative phase that in muscles involves the appearance of satellite cells.⁵⁰

My sense is that the concept of the plastic state and the conditions that favor it provide a very useful insight into successful strategies for connective tissue healing. There are some factors that *favor plastic activity* of muscle. These include hypervascularity (including ingrowth of blood vessels) and hyperinnervation of the tissue and surrounding areas. Activation of conditioned reflexes brought about by massage or exercise of the contralateral or adjacent muscles stimulates the plastic state.⁵⁰ Studitskii used various methods of interrupting the working activity of the muscle (including some irritants) to produce the state. Strohman finds that introducing a highly erratic stimulus activates stem cell growth.⁴⁹

Passive stretch appears to be another facilitative influence on plasticity.⁵¹ Both a high basal metabolic rate and adequate local tissue metabolism are helpful to plastic activity. Strohman reports his findings that local (hormone-like) growth factors in the tissue can activate satellite cell activity.⁴⁸ Studitskii emphasizes the importance of nerve influence (including trophic activity) for maintaining optimal metabolism of an organ or tissue and for facilitating the plastic state.⁵⁰

Dr. Robert Becker's research findings on the perineural Direct Current system of control and communication in the body were described earlier. Here is a view of his findings on regenerative growth, mostly with electromagnetism in regeneration of limbs in vertebrates, and bone fracture healing in humans.^{33,34} He defines regeneration as the formation of complex body parts involving growth from simpler into

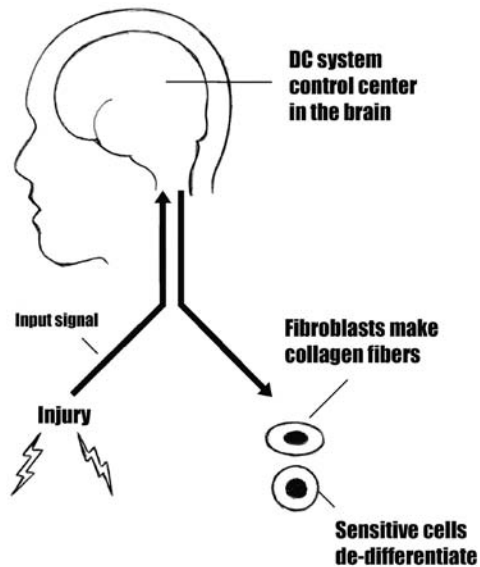


Figure 9
Direct current injury response and growth control system.

more complex cells (redifferentiation). Physiological repair is defined as cell proliferation of the same type, which heals wounds by closing the gap. This is distinct from a wound being patched over with scar tissue.

Becker identifies two basic stages of regeneration. The first stage starts with wound clean-up, which culminates in the de-differentiation of nearby cells to form a blastema. A blastema is a mass of primitive embryonic cells appearing at the site of an injury. De-differentiation is the process in which a mature, specialized cell returns to its original, embryonic, unspecialized state, and

apparently is stimulated by magnetic and DC flows after injury (“current of injury”). In the second phase, also stimulated by DC flows, the embryonic cells pile up as the blastema elongates; they then redifferentiate and take their proper place.³⁴

Invertebrates can regenerate entire limbs. Becker theorizes that mammals cannot because they seem to lack two elements necessary for the first stage of regeneration (see above). One is that there is a shortage of cells sensitive enough to de-differentiate in order to form a blastema. The second element is that mammals lack a high enough ratio of nerve to limb tissue to produce adequate strength of the electrical stimulus needed for de-differentiation. However, he devised some successful strategies for facilitating the phases of regeneration in mammals by applied electromagnetism. This was particularly notable in facilitation of fracture healing in humans.

Becker's research does show that collagen, so abundant in tendons and ligaments, is a piezo-electric generator, and also that collagen fibers can align in parallel in response to a weak DC flow. He feels that the potential repertoire of human cells is greater than is now realized and that there are possibilities for tissue regeneration that have not yet been actualized.^{33, 34}

There are some important similarities in the findings concerning the essential features of tissue regeneration, when Becker's work is compared to that of the researchers on muscle regeneration involving satellite cells. Both approaches describe a process wherein there is cellular de-differentiation into unspecialized cells (or in some cases the appearance of unspecialized stem cells from other sources), which subsequently redifferentiate into specialized cells for tissue production and maintenance.

The muscle regeneration and electromagnetic research have each found that it is vital to have adequate nerve currents in the tissue. Both have also found that regeneration is favored by a healthy, strong metabolic function in the tissue, facilitated by adequate vascular and lymphatic flows. Although tendon/ligament regeneration is not their main focus, Studitskii does mention findings that the localized fascias and tendons have a high degree of plasticity during muscle regeneration and that they repeatedly reorganize until a final structure is formed.⁵⁰

A direct focus on connective tissue healing (and its involvement with cellular differentiation from simpler cells) is found in the work of the Danish cell biologist A. Viidik. His research indicates that connective tissue wound healing involves fibroblasts that are derived from undifferentiated perivascular mesenchymal cells, some of which come from the small blood vessels supplying the tendon or ligament.²⁶ Certain conditions that encourage the plastic state are available for tendons and ligaments, and would seem to favor their healing process. Viidik states that regeneration of the original tissues in connective tissue injury is not exceptional, and cautions not to assume that healing by scar tissue formation is the only possibility.²⁶

Aside from the issue of cellular transformation, the fibroblasts themselves are active cells that can produce large amounts of collagen and ground substance. They are very active in wound healing and are capable of proliferation to repopulate a region.⁹⁹ Studitskii finds that fibroblasts play a major and essential role in the regeneration process.

An interesting perspective on the capabilities for tendon and ligament repair and regeneration is presented by the movement analyst, educator, and occupational therapist Bonnie Bainbridge Cohen. The educational/therapeutic approach she has developed, Body-Mind Centering, includes a segment of working with tendon and ligament problems; this reflects her view of these structures as being more responsive and functionally active than in the conventional model. Body-Mind Centering is empirically derived from 1) observation of sensing movement patterns and of clinical data, and 2) comparison of this with standard anatomical knowledge.⁵⁵ Regarding regeneration, Bainbridge Cohen believes that it is possible to reach a source of de-differentiated cells and activate them to effect de-differentiation.⁵⁶ She describes a structural matrix of the ligament that is comprised of small latticelike, electromagnetic hooklike connections. These “hook-ups” can realign into their innate pattern in response to therapeutic input, and effect significant tissue reorganization and healing.

She feels that overstretched, damaged tendons and ligaments can regain most or all of their proper tone, length, and thickness.⁵⁵ Their fibers can regain their proper spacing (proximity) and alignment. They can knit together, or loosen as needed. She states that conventional approaches have little idea of the actual possibilities for dramatic change of structure.⁵⁶

Bonnie Bainbridge Cohen sees the role of ligaments as providing guidance, efficiency, and clarity to movement and alignment, and setting a pattern for muscular response.^{55, 57} The ligaments provide an automatic movement control, and (as Basmajian also says) they fatigue more slowly than muscles, thus playing a vital role in weight-bearing and maintenance of posture.²¹ Her view is that one can initiate movement from the tendons or ligaments, and that activating the ligaments adds to movement clarity and range. The activation will decrease muscular effort and tension. It is important to use the ligaments in movement. Body-Mind Centering uses a manual therapy approach that is extremely specific and detailed in its treatment of tendons and ligaments. She feels that there is some contractile capacity of their tissue and that they have a contractile function.^{56, 57} Bainbridge Cohen's perspective is that the traditional view sees ligaments as too passive; they have a fuller, more active role and healing capacity.

Summary

Structure, properties, and function of tendons and ligaments are described in this chapter. We have seen the process of tendon/ligament injury and the anatomical and physiological changes in continuing dysfunction. The normal remodeling process of healthy connective tissue portrayed in this chapter is a foundation for the recovery process. Many of these elements are at work in the situation of the dancer's tendon injury in Chapter 1 and in the cases of people to be reported later.

The standard medical view of ligaments is that they are passive rubber band-like structures. They are seen as "fixed-length stays" whose neural function consists of some proprioception and involvement in a simple reflex loop system that provides protection for the joint.

Research findings demonstrate that ligaments (and tendons) are more active structures having a significant role in weight-bearing and postural control, ongoing active cellular function, responsive to electrical and magnetic activity, and containing tissue components possessing some contractile capacity. These findings show a relatively extensive neural role for these structures with involvement in the gamma motor system and substantial provision of vital afferent input to the CNS. This is essential to coordination of movement and posture. Some functional aspects of ligaments seen in these research findings are favorable factors in potential for healing from injury.

The standard medical view of the injury healing capacity is that once it has been stretched beyond the limits of the plastic range, a ligament becomes permanently lax and cannot regain its normal tone and structure. A chronic injury consisting of a significant degree of fibrous rupture of a tendon or ligament is considered to have little or no possibility of recovery without surgical treatment. Surgery itself often does not have a favorable prognosis for promoting full, lasting recovery.

Increased healing capacity for tendons/ligaments is indicated in recent research. We can be encouraged by the chapter's evidence for the factors listed below.

Factors that enhance tendon/ligament repair described in this chapter appear to comprise a *basic bodily healing response* that may function in any area. These elements are reactions to injury, promoting the healing process. While many factors also lead to recovery, the following list emerges from the research findings and theory in the preceding pages. A basic healing response for an injured structure is:

- Appearance and/or proliferation of the types of cells in the injured tissue that are necessary for its repair. I have mentioned stem cell (less specialized) activation that leads to re-differentiation into specialized cells. Another possibility is proliferation of characteristic cells (e.g., fibroblasts) in the tissue.

- Acceleration and intensification of normal tissue renovation and remodeling to restore a healthy structure (reversal of tissue degeneration and the proliferation of fibroblasts in response to injury mentioned above are examples of this mechanism in connective tissue).

- Activity of hormone-like growth factors in the inflammation stage and repair process.

- Electromagnetic activity involving an electrical current circulatory system (this may be a perineural Direct Current control system) responding with a “current of injury.” This can guide tissue remodeling and encourage the reparative function of the cells.

- Neural feedback circuits connect to the central nervous system and promote healthy autonomic and somatic neural control of metabolic and movement/postural activity for the injured area.

- The “plastic state” promotes tissue regeneration. Plasticity here is the capacity for structural change. This particular reactive state of the tissue appears in response to injury and in muscle involves activation of satellite (stem) cells. Plastic activity is facilitated by several factors including plentiful vascular and neural activity in the injury area, along with high basal metabolism and adequate local tissue metabolism.

In addition to the elements of a general healing response, there are other specific factors that can promote tendon/ligament recovery. One factor is the contractile and locomotive capacity of some of their tissue components, along with connective tissue motility (generating of forces from within).

The continuous metabolic turnover and remodeling process (self-renovation) of connective tissue are also health-enhancing influences. Collagen tissue appears to have capacity for repairing its torn microfibers and producing new ones. Plentiful neurotrophic supply of nutrients to the injury area can promote recovery.

Certain therapeutic strategies emerge from the referenced sources that would encourage the mechanisms that allow tendon and ligament

healing. These can enhance all the processes I have described as elements of healing response. For the chronically injured structure the strategies are:

- a) Increase its oxygenation
- b) Raise its metabolic rate (energy level of the tissue)
- c) Ensure adequate vascular and lymphatic flow
- d) Reduce inflammation
- e) Remove excess waste material, deposits from the tissue
- f) Promote healthy cross-linking of collagen fibers
- g) Normalize the electromagnetic climate of the body region
- h) Restore normal mechanics to the adjacent joints
- i) Establish normal, balanced patterns of afferent input from the joint and tendon/ligament
- j) Restore an adequate quantity of its ground substance (ratio of ground substance to fiber)
- k) Reestablish the normal mobility and alignment of the individual fibers and their bundles.

Chapter 5 depicts these anatomical/physiological changes occurring as the structure heals, as they may be promoted by manual therapy.

Findings of the various scientific researchers in this chapter are a basis for its view of tendons/ligaments' favorable factors and capacities for recovery. This view points to greater possibility for their healing from chronic injury than in the conventional model. Expanded healing potential is in line with research data and clinical theory of researcher-clinicians such as Becker, Bainbridge Cohen, Hunt, Korr, Curwin, and Stanish. The upcoming material on manual therapy and case study accounts will show how this chapter's capacities, favorable factors, and strategies can be utilized in a manual therapy approach to promote recovery.