MYOFASCIAL MANIPULATION
Theory and Clinical Application

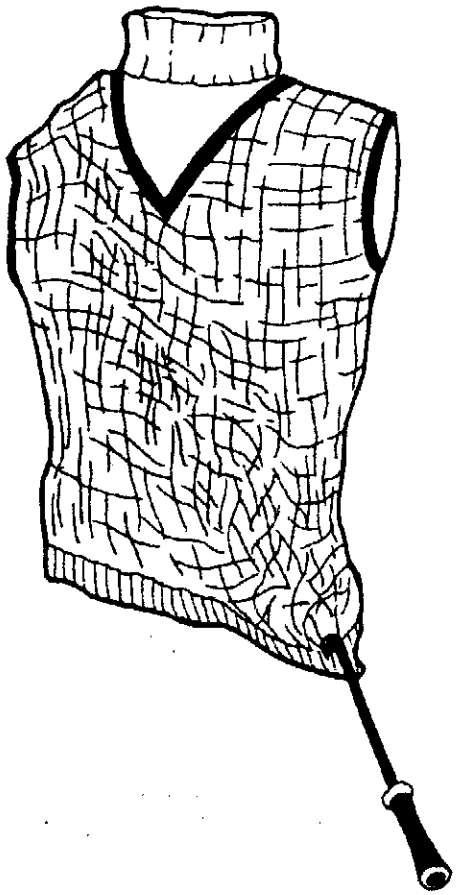

Figure 2-3 The fascial sweater concept showing that a fascial restriction in one area will strain areas away from the restriction and cause abnormal movement patterns. *Source*: Reprinted from *Rolfing: The Integration of Human Structures* (p 39) by I Rolf with permission of the Rolf Institute of Structural Integration. © 1977.

of the movements is in the moderate or midrange, with integration of cervical and lumbar traction. The oscillations and rocking techniques serve as relaxation techniques that encourage the patient gradually to relinquish control. Finally, the active movement part of the treatment serves as a neuromuscular reeducation technique similar in principle to Feldenkrais work. The idea is to alter the patient's neurophysiological set and give the patient the tools to maintain the changes.[11] The therapist is not attempting to make mechanical changes in the soft tissues, but is trying to alter the neuromuscular set to establish more normal movement patterns.

MOVEMENT APPROACHES

The movement approaches differ from the others in that the patient actively participates in therapy. Both autonomic and mechanical approaches rely on the clinician to impart the changes and movement. In the movement approaches, the clinician guides the patient through a series of movements to change aberrant patterns and retrain into more efficient movements and postures.

Alexander

F. Matthias Alexander was a Shakespearian orator at the turn of the twentieth century, who developed a consistent problem in projecting his voice. He began studying the relationship of head and neck posture in relation to voice projection, and from that developed a system of movement that can teach the entire body to become more efficient, regardless of the activity. The technique objectives are improvements in both posture and body mechanics. Many vocalists, musicians, and other performing artists use the Alexander technique to improve efficiency.

Since Alexander's recurrent laryngitis persisted despite prolonged periods of rest, he set up a system of mirrors through which he could observe himself speaking in his professional oratorical voice. He observed a tendency to pull his head back, depress his larynx, and inhale through his mouth. After repeated practice sessions, he was able to hold his head and neck in a more efficient posture, and with time, his voice projection improved and his laryngitis subsided. As time passed, Alexander noticed that the "dysfunctional" head tilting was not an isolated movement, but was coordinated with other dysfunctional patterns throughout his body such as lifting the chest, arching the back, and tensing the legs and feet.[12]

Alexander theorized that in each human being, there existed an integrating mechanism that produced more optimal coordination and functioning. Alexander wrote:

> I discovered that a certain use of the head in relation to the neck, and of the head and

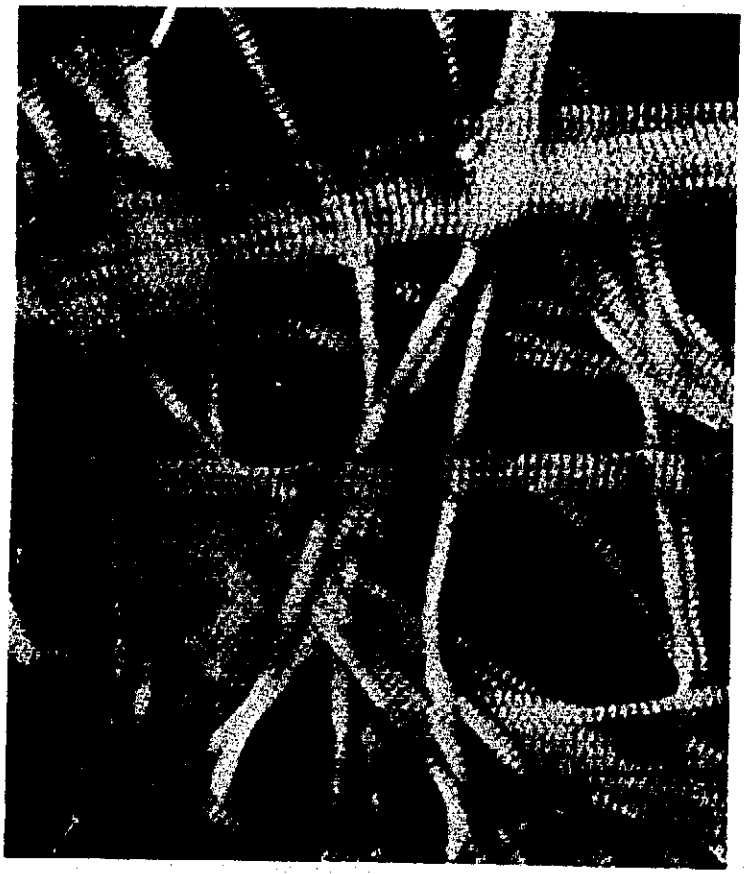

Figure 3-17 Prepared micrograph of loose irregular connective tissue, showing both multidirectionality and low density of connective tissue fibers. *Source*: Reprinted from *Bailey's Textbook of Histology* (p 117) by WM Copenhaver, RP Bunge and MB Bunge with permission of Williams & Wilkins, © 1975.

speed of contraction or relaxation, biochemistry and metabolism, and in circulation.

Mechanism of Growth in Skeletal Muscle

The total number of actual muscle fibers in a muscle is reached sometime before birth. Longitudinal growth in a muscle is accomplished in early years by an increase in the length of the individual *sarcomeres* and by addition of sarcomeres. Increases in diameter are accomplished by the addition of myofilaments in parallel arrangement. Likewise, the muscle shortens by losing sarcomeres and decreases in diameter by losing myofilaments. With prolonged disuse, the muscle fibers degenerate and the tissue is replaced with less metabolically active connective tissue. Human skeletal muscle, however, does have some limited regeneration potential. Satellite cells, which are believed to be a persisting version of the prenatal myotubes, can become activated to produce a limited amount of new muscle fibers.

Cellular and Histological Organization of Skeletal Muscle

The contractile proteins of striated muscle are actin and myosin. The actin and myosin interact in a ratchet-type manner to shorten the muscle (Figure 3-18). Actin and myosin filaments are contained in the functional contractile unit of muscle called the *sarcomere*. The transverse alignment of sarcomeres in adjacent myofilaments gives this tissue the striated appearance. The striations result from a series of bands (Z, A,

Chapter 4

Histopathology of Myofascia and Physiology of Myofascial Manipulation

HISTOPATHOLOGY OF MYOFASCIA

In the previous chapter, the normal histology and biomechanics of myofascial tissues were discussed to lay a groundwork for an understanding of histopathology and pathomechanics of these tissues. This chapter will deal with histological and biomechanical changes that occur in myofascial tissues when subjected to immobilization, trauma, and remobilization; as well as the physiological effects of massage and myofascial manipulation. With an awareness of the changes that occur in myofascial tissues under dysfunctional and therapeutic conditions the physical therapist will have realistic expectations of what can be clinically accomplished. The intuitive aspects of myofascial manipulation must always be balanced by a solid understanding of the tissues and their response to dysfunction.

RESPONSE OF MYOFASCIAL TISSUE TO IMMOBILIZATION

Connective tissue has a characteristic histological and biomechanical response to immobilization. Most of the currently available research, however, focuses on animal studies in which an area of the body is immobilized for a period of time, after which the connective tissue is histologically and biomechanically analyzed. Several factors must be considered before applying the results of these studies to the general rehabilitative population. The first is that these are animal studies; the results of which should be cautiously applied to the general human population. Second, and of greater clinical importance, many of the studies that will be discussed in this chapter deal with the response of "normal," or nontraumatized, connective tissue to immobilization, and do not necessarily address the responses of traumatized and/or scar tissue. In the general orthopedic setting, connective tissue that has been immobilized has also been traumatized. Trauma does affect the histology and biomechanics of the healing of connective tissue. Also brought into the picture is the process of scar formation, and the effects of immobilization on the developing scar tissue. All of these clinical scenarios will be addressed in detail since the response of normal connective tissues to immobilization provides a basis for understanding traumatized conditions.

Connective Tissue

Many studies of immobilization of connective tissue have been performed by Akeson, Amiel, Woo, and their various associates.[1–6] In these studies of animal connective tissues, joints (usu-

ally the knee) of laboratory animals were immobilized by internal fixation for periods of from 2 to 9 weeks. A pin was placed from the proximal one third of the femur to the distal one third of the tibia to presumably avoid traumatizing the knee joint. The animals were then sacrificed at various times of immobilization and the periarticular connective tissues were analyzed macroscopically, histochemically, and biomechanically. From a macroscopic standpoint, the authors found fibrofatty infiltrate, especially in the capsular folds and recesses. The longer the immobilization, the greater amount of infiltrate was found, along with a change in the infiltrate's appearance, which became more fibrotic, creating macroscopic adhesions in the recesses.

Histological and histochemical analyses showed several significant changes, the primary one being a significant loss in ground substance, with no significant collagen loss. The primary components of lost ground substance were the glycosaminoglycans and water. The authors reported a 30% to 40% loss in both sulfated and nonsulfated groups. Since the primary purpose of the nonsulfated group (hyaluronic acid) is to bind water, the water loss is easily explained.

As noted in the previous chapter, one of the primary purposes of the ground substance is to lubricate the area between adjacent collagen fibers. Collagen fiber lubrication is associated with the maintenance of the so-called *critical interfiber distance*. This is the distance that must be maintained between collagen fibers to allow them to glide smoothly and to prevent microadhesions between fibers. When the critical interfiber distance is not maintained, the collagen fibers approximate and eventually become cross-linked by newly synthesized collagen (Figure 4-1). Also, since collagen fibers are laid down according to the stresses (or lack of stresses) applied, collagen in immobile connective tissue is arranged haphazardly.[7] The newly synthesized collagen then binds adjacent collagen fibers, decreasing the extensibility of the tissue (Figures 4-2 and 4-3).

Several factors explain why significant amounts of ground substance are lost, yet collagen is not. First, the half-life of nontraumatized collagen is 300-500 days while the half-life of ground substance is 1.7 to

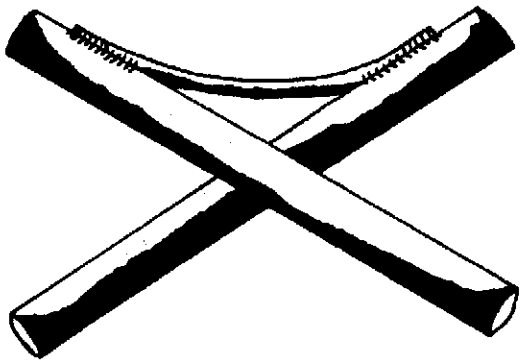

Figure 4-1 Drawing showing the laying down of newly synthesized collagen, forming cross-links onto existing collagen fibers. These cross-links are believed to be responsible for decreased extensibility in immobilized connective tissue. *Source*: Reprinted with permission from R Donatelli and H Owens-Burkhart, Effects of immobilization on the extensibility of periarticular connective tissue, *Journal of Orthopaedic and Sports Physical Therapy* (1981;3:67–72), Copyright © 1981, American Physical Therapy Association, Orthopaedic and Sports Physical Therapy Sections.

7 days.[8–10] Also, with immobilization times of less than 12 weeks, collagen synthesis occurs at the same rate as collagen degradation. After 12 weeks, however, the rate of collagen degradation exceeds the rate of synthesis, and net amounts of collagen are lost.[11]

Biomechanical analyses indicated that 10 times the torque required to move a normal joint was required to move the immobilized joints. After several repetitions, the amount of torque required to move the immobilized joint was reduced to 3 times that of a normal joint. The biomechanical implication is that fibrofatty macroadhesions and microscopic adhesions in the form of increased collagen cross-linking contributed to the decreased extensibility of the connective tissue.[1–6]

In a study performed by Evans et al,[7] experimentally immobilized rat knees were remobilized either by high-velocity manipulation, by range of motion, or both. The investigators found that, with manipulation, the macroadhesions were ruptured, and partial joint mobility was restored. If joint motion was allowed subsequent to the manipulation, functional range was regained.

Range of joint motion, along with freedom of movement, produced the same effect, although

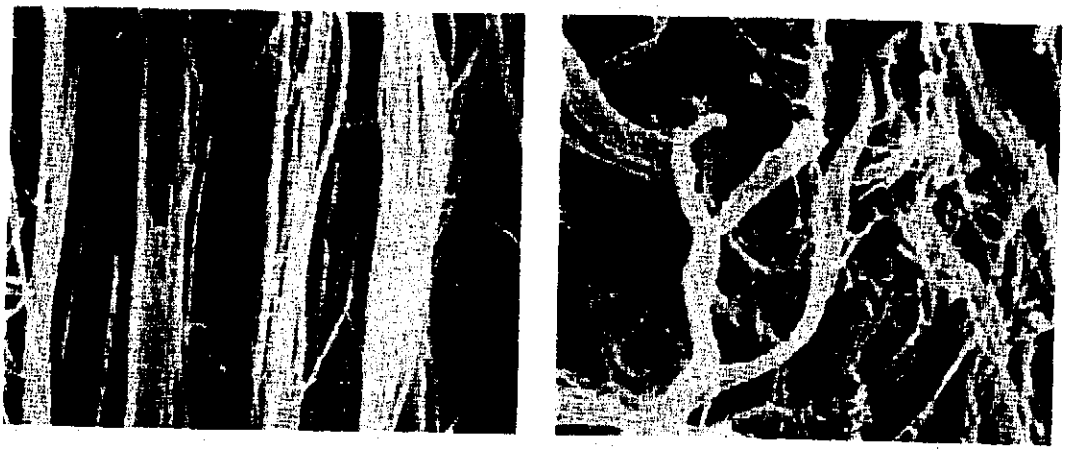

Figure 4-2 Electron micrograph of normal ligament (left) and healing scar at 2 weeks (right). *Source*: Reprinted from *Injury and Repair of the Musculoskeletal Soft Tissues* (p 112) by SL-Y Woo and JA Buckwalter with permission of the American Acadamy of Orthopaedic Surgeons, © 1987.

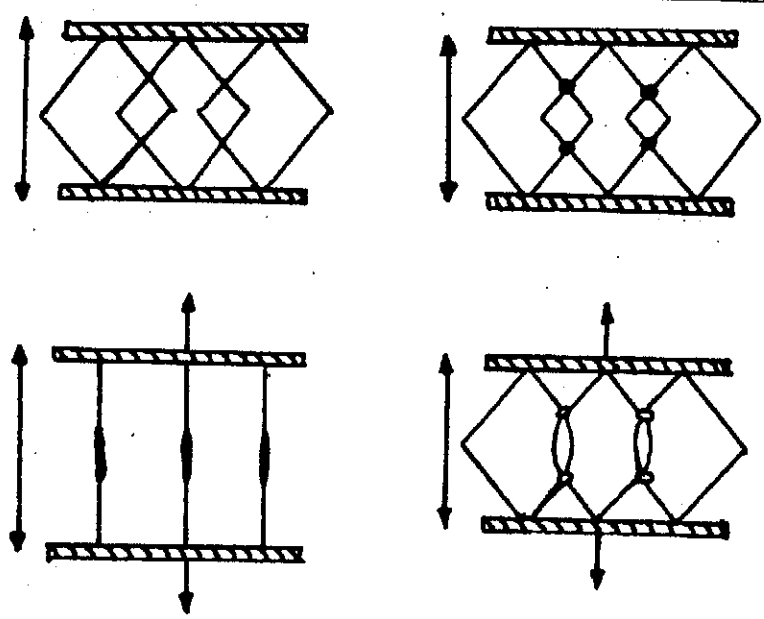

Figure 4-3 Schematic drawing representing normal connective tissue extensibility (left) and decreased connective tissue extensibility because of cross-link formation (right). *Source*: Reprinted with permission from R Donatelli and H Owens-Burkhart, Effects of immobilization on the extensibility of periarticular connective tissue, *Journal of Orthopaedic and Sports Physical Therapy* (1981;3:67–72), Copyright © 1981, American Physical Therapy Association, Orthopaedic and Sports Physical Therapy Sections.

more gradually; after 35 days the joints were histologically indistinguishable. Rat knee joints immobilized for more than 30 days, however, did not regain full functional range. Movement restores the normal histological makeup of connective tissue, but the longer the period of immobilization, the lower the potential for achieving optimal results.

In summary, immobilization of connective tissue generally results in loss of ground substance with no net collagen loss (with immobilization periods of less than 12 weeks). The

loss of ground substance also allows for significant water losses. Histologically, this results in decreased tissue extensibility due to the inability of the collagen fibers to maintain the critical interfiber distance, and the subsequent formation of microscopic collagen cross-links. At the macroscopic level, immobilization causes the formation of fibrofatty macroadhesions that become progressively more fibrotic with increased immobilization times. The studies also indicate that all periarticular connective tissues responded in the same basic fashion. Ligament and capsule surrounding fascia all had the same basic response to immobilization. Remobilization of the tissues causes a reversal of effects, provided the immobilization time has not been unreasonably long.

Traumatized vs. Nontraumatized Connective Tissue

Recently, questions have arisen about how traumatized connective tissue response to immobility differs from that of nontraumatized tissue. The previous studies have dealt with the response of nontraumatized connective tissue to immobilization. Some consider internal fixation of a limb to be a trauma-inducing form of immobilization, even though the fixation is located some distance from the tissue studied. In a study performed by Flowers and Pheasant,[12] human digits were casted for a period of several weeks and then examined. The range of motion lost during the immobilization period was regained within one treatment session of approximately 20 minutes. The implication of this study and of the previous immobilization studies is that *when connective tissues of synovial joints are immobilized in the presence of inflammatory exudates, joint contractures occur, and result from remodelling and shortening of connective tissues. When a limb is immobilized without inflammatory exudates being present, no contracture occurs, even after weeks.*[13,14] Apparently, a catalyst is needed to begin the process of contracture—the catalyst is traumatic exudate.

The clinical implications are twofold. First, patients entering physical therapy for rehabilitation following injury or surgery and subsequent immobilization will have connective tissue changes as described above. Second, a combination of two processes is occurring—scar formation and fibrosis. Scar formation occurs in areas that sustained direct insult and are in need of regeneration and repair. Fibrotic changes occur in tissues surrounding the scar area that were not directly traumatized. Traumatic exudates infiltrate these surrounding, nontraumatized areas and, acting as catalysts, create changes in the connective tissues.

Scar formation and fibrosis are two completely different histological processes, although some similarities exist. Scar formation is a localized response, with activity limited to a traumatized area, but fibrosis is a homogenous change in the "fabric" of the connective tissue. Limitation in mobility caused by scar tissue results from the lack of extensibility of the scar tissue and from the adhesions formed with adjoining healthy connective tissue. Limitation in mobility caused by fibrotic changes results from the lack of extensibility of the entire tissue.

For example, a shoulder may be frozen due to a macroscopic scar adhesion in the folds of the inferior capsule. A manipulation under anesthesia would tear the scar adhesion and restore mobility. A frozen shoulder may also be caused by a capsulitis, where the entire capsule shrinks (the analogy here is the size 5 capsule and a size 8 glenohumeral joint). The distinction is that homogenous changes in the capsule, rather than a single scar adhesion, limit motion. A manipulation under anesthesia may not be as successful in such a case, since an entire tissue is responsible for the immobility. The benefit of the increased mobility is outweighed by the potential damage to the capsule fabric and the restimulation of the fibrotic cycle.

Scar Formation Process

The scar formation process is not a cyclic but a linear process, with distinct stages and an end point. The scar process is initiated by specific trauma to an area and has four distinct stages: (1) the inflammatory stage, (2) the granulation stage, (3) the fibroplastic stage, and (4) the maturation stage.[13,14]

The inflammatory stage begins immediately after insult or trauma and lasts 24 to 48 hours. The trauma causes a disruption of tissues, blood vessels, and lymphatic vessels. Due to the damage incurred, a traumatic exudate acts to close the wound. Phagocytosis of damaged tissue is necessary to prepare the wound for healing and to prevent infection. Specialized white blood cells (macrophages and histiocytes) migrate into the area to begin clean up. At this point, any further movement in the area may create more tissue disruption and/or clot disruption.

The granulation stage begins as the area is débrided by the macrophages and histiocytes. The granulation stage is named such because of the appearance of capillary buds that macroscopically look like granules. Increased connective tissue vascularity is necessary to meet the increased metabolic demands of the tissue during healing.

The third stage of scar formation is called the fibroplastic stage because of a proliferation of fibroblasts and accelerated collagen synthesis. As the fibroblasts proliferate, new collagen is laid down in the area of the wound, replacing traumatic exudates. During this time of accelerated synthesis, the collagen is immature and held to other collagen fibers by weak, electrostatic bonds. Vascularity remains high during this stage because of the tissue's increased metabolic demands. Because the vascularity is still increased, the immature scar tissue has the characteristic pink coloring. Wound closure usually occurs during this stage and the time frame varies, depending on the normal vascularity and metabolic rate of the tissues. In tissues of high metabolic activity (muscles, skin, etc), wound closure occurs in 5 to 8 days. In tissues of lower metabolic activity (ligament and tendon), wound closure occurs in 3 to 5 weeks.[14]

The final stage of scar formation is the maturation stage. During the maturation stage, the collagen fibers are growing stronger and more resistant to outside forces. The bonding between collagen fibers is primarily covalent, requiring more energy to break. During this time, the scar tissue is responsive to manual therapy, but the progress will be somewhat slowed (Figure 4-4).

Cycle of Fibrosis and Decreasing Mobility in Connective Tissue

The fibrotic process is histologically distinct from the scar formation process. The fibrotic process in connective tissue is a "homogenous" process involving an entire tissue area or the entire tissue "fabric," and does not have clear-cut stages as does the scar tissue formation process. The fibrotic process is cyclical in nature, while the scar formation process is a linear process that has a distinct end. The fibrotic process in connective tissue can continue as long as the irritant is present.

The fibrotic process is generally initiated by the production of an irritant—possibly traumatic exudates from nearby acutely inflamed traumatized tissue or a low-grade irritation-inflammation of the tissue. The low-grade irritation may be caused by arthrokinematic dysfunction, poor posture, overuse, habit patterns, or structural or movement imbalances. A rotator cuff irritation, for example, may be caused by a poor tennis service, poor sleeping postures, occupational overuse syndromes, etc. The mechanical irritant produces a low-grade inflammation, which then starts the process. With an inflammatory response, macrophages are activated to clean and débride the area. Inflammatory exudates, along with damaged collagen and other waste products, are carried away. The increased metabolic activity in the area stimulates the body to increase the area's vascularity. With increased vascularity and débridement of damaged collagen (from microtrauma), fibroblasts are activated to replace lost collagen. Since the inflammatory process is generally painful, the joint is not being moved in proper fashion. The collagen begins to be laid down in more haphazard arrangement since adequate stress is not being placed on the tissue, and cross-linking with other preexisting collagen fibers begins. At one point, myofibroblasts appear in similar fashion as in scar process. The myofibroblasts, which contain significant amounts of actin and myosin in the cytoplasm, anchor to adjacent collagen fibers and contract, shrinking the tissue. The tissue shrinkage results in further dysfunctional movement, which, in turn, creates more mechanical

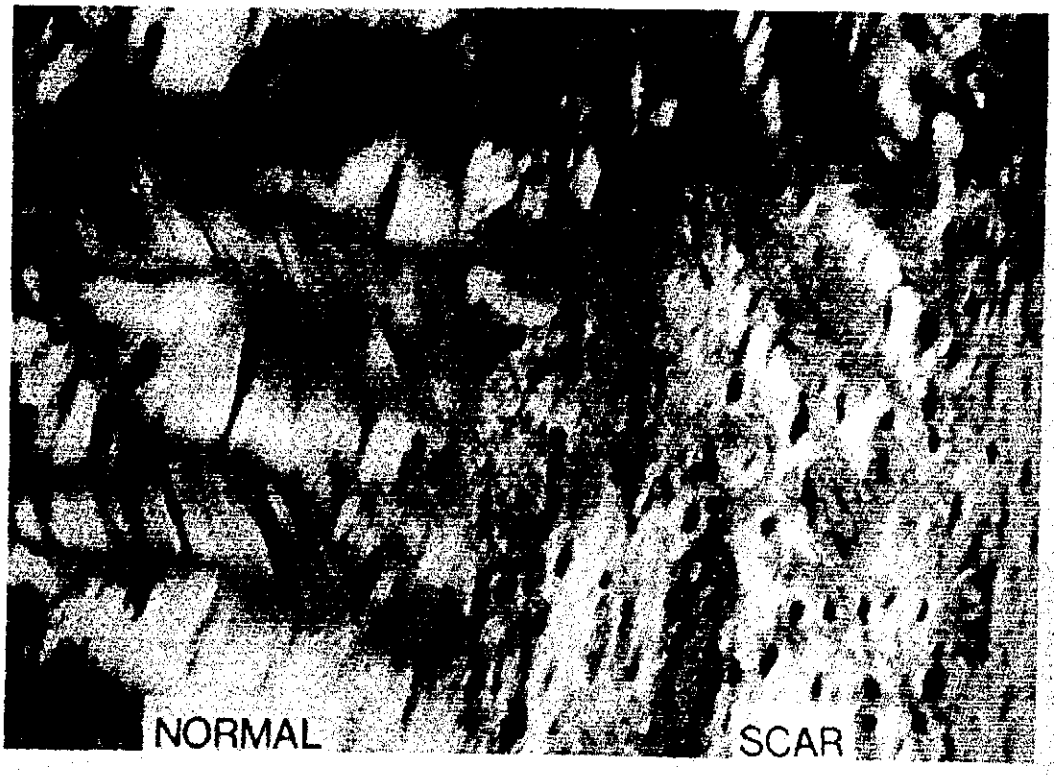

Figure 4-4 Light microscopy showing normal ligament with normal crimp pattern (left) and scarred ligament showing disorganized crimp patterns (right). *Source*: Reprinted from *Injury and Repair of the Musculoskeletal Soft Tissues* (p 111) by SL-Y Woo and JA Buckwalter with permission of the American Acadamy of Orthopaedic Surgeons. © 1987.

stresses and more chronic irritant (Figure 4-5). As long as an irritant is present, the cycle continues.

Muscle Tissue

The response of muscle tissue to immobilization is less simplistic and more multifactorial than the response of connective tissue to immobilization. Being a contractile tissue, a muscle can be passively or actively immobilized and/or the muscle may be immobilized in a shortened or lengthened position. The muscle may be innervated or denervated, or predominantly slow twitch or predominantly fast twitch. Being a highly metabolic tissue, the immobilized muscle can undergo greatly varying metabolic changes, depending on its activity level. The purpose of this section is to outline briefly the histological response of muscle tissue to immobilization and to review the various factors influencing immobilized muscle that are the most applicable to myofascial manipulation.

One of the classic works on muscle response to immobilization was performed by Tabery et al.[15] In this study, cat soleus muscles were immobilized at various lengths and for various lengths of time. The animals were immobilized by plaster cast. Some of the animals were killed and the muscles were biomechanically and histologically analyzed. Biomechanically, the passive length-tension was increased in the muscles immobilized in the shortened position, probably because of the connective tissue changes within and surrounding the muscle. Muscles immobilized in the lengthened position had no significant changes in the passive length-tension characteristics. From a histological standpoint, the muscles immobilized in the shortened position had a 40% loss of sarcomeres, with an overall decrease in fiber length. The muscles

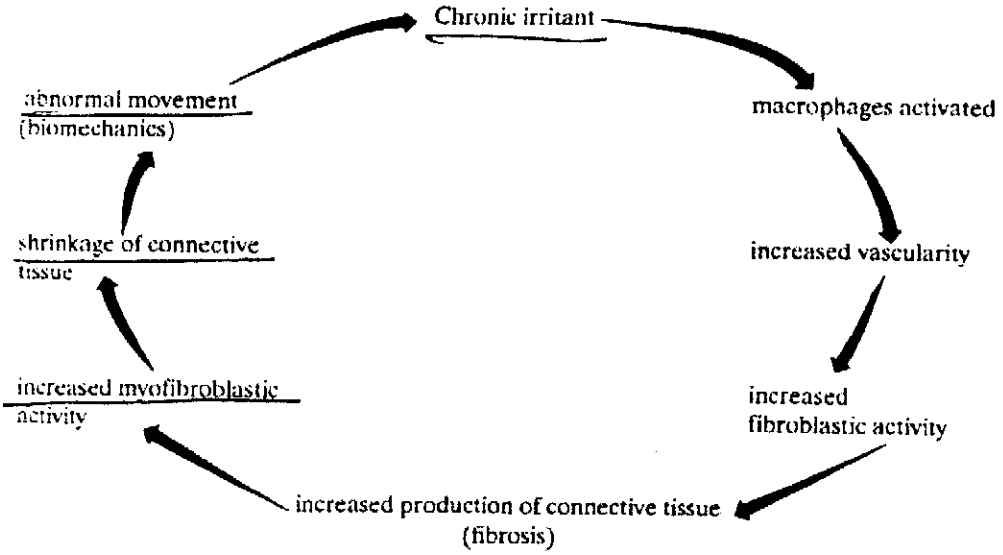

Figure 4-5 Cycle of fibrosis and decreasing mobility.

immobilized in the lengthened position exhibited a 19% increase in sarcomeres and an overall increase in fiber length. After 4 weeks of remobilization, the number of sarcomeres in the muscles returned to normal. This study illustrates the principle that muscle tissue will adapt to change in length by increasing or decreasing sarcomeres *in order to keep sarcomeres at optimal lengths.*

In a follow-up study performed by Tabery and Tardieu, muscle changes caused by prolonged *active* shortening were studied.[16] Sciatic nerves of guinea pigs were stimulated for 12 hours in either the shortened or lengthened position. The muscles stimulated in the shortened range had a 25% loss of sarcomeres after only 12 hours of contraction. Sarcomeres were completely recovered in the muscles between 48 and 72 hours. The implication of these studies is that muscles passively shortened lose sarcomeres at a much slower pace than muscles actively shortened.

The clinical implication of these findings relates to the types of immobilization that occur in the practice setting. Immobilization may occur artificially (external or internal fixation), or as a physiological mechanism. In the clinical setting, immobility may be due to trauma, past or present. A good example is the whiplash injury, in which immobilization is caused intrinsically by the cervical and upper thoracic paravertebral muscles, the scapulothoracic muscles, and the shoulder girdle muscles. In many cases, the surrounding musculature remains tonically active long after the facet or ligamentous sprain-strain has healed. The body learns a new recruitment pattern for the surrounding muscles, and this hypertonic pattern remains long after healing. The muscles are then actively "immobilized," causing some of the histological changes mentioned above. Often, the most difficult part of the therapeutic process is dealing with this hypertonicity, which is secondary to the original injury.

PHYSIOLOGY OF MYOFASCIAL MANIPULATION

Massage may be defined as systematic, therapeutic, and functional stroking and kneading of the body. Since massage and soft tissue mobilization are interchangeable in concept, the available research on massage's effects on the body need to be explored. These effects include circulatory changes, blood flow changes, capillary dilation, cutaneous temperature changes, and metabolism. Most of the studies performed