

Thermal Chondroplasty of Chondromalacic Human Cartilage

An Ex Vivo Comparison of Bipolar and Monopolar Radiofrequency Devices

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ABSTRACT

We compared the effects of treatment with bipolar and monopolar radiofrequency energy on 30 osteochondral sections harvested from 22 patients with spontaneously occurring chondromalacia who were undergoing knee arthroplasty. Specimens with chondromalacia grades 2 or 3 were randomly assigned to one of two bipolar or one monopolar treatment groups. All samples were marked and mounted on a jig to allow simulation of an arthroscopic surgical procedure with a flow rate of 100 ml/min of a balanced electrolyte solution at 22°C. Under arthroscopic visualization, the designated area was treated until smooth, and the total treatment time was recorded. There was no difference in patients' ages, chondromalacia grade, or cartilage thickness among groups. Significant chondrocyte death, as determined by cell viability staining with confocal laser microscopy, was observed with each group. The bipolar devices produced significantly greater depths of chondrocyte death ($2228 \pm 1003 \mu\text{m}$ and $2810 \pm 517 \mu\text{m}$) than did the monopolar device ($737 \pm 391 \mu\text{m}$). The bipolar devices caused cell death to subchondral bone significantly more often (13 of 20 specimens) than did the monopolar device (0 of 10 specimens). Caution should be used in treating fibrillated cartilage with radiofrequency energy, particularly with the bipolar devices tested.

Numerous methods have been described for treatment of localized and diffuse degenerative articular cartilage. Techniques such as partial or full-thickness mechanical debridement, marrow stimulation, autogenous/allogenic grafting in the form of osteochondral mosaicplasty or chondrocyte transplantation, and thermal chondroplasty all have been described and used to treat degenerative cartilage.^{4, 5, 7, 10, 14, 20, 21, 28, 30, 32, 35, 41, 47, 48, 50} The goal of these techniques is either to resurface the articular surface, ideally with hyaline or hyaline-like cartilage, or to smooth the fibrillated surface and minimize further cartilage degeneration through mechanical debridement or thermal chondroplasty.

Either partial-thickness or full-thickness mechanical debridement yields a relatively smooth surface macroscopically at the time of the operation; however, the surface typically undergoes degeneration over time. Microfracture of the subchondral bone bed is designed to allow marrow elements to fill the cartilage defect and form a stable articular surface, although most studies demonstrate that this technique typically yields tissue that is more fibrous in nature and not long-lasting.³⁶ Autogenous grafting, either in the form of osteochondral mosaicplasty or chondrocyte implantation has become popular over the past several years, with variable results.^{2, 3, 15, 17, 22, 36, 42, 43}

Over the past 3 years, the use of thermal chondroplasty for the treatment of degenerative cartilage has increased dramatically, particularly for the treatment of chondromalacia. Radiofrequency energy can be readily applied arthroscopically with relatively inexpensive equipment. Unfortunately, there are few basic scientific studies that have evaluated this treatment modality for chondroplasty, and the results of those that have been conducted are contradictory.^{21, 28, 48} Turner et al.⁴⁸ compared treatment

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with a bipolar radiofrequency device and mechanical debridement in a sheep model. They reported improved histologic appearance of the cartilaginous surface as long as 24 weeks after the surgical procedure. Repair tissue was subjectively scored using both gross and histologic appearance of the cartilage as the outcome measure. Kaplan et al.²¹ subsequently evaluated bipolar radiofrequency energy treatment of human chondromalacic cartilage in an ex vivo model. In their study, cartilage was evaluated using standard histologic techniques, and the authors concluded that bipolar radiofrequency energy was safe for use in thermal chondroplasty. Unfortunately, both of these studies used light microscopy for evaluation of cell viability, an insensitive technique, rather than the more accepted vital cell staining in conjunction with confocal laser microscopy.^{21,30,37,48}

Research conducted in our laboratory strongly contradicts the results of these previous studies. Lu et al.,³⁰ in a sheep model of partial-thickness articular cartilage defects, evaluated the long-term effects of monopolar radiofrequency energy application, compared with no treatment and with full-thickness debridement with microfracture. The authors demonstrated that a stable articular surface could be achieved with monopolar radiofrequency energy application and stability was maintained up to 6 months after the operation. However, all chondrocytes were dead within the defect in this relatively thin (mean thickness, 500 to 750 μm) cartilage model. The authors used vital cell staining with confocal laser microscopy to evaluate cell viability within the cartilage. Using a bovine articular cartilage model, Lu et al.²⁸ compared the three radiofrequency devices most commonly used for thermal chondroplasty. They concluded that treatment with the two commonly used bipolar radiofrequency energy systems resulted in much greater depth of chondrocyte cell death than did treatment with the monopolar system, and bipolar radiofrequency energy resulted in full-thickness chondrocyte death down to and including subchondral bone in the majority of applications. This study was criticized for its use of normal articular cartilage and for the absence of lavage flow while thermal chondroplasty was performed.

Further concern about the safety of thermal chondroplasty was raised by Lu et al.²⁹ in a comparative study of human chondromalacic cartilage treated with bipolar radiofrequency energy using lavage flow rates, lavage temperatures, and methods identical to those reported by Kaplan et al.²¹ In addition to light microscopy, confocal laser microscopy was used to evaluate cell viability within the cartilage. The authors of this study concluded that thermal chondroplasty using the "safe" settings proposed by Kaplan et al.²¹ yielded cell death from 1.5 to 2.4 mm deep. In this noncontact application with very limited exposure times (3 seconds), penetration of the subchondral bone occurred in 5 of 36 samples.²⁹

Given the criticisms of the previous study by Lu et al.²⁸ for its use of normal bovine cartilage, we compared the three most commonly used radiofrequency devices for thermal chondroplasty using naturally occurring fresh human chondromalacic tissue specimens. Chondroplasty

was performed in an ex vivo setting under arthroscopic visualization at the flow rates and lavage solution temperatures that are used clinically. We hypothesized that vital cell staining, using confocal laser microscopy, would demonstrate significant and full-thickness chondrocyte death from treatment with the bipolar radiofrequency energy devices, as demonstrated in our previous bovine study.

MATERIALS AND METHODS

All procedures were approved by the Human Subjects Committee and the Institutional Review Board at the universities involved in the described research. Osteochondral sections from the femoral condyles, the trochlear groove, the articular surface of the patella, and the tibial plateau from 22 patients undergoing knee arthroplasty were harvested and coded to prevent subject identification. Specimens were maintained at 4°C until the time of testing, at which time they were warmed to 37°C and then tested in room-temperature (22°C) irrigation solution. Osteochondral sections were graded macroscopically under arthroscopic visualization by two investigators (RBE, YL) and sequentially assigned to treatment groups to evenly distribute grades and age groups across treatments. Sections graded 2 (superficial cartilage fibrillation) or 3 (fibrillated cartilage surface with fissuring) from the patella and femoral condyle were used for this project.^{8,26,27} The subjects' age, sex, and the location from which the osteochondral section was obtained were recorded. After classification, a circular 1.5 cm diameter area (1.8 cm²) was marked on the cartilage surface. The order of treatment was assigned randomly before the beginning of the study.

Three radiofrequency generator/probe combinations were evaluated: ArthroCare 2000 with CoVac 50 probe, setting 2 (ArthroCare, Sunnyvale, California); Mitek VAPR version 2.1 with 3.5-mm side-effect probe, setting V2-40 (Mitek Corporation, Norwood, Massachusetts); and Vulcan EAS 3.12 with a TAC-C probe, temperature control setting (70°C, 30 watts) (ORATEC Interventions, Inc., Menlo Park, California). All samples were mounted in a custom-designed jig (Fig. 1) to allow simulation of an arthroscopic surgical procedure with a flow rate of 100 ml/min of a balanced electrolyte solution at room temperature (22°C). This flow rate was based on a calculated mean flow rate used during arthroscopic surgery and was selected to simulate intraarticular flow during arthroscopy while maintaining uniformity across treatment groups. The designated area was treated under arthroscopic visualization until smoothing and removal of surface fibrillation were accomplished, and total treatment time was recorded. The end point was reached when fine fibrillations had been eliminated and the surface was smooth in appearance and when probed. Regions with delaminated cartilage were avoided during the selection of tissue, and no attempt was made to ablate tissue fragments or large fronds. The movement of the probes was related to the response of the tissue but was usually 15 to 20 mm/sec across the articular surface. The same person treated all of the sections. It was not possible to blind operators as to the probe/generator that was used because

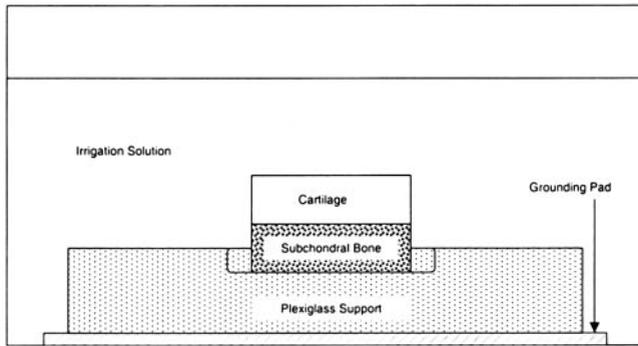


Figure 1. Schematic of the chamber used to treat osteochondral sections with bipolar and monopolar radiofrequency energy devices. Osteochondral sections were positioned on an acrylic block to insulate them from the grounding pad when monopolar radiofrequency energy was used.

all treatments were observed arthroscopically and each of the probes has a distinct appearance and each generator makes a distinct sound when activated.

After treatment, adjacent sections were prepared for cell viability staining with confocal laser microscopy and matrix architecture evaluation with light microscopy. To avoid thermal injury, we used a diamond wafering blade (Isomet 2000 Precision Saw, Buehler Ltd., Lake Bluff, Illinois) to cut 1.5-mm serial sections from the treated region under constant irrigation with phosphate-buffered saline solution at room temperature (22°C). In addition, sections were cut from areas distant to the treated area to assess overall cell viability and the effects of irrigation fluid used during treatment. After cutting, the sections were placed in 1.5-ml vials with 1.0 ml of phosphate-buffered saline solution and placed in a refrigerator at 4°C for 120 minutes before cell viability staining.

Cell viability was determined with use of ethidium homodimer (10- μ l) and calcein (0.5- μ l) fluorescent stains (LIVE/DEAD Viability/Cytotoxicity Kit [L-3224]), Molecular Probes, Eugene, Oregon) and confocal laser microscopy, as previously described.³⁰ The 1.5-mm osteochondral sections were incubated with the staining agents for 30 minutes at room temperature, placed on a glass slide, and moistened with several drops of phosphate-buffered saline solution. A confocal laser microscope (MRC-1000, Bio-Rad, Hemel Hempstead/Cambridge, England) equipped with an argon laser and necessary filter systems (fluorescein and rhodamine) was used with a triple-labeling technique. In

this technique, the signals emitted from the double-stained specimens can be distinguished because of their different absorption and emission spectra.^{12, 16, 29, 30, 37} These images were displayed on a monitor in a red, green, and blue mode. Untreated control regions on each treated osteochondral section were examined to ensure chondrocyte viability. All cartilage samples were examined blindly.

The sections used for light microscopy were fixed in 10% neutral-buffered formalin, decalcified, and embedded in paraffin. Sections of 5.0- μ m thickness were cut and stained with hematoxylin and eosin^{11, 31} to determine chondrocyte cytoplasmic and nuclear morphometry and shape and with safranin O^{11, 24, 25, 31, 40} to stain proteoglycan within the extracellular matrix. All sections were coded to prevent identification of the probe type or setting used. The sections were examined and the chondrocyte appearance and safranin O staining were assessed.

The depth of chondrocyte death and cartilage thickness of each section in the confocal laser microscope image was determined for each region treated with radiofrequency energy, with all images coded to prevent identification of the probe and treatment time applied. The confocal laser microscope was calibrated using a micrometer measured through the objective lens ($\times 2$) used for this project ($\times 20$ total magnification, objective + eyepiece magnification). The pixel length measured on images was converted to micrometers, as previously described.³⁰ The depth of chondrocyte death and cartilage thickness of sections were determined for each confocal image of the osteochondral sections with Adobe PhotoShop (Adobe PhotoShop, version 5.5, San Jose, California).²⁸⁻³⁰ Differences in depth of chondrocyte death and treatment time among groups were assessed with analysis of variance (SAS, version 7.1, SAS Institute, Cary, North Carolina). When differences were identified among groups, Duncan's post hoc test was employed. Penetration to the subchondral bone among treatment groups was compared with Fisher's exact test. The Wilcoxon signed rank test was used to compare cartilage grades and sex differences among groups. *P* values less than 0.05 were considered significant.

RESULTS

There were no differences in patient age, chondromalacia grade, or total cartilage thickness among the groups (Table 1). Each radiofrequency device was able to contour the articular surface and ablate fine surface fibrillations, but

TABLE 1
Comparison of the Results of the Three Radiofrequency Energy Devices^a

Radiofrequency device	Patient age (years)	Chondromalacia grade	Cartilage thickness (microns)	Treatment time (seconds)	Depth of chondrocyte death (microns) ^b
ArthroCare 2000	64 \pm 8	2.1 \pm 0.3	2853 \pm 799	24.2 \pm 9.0	2228 \pm 1003
Mitek VAPR	67 \pm 11	2.2 \pm 0.4	3362 \pm 657	23.1 \pm 6.9	2810 \pm 517
ORATEC Vulcan	66 \pm 7	2.2 \pm 0.4	3092 \pm 1103	39 \pm 18.5 ^c	737 \pm 391

^a Data represent mean \pm SD.

^b Significant differences between all three groups (*P* < 0.05).

^c Significantly different from the ArthroCare and Mitek groups (*P* < 0.05).

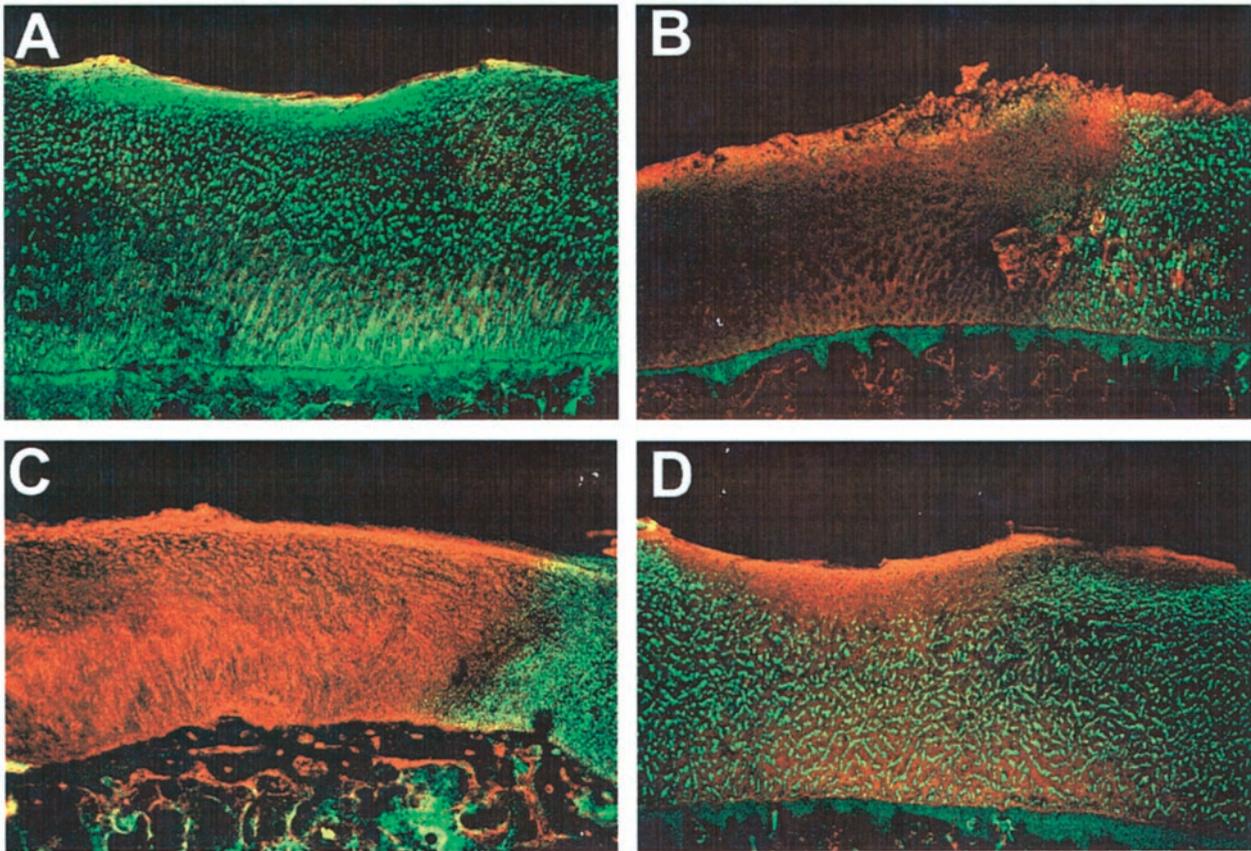


Figure 2. Confocal laser microscopy of tissue sections treated with radiofrequency energy ($\times 20$). A, control section; B, ArthroCare 2000; C, Mitek VAPR; D, ORATEC Vulcan. On each image, the top is the cartilage surface, the bottom is the subchondral bone, the green dots are live chondrocytes, and the red dots are dead chondrocytes.

none of the devices were used to ablate large fronds (>1.0 mm). Macroscopically, radiofrequency energy treatment resulted in smoothing, contouring, and shrinkage of the articular surface. A slight alteration in color, from white to

light yellow, occurred during thermal chondroplasty. In all cases, radiofrequency energy was applied in a manner that avoided charring the articular surface. Blanching of the articular surface was minimized by avoiding contact of

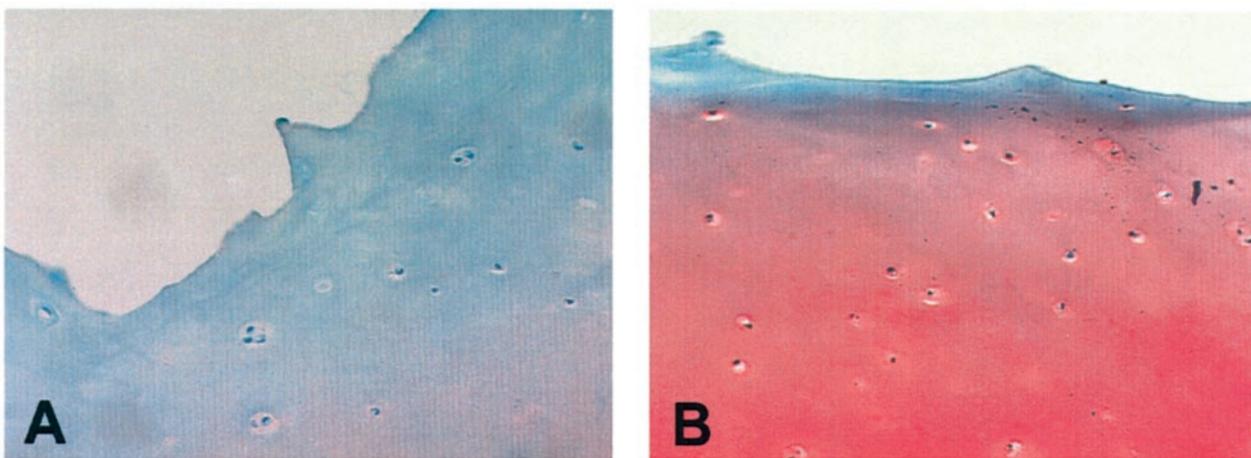


Figure 3. Safranin O-stained light microscopy images of chondromalacic cartilage treated with bipolar radiofrequency energy (A) and monopolar radiofrequency energy (B). Note the loss of staining in the section treated with bipolar radiofrequency energy compared with the monopolar-treated section. (original magnification, $\times 200$)

the bipolar devices with the articular surface. In all samples, control cartilage demonstrated viable chondrocytes with no cell death (Fig. 2).

The bipolar radiofrequency energy devices produced chondrocyte death to a greater depth and smoothed the surface faster than did the monopolar device ($P < 0.05$) (Table 1) (Fig. 2). Chondrocyte death to the level of the subchondral bone occurred with each bipolar device evaluated (ArthroCare, 6 of 10, and Mitek, 7 of 10) but not with the monopolar device (ORATEC, 0 of 10) ($P < 0.05$). Light microscopic analysis revealed reduced proteoglycan staining in the cartilage regions treated (Fig. 3).

DISCUSSION

The results of this study confirm those of our previous *ex vivo* and *in vivo* studies that evaluated the use of radiofrequency energy for thermal chondroplasty.^{28–30} The present study was designed to mimic the clinical application of radiofrequency energy for thermal chondroplasty of human chondromalacic lesions as closely as possible. The manufacturer's recommended settings (as published at the time this work was conducted) were used to perform this study. It was not the goal of this project to determine the optimal power setting and probe combination to perform safe yet effective thermal chondroplasty with radiofrequency energy. Fresh osteochondral specimens were treated using room-temperature lavage (22°C) at flow rates that are used clinically (100 ml/min) under arthroscopic visualization (G. Fanton, MD, personal communication, 2000). Lesions were treated as they would be clinically: noncontact for the bipolar devices and light contact for the monopolar device. In all cases, the surface was treated until surface fibrillations were smooth, and caramelization and char of the surface were avoided. Mean treatment times varied from the mid-20-second range for both bipolar devices to approximately 49 seconds for the monopolar device. Despite the care taken to minimize energy delivery or overheating of the cartilaginous surface, significant chondrocyte death occurred in all groups, with three to four times greater depth of chondrocyte death caused by the bipolar devices than by the monopolar device. Full-thickness chondrocyte death, down to and including the subchondral bone, occurred in 6 of 10 ArthroCare applications and 7 of 10 Mitek applications. Penetration of the subchondral bone did not occur with use of the ORATEC monopolar device, despite treatment times that were almost double those of the bipolar devices.

This study confirms that the power-controlled bipolar radiofrequency energy devices tested cause much greater cell death and have a higher likelihood of penetrating the subchondral bone than does the temperature-controlled monopolar device. There are two major differences between the bipolar devices used in this study and the monopolar device. First, the bipolar devices deliver energy through the irrigating solution that surrounds the probe tip. Conductive heating of the adjacent cartilage matrix results in contouring of the surface. It would seem logical that lower settings and reduced application time would reduce the depth of thermal injury to chondrocytes. How-

ever, it is unknown whether lower settings would allow contouring of the articular surface or whether excessive time would be required to achieve the desired smoothing, resulting in excessive temperatures in the cartilage matrix. In addition, previous work has demonstrated that treatment times of 3 seconds have produced chondrocyte death at a depth of $1930 \pm 600 \mu\text{m}$ with the ArthroCare probe (CoVac50) and the setting 2 that was used in this work.²⁹

Energy from the monopolar system may travel through one of two routes or some combination of the two. It may pass through the articular cartilage, subchondral bone, and surrounding soft tissues to the grounding plate, or, as likely occurs in this situation, through the lavage solution, joint capsule, and surrounding soft tissue to the grounding plate. In reality, the energy paths are likely to be some combination of the two. During the application of monopolar radiofrequency energy to articular cartilage, the preferential path is likely to be through the irrigation solution rather than through the subchondral bone because it is the path of least resistance. The second major difference between the bipolar devices available on the market at the time of this study and the monopolar device is the absence of a temperature control in the bipolar devices. The only mechanism for controlling temperature with the bipolar devices is through power modulation, the set power. In contrast, the monopolar device uses a thermocouple imbedded in its tip to control the surface temperature of the probe by modulating the power delivery. Studies have shown that the precision and accuracy of temperature delivery for the Vulcan EAS (version 3.12) system is extremely high (precision, 97.5%; accuracy, 96.2%).¹³ Because the temperature of the probe tip is controlled by oscillating the power, the delivered power during monopolar application is much lower than that for the bipolar devices. Typically, the monopolar device operates at approximately 10 watts during thermal chondroplasty, whereas the bipolar devices operate at 20 to 40 watts or higher. Recently, Mitek has introduced the VAPR-II, and this generator has the ability to modulate temperature through thermistors within the probes. Future work will be needed to investigate the ability of this device to contour articular cartilage and its effect on chondrocyte viability.

Thermal injury in the cartilage matrix has far different ramifications than injury to soft tissue structures, such as the joint capsule or intraarticular and periarticular ligament structures. The absolute temperature that is required to modify the cartilage matrix is unknown. We do know that type I collagen is denatured at 65°C and that optimal capsular shrinkage occurs at approximately 75°C. At 75°, thermal necrosis extends 3 to 5 mm below the synovial surface. However, in capsular tissue, with proper postoperative motion restriction and physical therapy, the mechanical characteristics of the capsule are restored in 6 weeks. Recent articles have documented the successful use of radiofrequency energy on ligament tissue,³⁸ but the risk of excessive application of radiofrequency energy or of resumption of activity too rapidly after the thermal modification of ligament structures such as the ACL has also

been demonstrated.^{39,44} However, thermal injury to chondrocytes poses an even greater problem because of the inability of cartilage to repair itself, especially when the cells responsible for matrix homeostasis have been eliminated.

There has been controversy regarding the cause of cell death that occurs during thermal chondroplasty. In a recent study, Edwards et al.¹³ used fluoroptic thermometry to confirm that higher temperatures are achieved with the ArthroCare bipolar device as compared with the ORATEC monopolar device. During chondroplasty with the ArthroCare bipolar device, temperatures in excess of 75°C were measured as deep as 2 mm underneath the cartilaginous surface. With the ORATEC monopolar device, cartilage temperatures were consistently below 40°C at 2 mm under the surface. Several studies have demonstrated that temperatures in excess of 55°C will result in cell death.^{1,23} Our study confirms the findings of previous confocal laser microscopic studies that used vital cell staining by demonstrating that temperatures sufficiently high to kill cells are achieved within the cartilage matrix during chondroplasty.

The validity of the use of confocal laser microscopy with vital cell staining to determine cell viability within cartilage has been questioned by some investigators.⁵¹ With use of this procedure, ethidium homodimer penetrates the dilated pores of dead or dying cells, causing these cells to fluoresce red. Calcein green is transported into viable cells and converted to calcein AM via esterase activity. This technique has been used for many tissues, including neural and cardiac tissue and cartilage.^{9, 12, 18, 19, 33, 45, 46} Previous *in vivo* and *in vitro* studies have validated this technique in cartilage.^{12, 16, 30, 37} In an *in vivo* sheep study, Lu et al.³⁰ demonstrated that monopolar radiofrequency energy caused approximately 300- μ m of cell death immediately after its application. Animals were observed for as long as 6 months after the procedure. In no case did cells that had previously fluoresced red subsequently fluoresce green. In fact, further cell death occurred over time, resulting in a greater degree of chondrocyte death than was indicated by the initial energy application. Lu et al.³⁰ hypothesized that continued necrosis or subsequent apoptosis of adjacent chondrocytes, or both, may have been responsible for the continuing cell death that occurred over the course of the study.

Some investigators have argued that pore dilation occurs from the heat generated by thermal chondroplasty, causing leakage of ethidium homodimer into otherwise viable cells.⁵¹ There are two major drawbacks to this reasoning. First, vital cell staining is performed at 22°C after the sections have been stored at 4°C for 120 minutes, so the cells are no longer heated while they are being stained. Second, if ethidium homodimer is able to penetrate the pores in these cells, then many other solutes, including numerous cations and anions, may also be able to penetrate the cell, likely resulting in cell death. It is important to emphasize that this method uses a double-staining technique, and in no case have we observed cells that stain both red and green. Therefore, for these theoretically viable cells to fluoresce red because of pore dilata-

tion and not also fluoresce green would be a further indication that they are dead.

Another potential cause of a false-positive reading (red-staining cells) is that the negatively charged matrix attracts ethidium homodimer, thereby staining the matrix red.⁵¹ This would not explain why the cells in this region do not also fluoresce green, which is not the case after radiofrequency energy application. Investigators have also claimed that heat shock to the cells may occur during this application, causing temporary uptake of ethidium without the ability to convert calcein to calcein AM.⁵¹ In no case did previously red-stained cells subsequently demonstrate viability by fluorescing green in this *in vivo* study or in the previous study by Lu et al.³⁰ These issues highlight two factors that are important when vital cell staining and confocal laser microscopy are performed. First, adequate controls must be incorporated into the study design, and, second, all groups must be treated identically during vital cell staining and confocal laser microscopy. In all cases that have been reported evaluating cartilage viability after radiofrequency energy application, bipolar application has caused significantly more death, down to and including the subchondral bone, compared with monopolar application.^{28,29}

Many questions remain after this study. There have been only two studies that have evaluated the potential long-term ramifications of radiofrequency energy application. Turner et al.⁴⁸ evaluated the use of bipolar radiofrequency energy for chondroplasty in a sheep model and concluded that tissue specimens from treated regions had better histologic grades (less surface irregularities, less chondrocyte cloning, less hypocellularity, better staining matrix, and less light microscopic evidence of chondrocyte death) than did those from mechanically debrided sites. They used only light microscopy to evaluate chondrocyte viability. In the study by Lu et al.³⁰ in a sheep model, a stable cartilaginous surface was present after monopolar radiofrequency energy application for as long as 6 months after the procedure, although all cells within the treated region were dead. Unfortunately, the sheep cartilage used in that study was relatively thin (750 to 1000 μ m). After the 250- μ m partial-thickness defect was created, only approximately 500 to 750 μ m of cartilage thickness remained before application of radiofrequency energy. Monopolar radiofrequency energy application with current technology kills cells to as deep as 737 μ m, as demonstrated in the current study. Animal models must use cartilage that more closely mimics the thickness of human cartilage to better understand the effect of either partial-thickness or full-thickness chondrocyte death on the long-term stability of the cartilaginous surface.

Theoretic benefits of thermal chondroplasty include the ability to accurately debride cartilaginous defects without damaging adjacent unaffected cartilage, to physically smooth the surface, and to apply energy via arthroscopic delivery. Stabilizing the surface with this melting process may possibly reduce the cyclic inflammation within the joint that can be caused by release of collagen and proteoglycan epitopes.^{10,21} Studies have shown that cartilage wear debris can cause cells within the synovial membrane

to release metalloproteinases, prostaglandins, free radicals, tumor necrosis factor, and interleukin-1.^{4–6,36,49} Each of these substances has the potential to degrade the cartilaginous surface and cause further release of cartilage epitopes. One of the major justifications for radiofrequency energy application is that, by sealing the surface with heat, the release of these inflammatory cytokines is minimized or eliminated, thereby reducing the joint inflammation and pain of patients with chondromalacia. Both human clinical trials and animal studies need to be conducted to determine whether radiofrequency energy actually results in this positive effect.

Chondrocytes within the articular cartilage bed have limited, if any, ability to heal after thermal trauma. Therefore, if a significant number of chondrocytes are killed during radiofrequency energy application, the matrix cannot be maintained and will likely fail over time because of cyclic fatigue. Previous studies conducted in our laboratory have demonstrated that, after chondrocyte death, proteoglycan concentrations within the articular cartilage decline dramatically.³⁰ The long-term effects of this loss of proteoglycan or maintenance of the normal matrix components have not been evaluated. It is our belief that, for thermal chondroplasty to be successful, chondrocyte death must be minimized while melting the surface. This can be achieved through delivery of lower power, through tight temperature control of the radiofrequency probe tip, and through minimal application of radiofrequency energy to the surface. It has been shown that current treatments, such as mechanical debridement, result in 150 to 250 μm of cell death over time after debridement of the surface.³⁰ We propose that the long-term goal of radiofrequency energy application should be to limit cell death to no more than that currently seen after mechanical debridement, that is, 250 μm or less. Currently, monopolar radiofrequency energy causes approximately 700 to 800 μm of cell death in cartilage that is typically 2000 to 5000 μm thick. The long-term effect of having 15% to 40% of the depth of the cartilage dead with a stable surface is unknown. Studies of the effect must be performed before this application can be recommended for use in patients with chondromalacia. Radiofrequency application for articular cartilage should be limited to experimental trials until the long-term ramifications of thermal smoothing with a depth of 700 to 800 μm of chondrocyte death are evaluated. We propose that, if surgeons plan to perform thermal chondroplasty on their patients, the safest device, the monopolar device, should be used only in areas that have been shown to have relatively thick cartilage (more than 2 mm thick).

Potentially, three questions should be addressed with regard to the use of radiofrequency energy for thermal chondroplasty: 1) How effective is the technique? 2) How safe is it? and 3) What are the long-term consequences of radiofrequency energy treatment? On the basis of *in vitro* studies and the anecdotal evidence of experienced sports medicine surgeons, radiofrequency energy has the ability to smooth the soft fibrillated surfaces encountered among patients with grade 2 and grade 3 chondromalacia. Mechanical debridement has been the standard of treatment,

but no ideal treatment has been identified. However, all surgeons should be cautioned that thermal chondroplasty with radiofrequency energy treatment has not been evaluated in a prospective manner, compared objectively with other treatment modalities, and results of this treatment have not been observed over an extended time course. In addition, the effect of arthroscopic lavage, the placebo effect on the patient, and the surgeon's influence on patient outcome cannot be underestimated.³⁴

The peer-reviewed literature offers conflicting opinions with regard to the safety of thermal chondroplasty performed with radiofrequency energy.^{21,28–30,48} However, the objective measurement of matrix temperatures and cell viability stains supports the conclusion that thermal chondroplasty with radiofrequency energy has the potential to create significant cartilage injury, potentially extending to the level of subchondral bone with resultant avascular necrosis. The long-term consequences are completely unknown at this time. Some information can be gleaned from the *in vivo* sheep study by Lu et al.³⁰ In this partial-thickness articular cartilage defect model, mechanical debridement and thermal chondroplasty with monopolar radiofrequency energy were compared. Over a 6-month period, continued fibrillation and fissuring was seen in the tissue of the mechanical debridement group. However, in the monopolar-treated group, a smooth articular surface remained despite full-thickness chondrocyte death in the treated regions.³⁰ Many questions remain to be answered: 1) How long will the matrix remain intact without viable chondrocytes supporting its function? 2) Will a load-bearing region remain stable in the same manner as the trochlear groove region studies by Lu et al.³⁰ 3) What is the response of the surrounding viable chondrocytes? Will they increase proteoglycan and collagen secretion or increase the release of degradative enzymes? 4) Is the surface more stable or has thermal modification lowered this region such that the surrounding normal cartilage is bearing the load? These and many other questions remain regarding the effects of radiofrequency energy on chondrocyte and articular cartilage matrix function.

In conclusion, thermal chondroplasty is an unproven technique and should not be performed in patients, particularly with bipolar devices, until further experimental animal studies and human clinical trials demonstrate the technique's safety and efficacy.

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