

# Thermal Modification of Connective Tissues: Basic Science Considerations and Clinical Implications

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## Abstract

*Thermal modification (shrinkage) of capsular connective tissue has gained increasing popularity as an adjunctive or even a primary procedure in the arthroscopic treatment of shoulder instability. Although the physical effects of heat on collagenous tissues are well known, the long-term biologic fate of these shrunken tissues is still a matter of debate. The temperatures required to alter the molecular bonding of collagen and thus cause tissue shrinkage (65°C to 70°C) are also known to destroy cellular viability. Therefore, thermally modified tissues are devitalized and must undergo a biologic remodeling process. During this remodeling, the mechanical properties of the treated tissues are altered (decreased stiffness) and can be at risk for elongation if the postoperative rehabilitation regimen is too aggressive. Although anecdotal reports suggest that thermal capsular shrinkage does have a beneficial effect, the exact mechanism responsible for this clinical improvement has yet to be fully defined. The reported improvement could be due to the maintenance of initial capsular shrinkage, secondary fibroplasia and resultant thickening of the joint capsule, a loss of afferent sensory stimulation due to the destruction of sensory receptors, or a combination of all three. The clinical role for thermal modification of connective tissues has not yet been defined, but it appears that it may prove most useful as a stimulant for inducing a biologic repair response.*

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The application of heat as a therapeutic modality has evolved from the ancient use of fire to sterilize wounds and control bleeding to the modern-day use of hyperthermia to treat various forms of cancer. This evolution arose from a better understanding of the tissue biology associated with the application of thermal energy and an improved ability to control the amount of heat delivered, as well as the precision of its application. Although the use of boiling oils, heated metals, and magnified sunlight in medicine has been replaced by modern electrocautery and laser energy, the major therapeutic uses of these sources of

heat in surgery remain essentially the same: hemostasis and tissue ablation.

Recently, the use of thermal energy has been proposed as a means of shrinking redundant or lax connective tissues through the well-established mechanism of collagen denaturation.<sup>1-18</sup> Although numerous *in vitro*<sup>4,7,8,10-12,15,16,19</sup> and *in vivo*<sup>6,9,17,20,21</sup> experimental studies have investigated the biology and biomechanics of thermally modified tissues, there is still no unanimity of opinion regarding the optimal therapeutic algorithm (e.g., the degree of tissue shrinkage required and the optimal postoper-

ative regimen), the ultimate mechanism of the reported clinical improvements, or the long-term fate of the thermally modified tissues. There is, however, agreement on the basic science of the thermal modification of connective tissues, as well as on some of the potential clinical implications associated with its use.

## Mechanism of Thermal Shrinkage of Collagen

Ligaments and joint capsule are composed primarily of type I collagen. The type I collagen molecule is made up of three polypeptide chains, which are stabilized in a triple-helix arrangement by intramolecular cross-links. These molecules are, in turn, aggregated into a parallel pattern to form collagen

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fibrils. This fibrillar arrangement is maintained by intermolecular cross-links and provides the tissue with its tensile properties. The intramolecular cross-links are reducible covalent aldehyde bonds, which are progressively replaced by irreducible multivalent cross-links as the tissue ages.

When collagen is heated, the heat-labile intramolecular cross-links are broken, and the protein undergoes a transition from a highly organized crystalline structure to a random, gel-like state (denaturation) (Fig. 1).<sup>22</sup> Collagen shrinkage occurs through the cumulative effect of the “unwinding” of the triple helix, due to the destruction of the heat-labile intramolecular cross-links, and the residual tension of the heat-stable intermolecular cross-links (Fig. 2).<sup>22,23</sup>

Collagen denaturation typically occurs at approximately 65°C. The precise heat-induced behavior of connective tissues (and the extent of tissue shrinkage) are dependent on several factors, including the maximum temperature reached and exposure time<sup>23</sup> and the mechanical stress applied to the tissue during heating.<sup>19</sup> The thermal properties of

a tissue can also vary with species, age, the pH and electrolyte concentration of the surrounding environment, the concentration and orientation of the collagen fibers, and the hydration level of the tissue.<sup>24,25</sup>

Increased collagen content, directionality of fiber orientation, and increased tensile load have been shown to increase the temperature necessary for shrinkage of connective tissue.<sup>24</sup> Aging has also been shown to increase this temperature, probably due to the age-related increased ratio of heat-stable to heat-labile cross-links.<sup>24,25</sup> In addition, increased pH of the surrounding medium and dehydration of the tissues are known to increase the shrinkage temperature.

On the macroscopic level, the effect of heat on dense, regularly oriented connective tissues (e.g., ligaments and tendons) may be summarized as shrinkage along an axis parallel to the dominant direction of fiber orientation accompanied by swelling on the transverse axis. In dense, irregularly oriented connective tissues (e.g., joint capsule), shrinkage still occurs along the long axis of the collagen fibers. However, because the fiber orienta-

tion is multidirectional in these tissues, the extent and principal axis of shrinkage are less predictable.

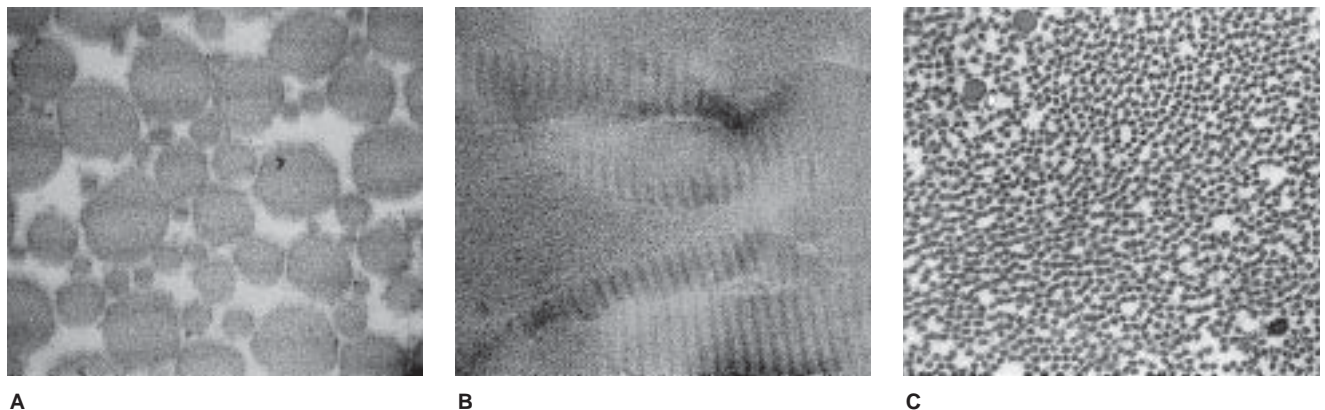
## Experimental Studies

Since the initial clinical application of thermal shrinkage in the shoulder joint, there have been numerous *in vitro* and *in vivo* studies that characterize the biomechanical and biologic effect of thermal modification on tendons, ligaments, and the joint capsule.

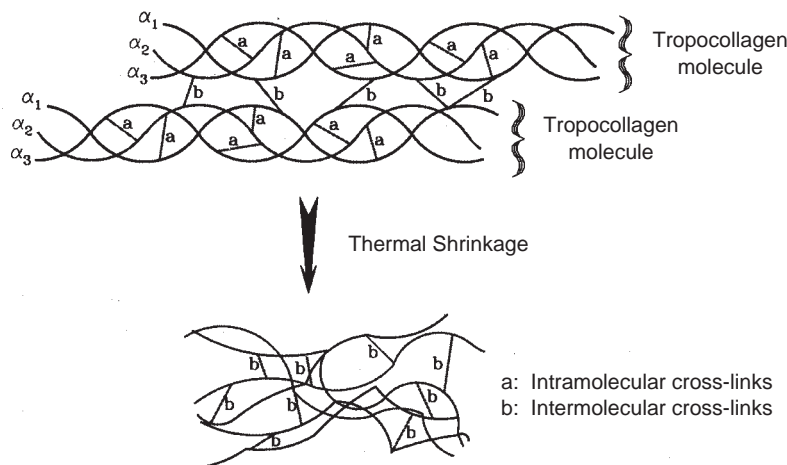
### In Vitro Studies

Examination and quantification of the thermal response characteristics of collagenous tissues are difficult due to the diversity of species and the compositional variance among tissues. In the past four decades, researchers have performed various experiments with tendon, ligament, skin, joint capsule, and corneal tissue specimens harvested from different species (cow, dog, rat, rabbit, sheep, and human).<sup>7,8,10,21</sup>

It has been established that when collagenous tissues are exposed to high temperatures (in the range of 65°C to 85°C for most tissue types



**Figure 1** A, Electron-photomicrograph of a cross section of a normal rabbit patellar tendon. Note the normal bimodal pattern of large- and small-diameter collagen fibers and the longitudinal orientation of all fibers (original magnification  $\times 34,000$ ). B, Cross section of rabbit patellar tendon immediately after thermal shrinkage. Note the random orientation of the collagen fibers (original magnification  $\times 34,000$ ). C, Cross section of a rabbit patellar tendon 8 weeks after thermal modification shows remodeling with small-diameter collagen fibers, indicative of scar tissue (original magnification  $\times 34,000$ ).



**Figure 2** Molecular mechanism of collagen shrinkage. The intramolecular cross-links within the tropocollagen molecules are denatured, but the intermolecular cross-links between the tropocollagen molecules are maintained.

and species), they undergo rapid denaturation.<sup>22</sup> The resultant effect of the microscopic phenomenon of collagen denaturation is tissue shrinkage.<sup>23</sup> At a constant temperature, shrinkage increases with increased exposure until it reaches a plateau value beyond which no additional shrinkage is observed. This ultimate value of shrinkage changes with the treatment temperature<sup>23</sup> and the mechanical load applied to the tissue during heating.<sup>19</sup> Heating to excessive temperatures for extended periods of time results in hyalinization, extensive thermal damage, and necrosis of the tissue.<sup>26</sup>

Hydrothermal heating experiments performed on specimens taken from different regions of cadaver glenohumeral joint capsules point to a correlation between the maximum temperature reached and the amount of tissue shrinkage.<sup>8</sup> Exposure to heating at 65°C for 10 minutes results in 10% shrinkage, whereas 60% shrinkage occurs after only 1.5 minutes at 80°C. These results support the axiom that the higher the maximum temperature reached during heating, the greater

the shrinkage. It is also evident from the same data that the time required to reach the same amount of shrinkage decreases with increasing temperature. A similar experiment with the use of radio-frequency heating of specimens harvested from sheep glenohumeral joint capsules demonstrated less than 4% shrinkage at temperatures below 65°C, but reached a peak value of 14% shrinkage at 80°C.<sup>11</sup>

Discrepancies among these results may have resulted from the interspecies differences in tissue composition and the possible nonthermal effects of the two heating modalities.<sup>3</sup> The variability of the amount of shrinkage of similar tissues from different species under identical heating conditions is well established in the literature.<sup>19,21</sup> It has also been shown that these variations correlate well with the differences in the physiologic body-core temperatures of the species. Another factor is that laser heating causes local dehydration of the tissues due to evaporation of fluids in the target volume, whereas this is not a problem with hydrothermal heating. It has also been postulated that laser

energy enhances healing by stimulating collagen synthesis; however, as yet no study has compared the nonthermal effects of the various healing modalities.

There are other differences among heating modalities. In hydrothermal heating, the tissue sample is immersed in a constant-temperature bath, and the tissue rapidly comes into thermal equilibrium with its surroundings. Therefore, a homogeneous temperature distribution can be obtained within the tissue. In laser or radio-frequency heating, the temperature distribution created within the tissue is a function of the probe-sweep velocity, the distance between the target tissue and the probe, and the heat-penetration depth. The combination of these factors may easily cause nonhomogeneous heating. Differences between the thermal histories of regions within the same tissue volume may cause significant changes in ultrastructure and overall behavior, resulting in nonuniform shrinkage.

Researchers realize that hydrothermal and laser heating cause differences in the resultant collagenous tissue ultrastructure,<sup>8</sup> but these variations have not been specifically attributed to either nonthermal effects or differences between applications. In addition, after heating, if the collagenous tissue is cooled to room temperature, some collagen molecules regain their triple-helix structure (renaturation). This may be accompanied by relaxation of 5% to 10% of the total amount of shrinkage obtained.<sup>19</sup>

The effect of mechanical stress on the shrinkage and postheating relaxation characteristics of bovine chordae tendineae has been investigated. In one study,<sup>19</sup> a fivefold increase in the applied stress caused about a 15% decrease in the final plateau value of shrinkage and delayed the renaturation process. The authors suggested that there is a time-temperature-load equivalency in the heat-induced



shrinkage of collagenous tissues. That is, these variables are to an extent interchangeable (they have similar effects on gross tissue behavior), and a change in any of them affects the shrinkage process. This hypothesis may have very important clinical applications, in that the same amount of tissue shrinkage may be obtained at a lower temperature, minimizing the risk of excessive loss of stiffness and thermal damage to the surrounding tissues.

Research performed on cadaver glenohumeral capsular tissues suggests that the stiffness of heated collagenous tissue decreases while viscoelastic properties remain unaltered.<sup>4,8</sup> Stiffness values down to 90% of normal have been measured after aggressive laser heating.<sup>4,8</sup> Similar to the shrinkage response, the decrease in stiffness is also dose-related.<sup>16</sup> The higher the heating (and thus the greater the shrinkage), the greater the loss of stiffness. It has been suggested that the loss of stiffness is of concern because it may make the tissues more vulnerable to postoperative mechanical loading.<sup>5,21</sup>

#### **In Vivo Studies**

There have been only a limited number of studies of the in vivo effects of thermal modification on connective tissue biology and biomechanics.<sup>6,8,17,20,21</sup> However, most researchers agree that after thermal modification and the accompanying local cell death, modified tissues undergo a repair response characterized by fibroplasia, neovascularization, and fibrovascular scar formation. In studies on sheep<sup>8</sup> and rabbits,<sup>6</sup> tissue has been shown to begin to regenerate only 7 days after treatment, as evidenced by hyperplasia and increased fibroblastic activity. Vascularization of the scar region and migration of more fibroblasts to the treated site accompany the long-term healing response of the tissue. After 1 month, newly

synthesized collagen molecules and small-diameter fibrils are present in the scar area.<sup>6,8</sup>

Subphysiologic loads are known to accelerate healing. However, it has been observed that when thermally modified tissue is subjected to physiologic loads in the immediately postoperative period, it can elongate to its preoperative length within 4 weeks.<sup>21</sup> Although the initial shrinkage values obtained in rabbit patellar tendons with laser treatment in both in vivo and in vitro experiments were the same, the in vivo study showed relaxation back to the original length by 4 weeks and beyond the original length by 8 weeks.<sup>21</sup>

A recent study has suggested that it may take as long as 12 weeks for thermally modified tissues to regain normal strength.<sup>20</sup> However, another study has shown that during that same period of time the tissue has an increased susceptibility to creep.<sup>17</sup> There is no unanimity of opinion as to the time at which thermally modified tissues regain their normal material and biologic properties. Anecdotal reports have described a total absence of capsular tissues at surgery in patients who had previously failed laser-assisted capsular shrinkage for shoulder stabilization. However, the mechanisms potentially responsible for this loss of tissue (e.g., overzealous shrinkage, extensive heat) have yet to be identified. These findings point to the need for further research on the response of thermally modified tissues in the postoperative period and the effects of various rehabilitation regimens (e.g., immobilization).

#### **Delivery of Thermal Energy**

Clinically, two types of energy systems have been used to thermally modify connective tissues: laser energy and radio-frequency energy.<sup>11,27</sup>

#### **Laser Energy**

The laser is an electro-optical device capable of efficiently transmitting energy in the form of an intense beam of light. The radiant energy of the laser beam can be transformed into heat energy through its interaction with tissues. This interaction is dependent on the wavelength of the laser and the properties of the tissue. Laser light is absorbed by water, and, as most biologic tissues are composed mainly of water, the tissues are heated.

In addition to the nature of the interaction with the tissue, the extent of the thermal effect of a laser is also controlled by the power density, the spot size, and the duration of application. The total power output of a laser is measured in watts, and the power density (measured in watts per square centimeter) is directly correlated with the thermal effect produced in the tissue. The spot size (the tissue area on which the laser light is concentrated) is an important factor in determining power density. Power density can be increased by increasing power output or by decreasing spot size (i.e., power density varies as the inverse of the area). The time of laser application can also affect the amount of energy delivered to the tissue and the resultant thermal effect. Pulsing the laser beam (rapidly alternating the beam off and on) creates a cooling effect between pulses in a fluid medium and can limit heat transfer to adjacent tissues. This helps limit the depth of penetration and tissue damage.

The holmium-yttrium-aluminum-garnet (Ho:YAG) laser was the first system used clinically for the thermal modification of connective tissues. No one therapeutic algorithm has yet been rigorously tested. Because of the noncontact nature of the Ho:YAG laser probe, there is no specific mechanism for temperature feedback; temperature control and ultimate thermal effect are de-

terminated by the surgeon through the control of spot size and the duration of laser application to a specific area. It has been suggested that the laser be set to a low power setting (10 to 15 W) and the beam defocused (by holding the tip of the probe 2 mm from the surface of the tissue). Once visible tissue shrinkage occurs, the laser beam is advanced to another area. However, it should be remembered that simply decreasing the spot size (focusing the laser beam by bringing the tip of the probe closer to the tissue) and/or keeping the beam focused too long on the same spot can result in tissue ablation.

### Radio-frequency Energy

Radio-frequency energy is a form of electromagnetic energy. When applied to tissues, rapidly oscillating electromagnetic fields cause movement of charged particles within the tissue, and the resultant molecular motion generates heat.<sup>3</sup> Electromagnetic energy can be applied between two points on the tip of a probe (bipolar) or between a single electrode tip and a grounding plate (monopolar). In monopolar devices, the molecular friction created within the tissues adjacent to the probe produces heat. Thus, the actual source of heat is the frictional resistance of the tissues rather than the probe itself. With bipolar probes, the electromagnetic energy follows a much shorter path through a conductive irrigating solution (electrolytes) or through the tissues between the tips of the probe. Less current is required with a bipolar device than with a monopolar device to achieve the same effect, because the current passes through a much smaller volume of tissue. In addition, the depth of tissue damage relative to the probe tip may be less with a bipolar device.

Similar to laser energy, the thermal effect of radio-frequency energy in tissues is determined by the level of energy (related power and im-

pedance), duration of treatment, and nature of the tissues. In addition, the electrode type (monopolar or bipolar), size, and shape all have an effect.

Some radio-frequency devices are capable of monitoring and/or controlling the temperature at the tip of the probe. Although this is thought to provide the highest margin of safety and efficacy, no data (clinical or experimental) have been published to support this claim. Indeed, as with the laser, the extent of thermal modification with radio-frequency devices is operator-dependent and relies on the visual observation of the tissue response. Because the tissue response is both time- and heat-dependent, the longer a probe (laser or radio-frequency) is left in one place, the greater the resultant tissue damage. In addition, the biologic variables of age and the thickness, hydration status, and quality of the tissue all affect the thermal response. Thus, application of a uniform amount of thermal modification with either class of devices appears, at best, subjective.

Although radio-frequency devices and lasers differ fundamentally in the way they generate heat within a tissue, both classes of devices are capable of producing temperatures within the critical temperature range (65°C to 75°C) for collagen denaturation and subsequent tissue shrinkage. Both types of devices have advantages and disadvantages (e.g., price, temperature monitoring). It must also be remembered that when it comes to cell viability and tissue response, heat is heat. Once critical temperatures are reached, cells will die, (45°C)<sup>2</sup> and collagen will become denatured (65°C)<sup>22,23</sup> no matter what the source of thermal energy. Therefore, claims of a better or different type of heat have little bearing on the biologic response of the tissue. Indeed, histologic, ultrastructural, and biomaterial alterations induced

by laser and radio-frequency energy have been shown to be similar.<sup>3</sup>

### Clinical Implications

Although the effect of heat energy on collagen molecules has been well established, the reason for the reported efficacy of this mode of therapy in the treatment of lax or redundant collagenous tissues is less clear. It has not yet been established what is really responsible for the clinical improvement after thermal capsular shrinkage—whether it is the initial connective tissue shrinkage, which appears to tighten and thus stabilize the joint; the fibroplasia, capsular thickening, and secondary cicatrix formation that occur in response to the traumatic insult; the loss of afferent sensory stimulation due to destruction of sensory receptors; or perhaps a combination of those three possible explanations.<sup>1,2,13</sup>

Although this fundamental question remains unanswered, certain clinical implications are clear. The initial degree of tissue shrinkage (and ultimate tissue damage) is directly related to the amount and rate of thermal energy applied. It has also been shown that the amount of tissue shrinkage is directly related to the loss of tissue stiffness.<sup>4,16,21</sup> Thus, the surgeon is faced with the dilemma of trying to minimize tissue damage while at the same time trying to achieve sufficient tissue shrinkage. Once shrunken (and devitalized), the tissues must be revitalized; during that period of repair, they are susceptible to elongation if left unprotected, due to compromise of their material properties.

### Maintenance of Initial Capsular Shrinkage

Experimental studies have shown that immediately after thermal shrinkage, connective tissues demonstrate a significant ( $P < 0.05$ ) loss

of stiffness.<sup>4,16,17,21</sup> This loss of stiffness has also been shown to be a significant important covariant of shrinkage (more shrinkage results in a greater loss of stiffness). Thus, tightening a lax joint by inducing thermal shrinkage in the joint capsule and/or ligaments makes these tissues weaker and more compliant, putting them at risk postoperatively.

It is also important to note that since most experimental studies have been performed on normal, healthy tissues, the heat response of abnormal tissues is unknown. It is possible that normal and abnormal tissues respond differently to thermal modification. One study has demonstrated an increased prevalence of reducible cross-links in joint capsules from joints demonstrating pathologic changes irrespective of the age of the patient.<sup>28</sup> This suggests that collagen from such joints may be more susceptible to thermal modification and could put them at even greater risk in the immediately postoperative period.

If the degree of tissue shrinkage is directly related to the loss of tissue stiffness in the immediately postoperative period, it is logical to assume that the degree of postoperative tissue repair is directly related to the extent of tissue damage. However, the application of thermal energy and the visual confirmation of the tissue response are both subjective. Indeed, in describing the technique, one author instructs that the probe should be swept slowly back and forth along the capsule "while the surgeon visually inspects for the capsular response."<sup>2</sup> With this in mind, it should be remembered that while it takes a temperature of at least 65°C to shrink connective tissues, a temperature of 45°C is lethal to most cells.<sup>26</sup> This suggests that after thermal application, at minimum, a significant portion of all visibly shrunk tissue has been

killed. In addition, it is possible that adjacent nonshrunk tissue has been subjected to lethal temperatures. Thus, the extent of actual tissue injury may not be accurately reflected by the visual impression of the amount of shrinkage.

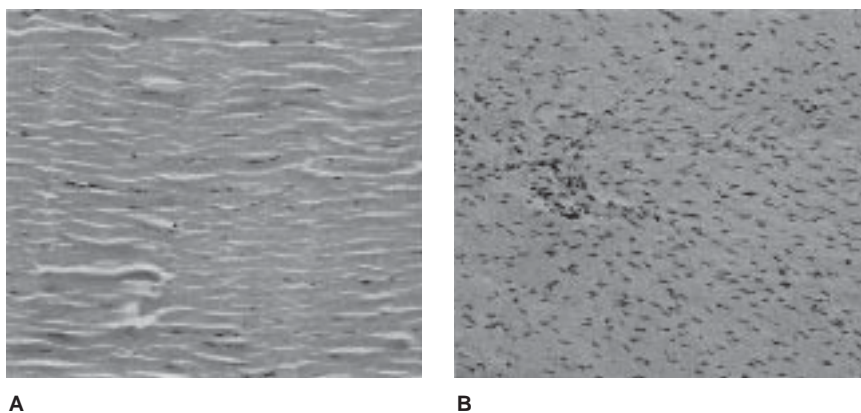
All traumatized or devitalized tissues induce an inflammatory response leading to repair and/or removal and replacement of the injured tissue. This process is more protracted after thermal injury than after traumatic incisional injury.<sup>26</sup> Numerous experimental studies have documented that thermally modified tissues undergo a repair and remodeling process characterized by fibroplasia (Fig. 3), increased collagen deposition, and a remodeling phase in which the collagen-diameter profile changes from a bimodal (large- and small-diameter) fiber pattern to a unimodal (small-diameter) fiber pattern indicative of scar tissue (Fig. 1, C).<sup>6,17,20,21</sup> These morphologic changes are associated with a decrease in tensile stiffness, making the tissue susceptible to elongation due to tissue creep.<sup>17,21</sup> Studies have suggested that at least 3 months is required before thermally modified tissue can fully recover its biomechanical properties.<sup>19</sup> Restriction

of activity during this time is a recommended consideration for the postoperative regimen. Thus, it appears that the maintenance of initial tissue shrinkage is directly dependent on controlling the postoperative stresses exerted on the tissue and is indirectly dependent on the amount of shrinkage induced at the time of surgery.

## Posttreatment Capsular Thickening and Cicatrix Formation

Inherent to all wound repair is a period of fibroplasia and collagen deposition that, in most cases, is directly related to the extent of tissue damage (caused by either the initial injury or the surgical treatment). Although extensive fibroplasia and subsequent tissue thickening are unwelcome sequelae of most surgical procedures, the creation of a hypertrophied joint capsule after thermal modification may actually contribute to the ultimate stability of the joint.

This method of shoulder stabilization has historical precedent. Hippocrates treated recurrent shoulder dislocations with insertion of a red-hot iron into the axillary region.



**Figure 3** Photomicrographs of a longitudinal section of a normal rabbit patellar tendon before (A) and 8 weeks after (B) thermal modification (hematoxylin-eosin, original magnification  $\times 100$ ). Note the marked fibroblastic response still present within the tissue at 8 weeks.



In the 1950s, Hackett<sup>29</sup> developed a treatment for the correction of lax ligaments and tendons by means of a minimally invasive technique involving the injection of various irritant solutions in an effort to incite a fibroblastic response. Focal injections of a dextrose solution were used to stimulate a generalized inflammatory reaction, which, in turn, led to the deposition of new collagen, making the thickened structure stronger and stiffer. Hackett termed this treatment "prolotherapy" (derived from the Latin root of "proliferate") and defined it as "the rehabilitation of an incompetent structure by the generation of new cellular tissue." He reported a gradual return of joint stability, but cautioned against the early mobilization of these joints, which he felt could result in disruption of the healing process and stretching of the tissue.

Some currently utilized open procedures for shoulder stabilization rely on tightening and reinforcement of the joint capsule to provide stability. It is logical to assume that the exposure and surgical trauma associated with these procedures could also result in postoperative

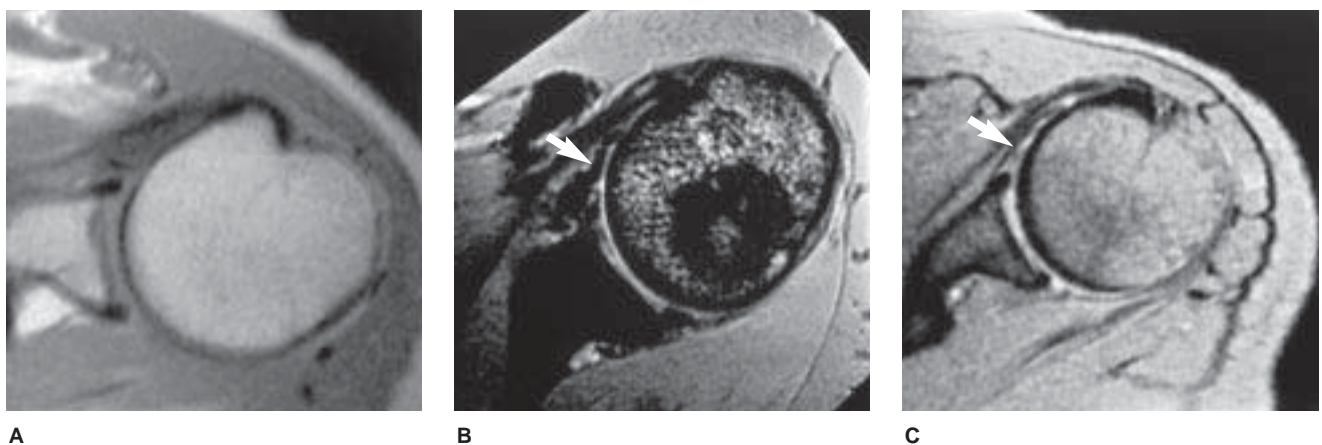
fibroplasia and thickening of the joint capsule, which may contribute to the ultimate stability of the shoulder. Therefore, the inability of arthroscopic shoulder stabilizations to equal the results of open stabilization procedures may be due, in part, to the decrease in tissue trauma (and attendant fibroplastic response of the joint capsule) associated with this minimally invasive technique. The use of thermal energy to incite an inflammatory cascade in the joint capsule and produce a fibroplastic response may induce the secondary capsular thickening seen in open stabilizations (Fig. 4).

A possible consequence of the creation of such a proliferative response is that excessive fibroplasia due to extensive tissue trauma may cause problems in the joint (e.g., arthrofibrosis, adhesive capsulitis). Appropriate stresses are required to remodel these healing tissues. This is especially true in the shoulder, where the portions of the capsule contributing to joint stability vary according to arm position. As with healing in all connective tissues, rehabilitation regimens must be developed to coincide with the various phases of repair in order to per-

mit functional remodeling of the maturing tissues (Fig. 5). As yet unresolved is the question of which is the best postoperative protocol for modulation of scar formation and remodeling after thermal modification.

#### Loss of Afferent Sensory Stimulation

The joint capsule of the shoulder is a richly innervated structure containing a variety of sensory receptors.<sup>30</sup> It seems logical to assume that temperatures that denature and shrink collagen ( $>65^{\circ}\text{C}$ ) can also destroy these receptors and alter, if not completely disrupt, the afferent sensory pathway of the shoulder. It has been suggested that in some arthroscopic surgical procedures on the shoulder, destruction (excision) of neural elements may decrease painful stimuli and thus allow more joint movement, strength, and function.<sup>30</sup> Therefore, thermal modification of the shoulder joint capsule could result in local or regional anesthesia of the joint capsule, providing symptomatic relief. In addition, the loss of neurosensory input secondary to thermal modification could make the shoulder more susceptible to future injury due to the



**Figure 4** A, Magnetic resonance image of a normal shoulder joint. The anterior joint capsule is indistinct. B, Image of a shoulder of another subject 1 year after open stabilization. Note the increased thickness of the anterior joint capsule (arrow). C, Image of a shoulder of yet another subject 1 year after arthroscopic thermal modification of the anterior joint capsule. Note the thickening of the capsule (arrow).



**Figure 5** Arthroscopic view of the anterior capsule of a shoulder joint 1 year after arthroscopic thermal capsular shrinkage. Note that the bands of fibrous tissue that have formed (arrows) are oriented at right angles to the normal course of the inferior glenohumeral ligament.

loss of reflex-protective muscle activity. However, this theory has yet to be proved.

## Summary

Wound healing is a natural and well-orchestrated biologic event. Indeed, the ability of wounds to heal is the foundation on which the

practice of surgery is predicated. The successful surgeon maintains a delicate alliance with Mother Nature, balancing the magnitude of the surgical insult against the capacity of the tissue for repair. Thermal injury is one of the most traumatic insults a tissue can sustain, and the high degree of cell death and matrix alteration associated with thermal burns has been shown to result in a protracted healing time. Thus, the use of thermal energy as a stimulant for tissue shrinkage must be tempered with an appreciation of the biologic events that accompany this phenomenon. Furthermore, it must be realized that the initial degree of capsular shrinkage observed after the application of thermal energy may have little bearing on the long-term biologic and biomechanical status of the joint capsule. Therefore, the desire to see a redundant capsule shrink and become taut at surgery should be weighed very carefully against the level of damage imparted to the tissue to achieve this result. In the words of Hackett, the simple initiation of the healing response may be, in itself, sufficient "to rehabilitate an incompetent structure through the generation of new cellular tissue."<sup>29</sup>

It is probable that no one mechanism is totally responsible for the

reported clinical improvement after thermal modification of the joint capsule. Indeed, it is entirely possible that all three of the mechanisms discussed—initial shrinkage, postoperative capsular thickening, and loss of regional afferent sensory input—may be involved in the ultimate postoperative improvement reported by some clinicians. Careful, long-term follow-up of these patients is needed to provide some insight into which factors have the greatest impact on functional outcome after thermal modification of connective tissues.

Although the ultimate applications of thermal modification of connective tissues have yet to be completely defined, its ultimate role may be as a low-level stimulant for inducing a biologic repair response, rather than as a highly aggressive mechanism for primary tissue shrinkage. As with all procedures, long-term, prospective clinical studies are required to define the precise indications and the appropriate therapeutic algorithm for this treatment modality.

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