INTRODUCTION

Although arthroscopic treatment of the unstable shoulder remains a challenging area of clinical development, in recent years, a better understanding of joint pathomechanics, coupled with significant technological advances, have increased the multitude of techniques at the surgeon’s disposal. Successive clinical results have generally improved, but controversy still exists as to whether an arthroscopic approach can be effective for all indications and patients.

The introduction of thermal capsular shrinkage was perceived by many as the ideal solution—using an old idea with a new twist to deal quickly and simply with the complex problem of excess capsular laxity. Unfortunately, the expansion rate of clinical applications has, to some extent, exceeded the rate of discovery of the biological and mechanical effects of capsular tissues derived from basic science. As more experimental studies have emerged, short-term clinical reports have also appeared, and it is now an appropriate time to consider the practical role of this innovation in the treatment of shoulder instability.

Similarly to the evaluation of any new treatment modality, two fundamental factors must be considered: whether it works and whether it is safe.

SCIENTIFIC BASIS

The theoretical basis for thermal shrinkage is well established, and the details of numerous basic science experiments are covered in a series of review articles (1–3). Shrinkage is a property of the major structural component of connective tissues—type I collagen, which is arranged in a fibrillar network. Essentially, the crystalline triple helix undergoes a phase change at a critical temperature, as the heat-labile intramolecular hydrogen bonds dissolve. Analogous to melting, this phenomenon is associated with a significant shortening as the ordered quarter-stagger array of the collagen molecules collapses, leaving a tangled coil of individual α chains still linked together by heat-stable covalent intermolecular bonds. Ultrastructurally, the collagen fibrils enlarge and their margins become less distinct, coalescing with adjacent fibrils in a denatured mass of protein in which the resident fibroblasts also undergo necrosis.
In vitro, capsular tissues and ligaments can shorten up to 50% of their original unconstrained length, but this may take several minutes. In vivo, most studies have demonstrated shortening in the range of 15–20% during short-term application of thermal energy. This limit may restrict the extent of reduction in capsular volume that can be achieved using the shrinkage technique. Nonetheless, decreased joint laxity and translation have been shown experimentally after shrinkage, but this effect has a consequence in mechanical terms. An inverse relationship exists between the extent of shrinkage and the tissue stiffness; as the tissue shortens, it becomes more elastic and liable to creep and stretch \((4–6)\). Strength is also reduced, but the recovery rate is still somewhat controversial. Some studies have suggested that mechanical properties are back to normal within 30 d, but others have indicated that it may be up to 90 d before strength and stiffness are sufficiently recovered \((7,8)\).

The mechanism of remodeling of the thermally treated tissue is not well understood. The major issue is whether the denatured matrix provides a useful scaffold for rapid repopulation by fibroblasts and new collagen synthesis or instead must first be removed and replaced completely, e.g., when a soft tissue defect heals in classic scar formation. Some in vitro evidence shows that heat-treated tissue can enhance fibroblast migration, and focal areas of new collagen production have been observed. In one study, biopsies from the operated shoulder capsule were taken from 53 patients between 3 and 38 mo after treatment at the time of stabilization of the symptomatic contralateral shoulder. Even at 6 mo, there was increased cellularity when compared with normal capsule, but by 12 mo, the histology appeared normal \((9)\).

Recent work from our laboratory has shown that the ultrastructure of thermally treated rabbit ligament tissue is similar to the scar tissue after gap healing in ligaments, with a shift seen from the normal bimodal distribution of large and small collagen fibrils toward a unimodal population of smaller fibrils \((10)\). The ultrastructure of ligaments that have been surgically plicated to achieve shortening is distinctly different; the large fibrils are still present, but there are bundles of smaller fibrils interspersed between them, suggesting another mechanism of matrix production (Fig. 1). Although the pattern of remodeling is different, viscoelastic properties in both groups are similar at 90 d, but still far from those of the intact control ligaments (Fig. 2).

Fig. 1. These transmission electron micrographs represent the medial collateral ligament (MCL) of the adult rabbit (A) before treatment, (B) at 12 wk after treatment with thermal shrinkage, and (C) at 12 wk after surgical division and plication of the ligament.