

Rotator Cuff Tendinosis

Warren Hammer, MS, DC

Dr. Warren Hammer has practiced chiropractic for 41 years. He has written numerous articles and has lectured nationally and internationally on the subject of soft tissue relating to the spine and extremities. Dr. Hammer is presently doing postgraduate lecturing for Northwestern, Cleveland, National Health University, on the Graston technique. He recently completed the second edition of a textbook entitled *Functional Soft Tissue Examination & Treatment by Manual Methods: New Perspectives*, published by Aspen in 1999.

ABSTRACT

The term *tendinitis* is currently considered a misnomer due to the fact that biopsies of the rotator cuff, Achilles tendon, elbow, and patellar tendon have failed to exhibit acute inflammatory cells (1, 2). The model that has been used to explain tendinitis has been based on the inflammatory response to open wound healing. Most injuries seen in a chiropractic setting deal with the chronic overuse injury due to multiple microtraumatic injuries rather than the macrotraumatic injury. The present acceptable term to explain the chronic overuse problem is *tendinosis*, which is a degenerative rather than an inflammatory process. This concept must cause us to re-evaluate our current therapeutic methods and may even explain why, based on our current knowledge of soft-tissue pathophysiology, particular soft-tissue techniques are effective.

ETIOLOGY

The etiology of rotator cuff tendinosis or degeneration is due to a variety of factors that create weakness especially in the supraspinatus. Weakness will occur when eccentric tensile overload occurs at a rate greater than the ability of the cuff to repair itself. Trauma to the shoulder is another cause of weakness. The weakness of the cuff muscles, especially the supraspinatus, upsets the normal balance between the deltoid muscle and the cuff muscles. Instead of the cuff muscles doing their job of maintaining the humeral head in the glenoid cavity, their weakness results in the normally strong deltoid causing the humeral head to shear upward during shoulder elevation (Fig. 1). This causes the greater tuberosity and rotator cuff to abut against the undersurface of the acromion and the coracoacromial ligament, creating what is known as a secondary intrinsic impingement (3). This is differentiated from a primary extrinsic impingement, such as a hooked acromion, which is not nearly as common as intrinsic secondary type impingements. While there is normal degeneration of the cuff with age, cuff degeneration is much more of a problem often beginning in young people who overload their cuffs especially in swimming, racquet sports, or occupations where there is overuse. Even in normal shoulders that are overloaded and fatigued, there is upward migration of the humeral head on elevation of the arm (4).

An increase of more than 8% of the total length of a tendon results in a tear (5) and vascular disruption. It is the disruption of the endothelial wall that allows blood to enter the injured area and create the humoral chemical mediators involved in a typical inflammatory process.

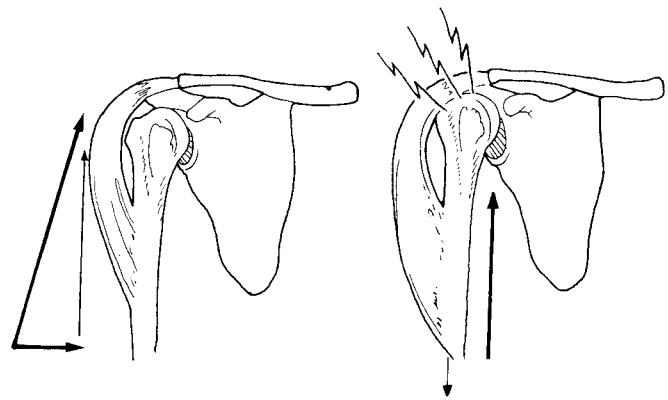


Figure 1. Independent deltoid muscle action. Dominant force pattern is vertical shear. Impingement against acromion is induced. (Reproduced, with permission, from Rowe CR. *The Shoulder*, New York: Churchill Livingstone, 1998.)

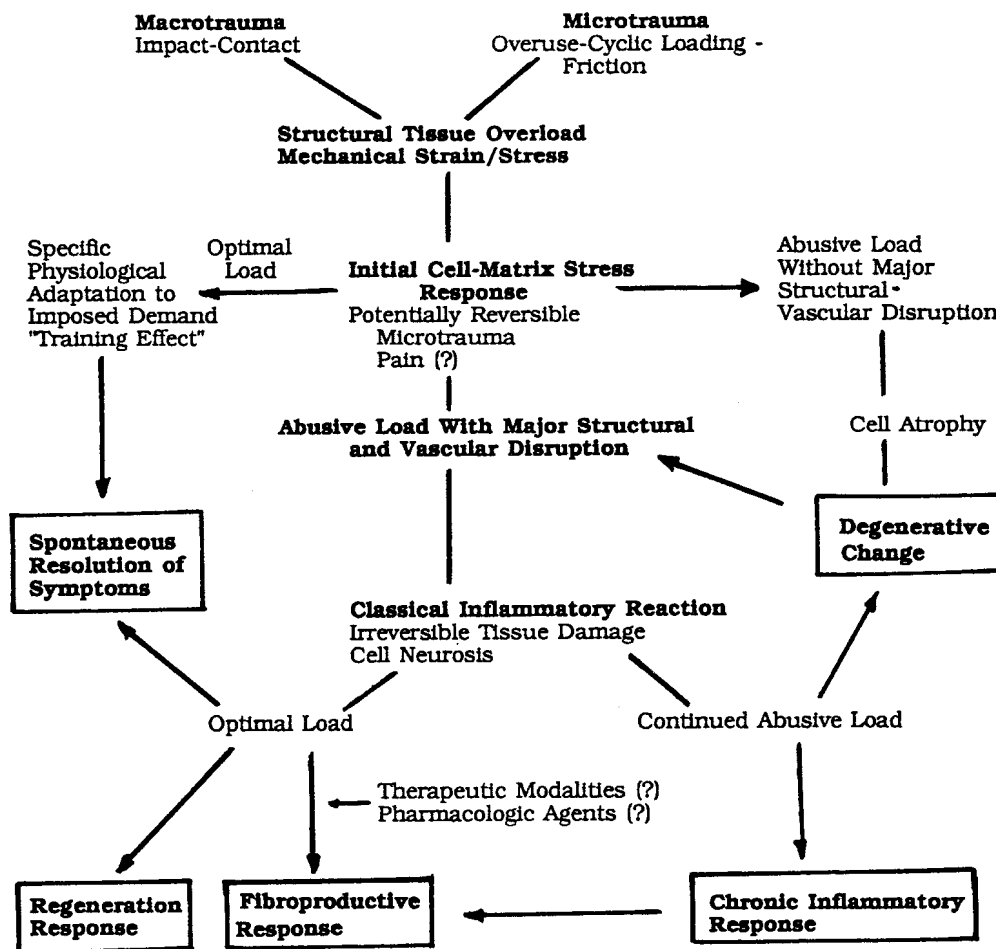


Figure 2. Schema demonstrating theoretical pathways of sports-induced inflammation. (Reproduced, with permission, from Leadbetter WB, et al. Sports-Induced Inflammation; Clinical and Basic Science Concepts. Park Ridge, IL: American Academy of Orthopaedic Surgeons, 1990.)

This represents a macrotraumatic event. Figure 2 represents the sequence of tissue change and possible end results stemming from a macrotraumatic and microtraumatic event. The microtraumatic event, unless disrupted by a major structural vascular change, can lead to the degenerative phase which is a change in tissue from a higher to lower or less functionally active form, and therefore more vulnerable to sudden dynamic overload or cyclic overloading. This leads to decreased protein synthesis, energy production, replication, storage and contractility (6), or tendinosis. There is an absence of macrophages and neutrophils. Of course, none of this pathology occurs if an "optimum load" to the cell/matrix stress is performed such as preventing abusive overload of the tissues allowing the tissue to repair itself. Leadbetter (6) defines injury as a failure of cell/matrix adaptation to load, whether sudden or accumulative secondary to cyclic overuse. It must not be assumed that all painful structures are inflamed. Connective-tissue pain can be perceived by the patient

as the result of nociception and a noxious chemical environment (pH level, the level of lactic acid, and the level of prostaglandins) (6).

HISTOPATHOLOGY

In tendinosis, there is hypertrophy and hyperplasia of fibroblasts, increased disorganized collagen production, and vascular hyperplasia. Nirschl refers to this as angiofibroblastic degeneration (3). The initial response in tendinosis due to a traumatic injury or repetitive overuse is fibroblastic hyperplasia in order to produce collagen locally. In tendinosis, the abnormal collagen production is not remodeled into normal tendon. Some of the fibroblasts return to their mesenchymal state and turn into cartilage cells, bone-forming cells, and vascular endothelium. Many fibroblasts are attracted from extrinsic sites. All of this represents the intrinsic capacity of tendons to heal themselves (7). The



Figure 3. Use of a Graston instrument on the supraspinatus insertion.

authors (7) feel that it is the lack of an effective vascular system that leads to the failure of the healing cycle in tendinosis.

EFFECTS OF MANUAL LOADING ON SOFT-TISSUE CELLS

In 1978, Curtis and Seehar (8) showed that chick embryo fibroblasts in dense culture showed increased mitosis in response to tensional forces. Frank and Hart (9) state that cells are now considered the true transducers of load and that cells in muscle, tendon, ligament, skin, and cartilage generally respond to “windows” of increased loading by increasing matrix synthesis, increasing metabolic activity, increasing their replication rates, and modifying their production of matrix components. Fibroblast proliferation and activation are necessary for tendon healing. “Activated fibroblasts are responsible for the further production of cellular mediators of healing and proteinaceous synthesis of collagen fibers” (10). Davidson et al. (11) produced a remarkable study on the effect of soft-tissue mobilization on the rat tendon. By way of light and electron microscopy, the effects of augmented soft-tissue mobilization (Graston technique) on injured rat Achilles tendon, which they referred to as tendinitis, caused an absolute increase in fibroblast proliferation. Gehlsen et al. (10) proved that the proliferative response of fibroblast proliferation was directly dependent upon the magnitude of the applied instrument. Maniotis et al. (12) proved that mechanical forces acting through integrin receptors on the surface of bovine capillary endothelial cells were transferred by cytoskeletal

components and caused realignment of nuclear structures. Mechanical stimuli have been shown to alter many cellular functions including ion transport (13), release of second messengers (14), protein synthesis (15), secretion (16), and gene expression (17).

GRASTON METHOD, FRICTION MASSAGE FOR TENDINOSIS

Kraushaar and Nirschl (7) feel that “the presence of red blood cells inside the abnormal vessels found in tendinosis suggests that vascular hyperplasia may lead to communication with an extrinsic healing response, provided that the immune system receives signals of a need for the healing process.” They mention that controlled exercise can deliver cyclical tensile loads to stimulate the remodeling of collagen which has failed in tendinosis. They state that a “fibroblast-driven process normally would be expected to integrate old and new collagen in order to contribute to the final stability of the matrix.” Graston technique is an instrument-assisted soft-tissue mobilization, which enables clinicians to effectively break down fascial restrictions by providing a controlled microtrauma to the affected soft-tissue structures (see Figs. 3–6). The instrument greatly magnifies the palpatory skill of the practitioner to find and free soft-tissue restrictions that often are unable to be palpated with the human hand. They provide the added

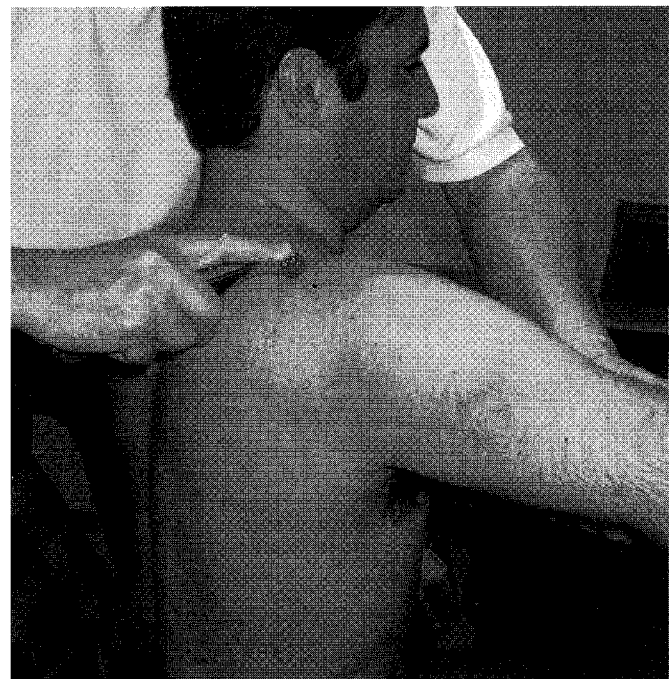


Figure 4. Use of a Graston instrument on the musculotendinous portion of the supraspinatus muscle.

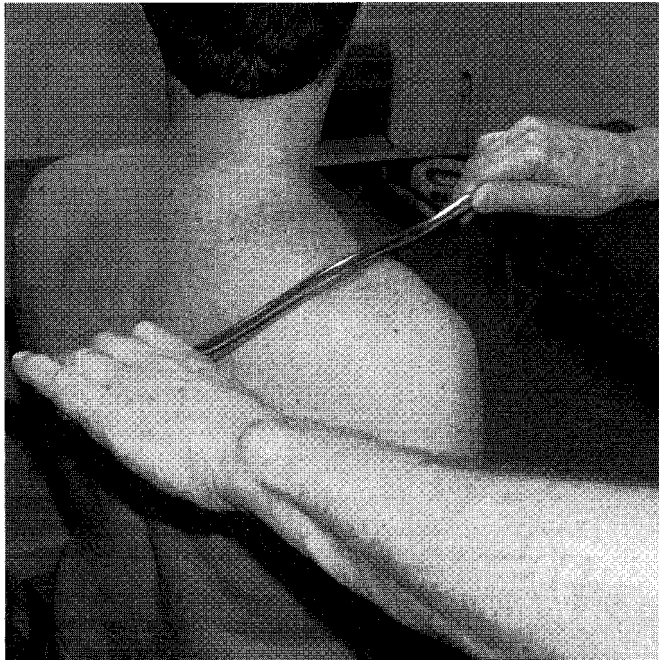


Figure 5. Use of a Graston instrument on the belly of the supraspinatus muscle.

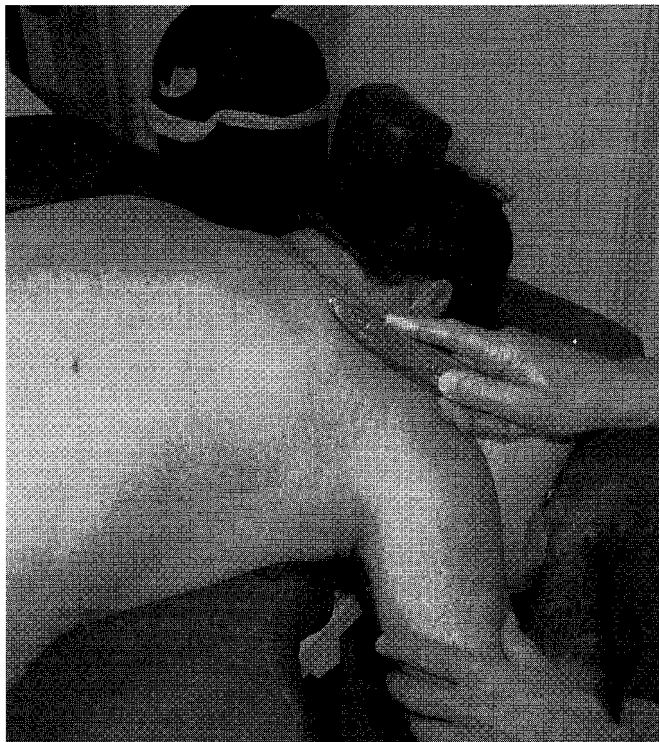


Figure 6. Use of a Graston instrument for the infraspinatus belly.

depth and magnitude necessary to produce fibroblastic proliferation and stimulate a local inflammatory response which can lead to remodeling and repair of the tendinosis lesion.

REFERENCES

1. Uthoff HK, Sano H. Pathology of failure of the rotator cuff tendon. *Orthop Clin North Am* 1997;28(1):31-41.
2. Astrom M, Rausing A. Chronic Achilles tendinopathy: a survey of surgical and histopathologic findings. *Clin Orthop* 1995;316:151-164.
3. Budoff JE, Nirschl RP, Guidi EJ. Debridement of partial-thickness tears of the rotator cuff without acromioplasty. *J Bone Joint Surg* 1998;80-A(5):733-748.
4. Burkhead WZ Jr, Burkhart SS, Gerber C, et al. Symposium: the rotator cuff: debridement versus repair — part, II. *Contemp Orthop* 1995:313-326.
5. Jozsa LG, Kannus P. Overuse injuries of tendons. In: *Human Tendons: Anatomy, Physiology, and Pathology*. Champaign, IL: Human Kinetics, 1977, pp. 164-253.
6. Leadbetter WB. Cell-matrix response in tendon injury. *Clin Sports Med* 1992;11:533-578.
7. Kraushaar BS, Nirschl RP. Current concepts review — tendinosis of the elbow (tennis elbow). *J Bone Joint Surg* 1999;81-A(2):259-278.
8. Curtis ASG, Seehar GM. The control of cell division by tension or diffusion. *Nature* 1978;274:52-53.
9. Frank CB, Hart DA. Cellular response to loading. In: Leadbetter WB, Buckwalter JA, Gordon SL, eds. *Sports-Induced Inflammation: Clinical and Basic Science Concepts*. Park Ridge, IL: American Academy of Orthopaedic Surgeons, 1990.
10. Gehlsen GM, Ganion LR, Helfst R. Effects of pressure variations on tendon healing. *Research Binder*. Muncie, IN: Performance Dynamics, 1998.
11. Davidson CJ, Ganion LR, Gehlsen GM, et al. Rat tendon morphologic and functional changes resulting from soft tissue mobilization. *Med Sci Sports Exerc* 1997;29(3):313-319.
12. Maniotis A, Chen C, Ingber D. Demonstration of mechanical connections between integrins, cytoskeletal filaments, and nucleoplasm that stabilize nuclear structure. *Proc Natl Acad Sci USA* 1997;94:849-854.
13. Schwartz MA, Lechen C, Ingber D. Fibronectin activates the Na/H antiporter by inducing clustering and immobilization of its receptor, independent of cell shape. *Proc Natl Acad Sci USA* 1991.
14. Letsou GV, Rosales O, Maitz S, et al. Stimulation of adenylate cyclase activity in cultured endothelial cells subjected to cyclic stretch. *J Cardiovasc Surg* 1990;31:634-639.
15. Thie M, Schlumberger W, Rautenberg J, Robeneck H. Mechanical confinement inhibits collagen synthesis in gel-cultured fibroblasts. *Eur J Cell Biol* 1989;48:294-301.
16. Wirtz H, Dobbs LG. Calcium mobilization and exocytosis after one mechanical stretch of lung epithelial cells. *Science* 1990;250:1266-1269.
17. Komuro I, Kathoh Y, Kaida T, et al. Mechanical loading stimulates cell hypertrophy and specific gene expression in cultured rat cardiac myocytes. *J Biol Chem* 1991;266:1268-1275.