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Monopolar Radiofrequency Energy Application to the Dorsal Extensor Tendon Apparatus in a Canine Model of Tendon Injury

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Abstract

Purpose— To evaluate the use of monopolar radiofrequency energy (MRFE) to shorten stretched dorsal extensor tendon apparatus (DETA) tissues in a canine model.

Methods— Eleven adult canine forelimbs were used in this *in vitro* investigation. The DETA tissue was isolated between the metacarpophalangeal and proximal interphalangeal joints in the third and fourth digits of each limb. Isolated tissue was stretched in all but 2 of the digits (control group). After tissue stretching, monopolar radiofrequency energy (MRFE) was applied to 18 of the digits at 1 of 3 temperatures: 50°C, 60°C, or 70°C (stretch-treatment group). Two digits were treated identically, but MRFE was not applied (stretch-only group). Tissue length was measured before and after stretching and after treatment. Percent stretch, percent shortening, and percent original length were compared among the 3 stretch-treatment groups. All DETA specimens were examined with light microscopy.

Results— Histologic changes were apparent in the stretch-treatment and stretch-only specimens compared with controls. Percent stretch was not significantly different between groups. Percent shortening and percent original length were significantly lower and higher, respectively, in the 50°C group than in the 60°C and 70°C stretch-treatment groups, which were not significantly different from each other. There was a significant linear correspondence between percent shortening and treatment temperature.

Conclusions— The application of MRFE at a temperature of 60°C and a power of 10 W appears to shorten stretched DETA tissue to approximately the prestretched length in an *in vitro* canine model. Further investigation is necessary to determine the effect of treatment on the tissue's mechanical properties, (*J Hand Surg* 2006;31A:1296–1302.

Keywords

Extensor; model; DETA; radiofrequency; tendon

Digital tendon injuries are common, and extensor tendons are particularly prone to damage.^{1–4} The anatomy of the dorsal extensor tendon apparatus (DETA) and the dynamic balance between the digital extensor and flexor mechanisms are well described.^{1,2,5} The proximal interphalangeal joint (PIPJ) is the most common joint in the hand to sustain injuries.⁴ Trauma to the DETA at the level of the PIPJ can result in specific pathologic conditions that are staged based on duration, deformity, and concomitant secondary changes.^{3,4,6,7} Because of its

complex structure and function, increases in length of the DETA severely affect finger function.^{1,3,8,9}

The extent of extensor mechanism damage can be difficult to assess acutely,^{10,11} and seemingly mild cases can result in chronic digital deformities that impair normal function.^{10–12} Treatments for DETA injuries are designed to allow the DETA to heal at its proper length.¹³ This is usually accomplished with splinting and physical therapy, although surgical management is necessary for established or refractory cases, and there are potential complications associated with open procedures.⁸ Standard splinting techniques are generally prolonged, and patient compliance can wane with time.^{1,8,14} The sheer volume of information surrounding the management of the various severities and stages of DETA disorders attests to the lack of a gold-standard therapy.^{2,3,6,8–10}

Monopolar radiofrequency energy (MRFE) is a modality that has been used to treat musculoskeletal pathologies, especially in the knee, shoulder, and hip.^{15–17} Monopolar radiofrequency energy has been used to stabilize joints by tissue shrinkage through thermal denaturation of collagenous tissue.^{15–18} In this article we explore MRFE application to digital tendinous tissue.¹⁹ Although there are established animal models for digital flexor tendon injuries, there is a lack of models for extensor mechanism trauma.^{20,21} The canine DETA at the level of the PIPJ is anatomically comparable with that of the human.²² Specifically, just proximal to the PIPJ, the canine DETA is composed of the central slip connected to 2 lateral bands by the triangular ligament, and the central slip inserts on the second phalanx. This study was performed to evaluate the use of MRFE to shorten stretched DETA tissues in a canine model. We hypothesized that shrinkage of redundant DETA tissue would correlate with MRFE energy application. Our goals were to identify an appropriate MRFE application technique to return tissue to the prestretched length and to characterize associated histologic tissue changes.

Materials and Methods

Forelimbs from 6 adult female dogs weighing 20.9 ± 1.0 kg (mean $\pm$ standard error of the mean [SEM]) were transected at the midradius immediately *post mortem*, wrapped in saline-soaked towels, sealed in 2 plastic bags, and stored at -20°C . Limbs were thawed and allowed to reach room temperature immediately before use. The third and fourth digits from each limb were randomly assigned to 1 of 5 treatment groups in a randomized block design: stretching and MRFE treatment at (1) 50°C , (2) 60°C , or (3) 70°C , all with power set at 10 W (stretch-treatment group) ($n = 6/\text{treatment group}$); (4) stretch-only group ($n = 2$); or (5) control group ($n = 2$). All tissue stretching and MRFE treatments were performed by a single investigator.

Each paw was clipped and cleansed, and a no. 10 scalpel blade was used to excise skin from the dorsal surface of all third and fourth digits. The DETA tissue was isolated between the metacarpophalangeal joint and PIPJ by dissection of the connective tissue just palmar to each lateral band from the proximal aspect of the first phalanx to the slip insertion on the second phalanx (Fig. 1). Single simple interrupted sutures (no. 3-0 nylon) were placed transversely, 15 mm apart in the central slip. The distalmost suture was just proximal to slip insertion. Tension (3 N) was applied to the proximal aspect of the central slip with a bar-type tension gauge (McMaster-Carr, Chicago, IL), and the distance between the distal and proximal aspects of the proximal and distal sutures, respectively, was measured with a dial caliper (Bestool-Kanon, Tokyo, Japan) with a resolution of 0.02 mm.

A previously described²³ stretching device was used to stretch DETA tissue in the stretch-treatment and stretch-only treatment groups (Fig. 1). The device had a hollow cylinder with a 7.5-mm inner diameter and a 10-mm outer diameter at the tissue interface surface. The rim of the surface was 2.5 mm wide and had a textured surface equivalent to 80-grit sandpaper. Within

the cylinder was a 10-gauge removable stainless steel hook (5.0-mm diameter, 1.5-mm depth) attached to a ratchet. The system permitted smooth hook retraction and locked in place every 1.5 mm. The midpoint of the length of the central slip between the 2 sutures was identified, and the hook was passed from medial to lateral beneath the central slip, lateral bands, and triangular ligament of the DETA at that level (Fig. 1). The hook was then retracted until its distalmost aspect was level with the textured distal cylinder rim, effectively containing the indicated DETA tissues within both the hook and cylinder of the device. Firm pressure was applied to the cylinder to engage the textured surface of the rim with surrounding tissue, preventing slippage of tissues contained within the device during hook retraction and limiting stretched tissue to that within the cylinder (Fig. 2). The hook was retracted at a rate of 0.4 to 0.5 mm/s to a displacement of about 3.0 mm, which was held for 1 minute then released.²⁴ After stretching, the distance between sutures was measured as before. Limbs were kept in normal saline at room temperature for 30 minutes, and measurements were performed again as described earlier.²⁵

The application of MRFE was accomplished by submerging each paw in room-temperature normal saline with the palmar aspect of each digit resting lightly on the 14 × 18-cm dispersive electrode of the MRFE generator (Vulcan Electrothermal Arthroscopy System; Smith & Nephew, Inc., Andover, MA). Energy was applied to the tissue with a 1-mm-diameter MRFE probe (miniTACS; Smith and Nephew, Inc., Andover, MA) in transverse passes at a rate of approximately 1 mm/s beginning just distal to the proximal suture and ending just proximal to the distal suture. The location of each probe pass was apparent because of a change in the appearance of treated tissue, and care was taken to avoid overlapping passes. Tissues in the stretch-only treatment group were treated identically with the MRFE probe, but energy was not applied. The distance between sutures was measured after MRFE probe application as described earlier.

The DETA tissues were collected by sharp transection approximately 3 mm beyond each suture. The tissue was fixed in 10% neutral buffered formalin. The samples were embedded in paraffin, and sagittal sections (5 μm) were processed for histologic staining with hematoxylin-eosin. The effects of both stretching and MRFE treatment on tissue fibrocyte morphology and collagen fiber architecture were subjectively evaluated with light microscopy.

Percent elongation, percent shortening, and percent original length were calculated for each specimen in the stretch-treatment and stretch-only treatment groups as follows:

$$\begin{aligned} &\text{percent elongation} \\ &= [\text{absolute value}(\text{prestretch length} \\ &\quad - \text{poststretch length}) / \text{prestretch length}] \times 100 \end{aligned}$$

$$\begin{aligned} &\text{percent shortening} \\ &= [\text{absolute value}(\text{prostretch length} \\ &\quad - \text{posttreatment length}) / \text{prostretch length}] \times 100 \end{aligned}$$

$$\begin{aligned} &\text{percent original length} \\ &= ([\text{absolute value}(\text{prestretch length} \\ &\quad - \text{posttreatment length}) / \text{prestretch length}] \times 100 + 100) \end{aligned}$$

The mean $\pm$ SEM for each variable was determined. One-way analysis of variance with Tukey *post hoc* analysis was used to evaluate differences among the 3 stretch-treatment groups (50°C, 60°C, 70°C) for all calculated values. Least squares linear regression was used to evaluate the relationship between treatment temperature and percent shortening in the 3 groups. Significance was set at p less than .05. All statistical analyses were performed with commercially available software (GraphPad Prism version 4.0; Graphpad Software, Inc., San Diego, CA). Based on preliminary findings that histologic changes were very consistent in the stretch-only and control treatment groups, only 2 digits were assigned to the stretch only and control groups to limit the number of specimens necessary for the study. These specimens were included to permit comparison of the tissue changes caused by stretching alone and those caused by stretching followed by MRFE treatment with controls. They were not included in any statistical analyses.

Results

Tissue disruption or laceration was not evident after stretching. Stretched tissue appeared flaccid compared with normal. The MRFE treatment caused visible tissue shrinkage and a change in color from glistening white to dull off-white. Distinct histologic changes were apparent in both the stretch-treatment and stretch-only treatment group specimens compared with the controls (Fig. 3). Control tissue had characteristic wavy, distinct collagen fibers and cigar-shaped fibroblast nuclei (Fig. 3A). Stretched tissue was characterized by diminished collagen fiber waveforms, less discernable collagen fibers, and elongated fibroblast nuclei (Fig. 3B). Fibroblast nuclei were pyknotic, and there was a complete loss of collagen fiber architecture and waveforms in the stretched treated tissue (Fig. 3C). Histologic changes were similar in all temperature treatment groups. There were clear demarcations between normal and stretched tissue and between untreated stretched tissue and treated stretched tissue.

Results for the stretch-only and 50°C, 60°C, and 70°C stretch-treatment groups, respectively, were as follows: percent stretch was $13\% \pm 1\%$, $12\% \pm 1\%$, $10\% \pm 1\%$, and $11\% \pm 1\%$ (mean $\pm$ SEM); percent shortening was 0.22% , $6\% \pm 0.3\%$, $12\% \pm 2\%$, and $15\% \pm 2\%$; and percent original length was $113\% \pm 2\%$, $104\% \pm 1\%$, $97\% \pm 2\%$, and $95\% \pm 2\%$. Percent stretch was not significantly different between the stretch-treatment groups (Fig. 4A). Percent shortening and percent original length were significantly lower and higher, respectively, in the 50°C treatment group than in the 60°C and 70°C treatment groups, which were not different from one another (Figs. 4B, C). Percent stretch did not change during the 30-minute period between stretching and treatment. There was a good linear correspondence between percent shortening and treatment temperature ($R^2 = 0.50$; $p = .001$) (Fig. 5).

Discussion

Appropriate animal models are critical to test hypotheses related to the treatment of tendon injuries. Although mechanical overloading is a common cause of tendon damage, it has not been studied extensively. Established tendon injury models include complete or partial tendon laceration and bony avulsions, but there is a paucity of information surrounding tendon stretch injury.²⁶ Canine models have been instrumental for studies of adhesion formation, healing, and suturing techniques.^{20,21,26,27} Flexor tendons have been the focus of the vast majority of previous work. Given the high incidence and potentially debilitating outcomes of digital extensor tendon injuries, there is a need for an appropriate, reproducible model. There are limited reports of tendinous or ligamentous stretch injury models. Canine anterior cruciate ligament laxity has been caused by surgical elevation of the ligament tibial insertion,²⁸ Lapin anterior cruciate ligament subfailure injury has also been produced *in vitro* via tensile load application to the femur–anterior cruciate–tibia complex.²⁹ The model described here was

designed to evaluate the effect of MRFE on tendinous stretch injury only, so rupture and laceration injuries were not included.

The model developed for this study allows limited, controlled, repeatable, stretch injury of the DETA. The central slip, triangular ligament, and lateral bands were all elongated together to mimic a single injurious event and to limit potential variability in component elongation. The third and fourth digits are close in size and are the longest digits of the canine forelimb. They were chosen for inclusion in the study to facilitate DETA isolation and manipulation, provide enough tissue to create isolated injuries, and limit potential variability due to disparate digit lengths. The model evaluated in this study is limited by the fact that the muscle bodies of the lower forelimb were transected and the tissues evaluated were previously frozen, so it is possible that the soft-tissue behavior and histologic appearance were not analogous to the *in vivo* situation. Based on published research, however, freezing has limited effects on the viscoelastic and tensile properties of ligaments.^{30,31} In addition, all samples were treated identically for purposes of comparison between treatment groups. This study was an initial investigation to evaluate the feasibility of MRFE application to stretched tendinous tissue with an *in vitro* model. Further work is necessary before consideration of potential clinical application.

All stretch procedures were performed by a single investigator to limit variation in the force application rate. The percentage of stretch was relatively consistent between specimens, attesting to the reproducibility of the technique on the selected digits. Stretched tissues were maintained in normal saline for 30 minutes after stretching to ensure that the increase in length was not due to creep. Because the initial stretched length was maintained, tendinous tissue was stretched to the point of permanent elongation.²⁵ The acute *in situ* injuries of the model used for this study are not likely representative of chronic *in vivo* injuries characteristic of most digital injuries. Although the model was suitable for the purposes of this preliminary study evaluating the effects of MRFE on stretched digital tendinous tissue, further work is necessary to assess the persistence of stretch over time and the effects of MRFE on chronically stretched or scar tissue.

Histologic changes in both untreated and treated stretched tissue are consistent with those previously reported from soft-tissue stretch and MRFE treatment soft-tissue studies, respectively.^{18,25,32,33} Based on the results of this investigation, MRFE application at a temperature of 60°C and a power of 10 W appears to be most appropriate to return stretched tissue to approximately the prestretched length. Although there was no difference between the 60°C and 70°C treatment groups and histologic changes after MRFE treatment were similar in all treatment groups, energy application at the lowest temperature and power necessary to return tissue to the pre-stretched length is advised to limit tissue compromise and damage to surrounding structures.¹⁷ There was a good correlation between tissue shortening and treatment temperature, as expected based on previous work. The MRFE treatment temperatures and power setting applied in this study were based on established parameters and the melting temperature of collagen.^{33,35} Further study is necessary to evaluate the effect of MRFE on surrounding tissues and to optimize application to tendinous tissue.

A wide variety of opinions regarding the treatment of acute and chronic closed extensor tendon injuries exist, and there is no single optimal treatment.³⁶ It is possible that probes can be designed for minimally invasive MRFE digit applications. There is currently no information on the effect of MRFE effect on the mechanical properties of extensor tendon tissue specifically, but it is well established that treatment compromises soft-tissue mechanical properties acutely.^{17,34} Quantification of the changes in mechanical properties reduced by MRFE in tendinous tissue is required before consideration of *in vivo* application.

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Figure 1.

Isolated canine DETA between the metacarpophalangeal joint (MCPJ) and the PIPJ. Just proximal to the PIPJ, the canine DETA is composed of the central slip (CS) connected to 2 lateral bands (LB) by the triangular ligament. The central slip inserts on the second phalanx (CSI). The locations of the 2 sutures placed in the central slip for this study are indicated (X). The midpoint of the length of the central slip between the 2 sutures was identified, and the hook of the stretching device was passed from medial to lateral beneath the central slip, lateral bands, and triangular ligament of the DETA at that level (dashed rectangle). The hook was then retracted until its distal most aspect was level with the textured distal cylinder rim of the device. Firm pressure was applied to the cylinder to engage the textured surface of the rim with

surrounding tissue (circle), preventing slippage of tissues contained within the device during hook retraction.

Figure 2.
Stretched canine DETA tissue between the sutures with normal tissue just beyond the sutures.

Figure 3.

Photomicrographs of (A) control; (B) stretched-only; and (C) stretched, MRFE-treated DETA tissue. Control tissue had characteristic wavy, distinct collagen fibers and cigar-shaped fibroblast nuclei (arrow), whereas elongated tissue was characterized by diminished collagen fiber waveforms, less discernable collagen fibers, and elongated fibroblast nuclei (arrow) and stretched, MRFE-treated tissue had pyknotic fibroblast nuclei (arrow) and a complete loss of collagen fiber architecture and wave forms (hematoxylin and eosin stain; magnification, $\times 400$; bar = $200\ \mu\text{m}$),

Figure 4.

Bar charts (mean ± SEM) showing (A) percent stretch, (B) percent shortening, and (C) percent original length of stretched, MRFE-treated DETA tissue in 3 temperature treatment groups. Bars with different letters were significantly different from each other ($p < .05$).

Figure 5.
Graph of percent DETA tissue shortening versus MRFE treatment temperature with calculated least squares linear regression line.