SCIENTIFIC ARTICLE

The Effect of Core and Epitendinous Suture Modifications on Repair of Intrasynovial Flexor Tendons in an *In Vivo* Canine Model

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Purpose To determine *in vivo* effects of modifications to core and epitendinous suture techniques in a canine intrasynovial flexor tendon repair model using clinically relevant rehabilitation. Our null hypothesis was that gap formation and rupture rates would remain consistent across repair techniques.

Methods We evaluated gap formation and rupture in 75 adult mongrel dogs that underwent repair of intrasynovial flexor tendon lacerations followed by standardized postoperative therapy. The current suture technique was a 4-0, 8-strand core suture with a purchase of 1.2 cm and a 5-0, epitendinous suture repair with a 2-mm purchase length and depth. We compared gap and failure by chi-square analysis to a historical group of *in vivo* repairs (n = 76) from the same canine model using 8-strand core suture repair with purchase of 0.75 cm and 6-0 epitendinous suture with a 1-mm purchase length and depth.

Results A total of 93% of tendons (n = 70) demonstrated gapping of less than 3 mm using the current suture technique. Five percent of tendons (n = 4) had a gap of 3 mm or greater, and there was 1 repair site failure. This was significantly improved over the comparison group of historical 8-strand core repair technique, which resulted in 82% (n = 62) of repairs with a gap of less than 3 mm and 7 failures (9%).

Conclusions In an *in vivo* model, current modifications to suture techniques for intrasynovial flexor tendon repair demonstrated significant improvements in gap formation and rupture compared with a similar technique using shorter purchase lengths and shallower purchase depth.

Clinical relevance Suggested repair modifications for the treatment of zone II flexor tendon transections demonstrate improvements in gap formation and tendon rupture *in vivo*. *(J Hand Surg 2012;37A:2526–2531.* *Copyright © 2012 by the American Society for Surgery of the Hand. All rights reserved.)*

Key words Tendon, repair, intrasynovial, zone II, core suture.

Over the past 3 decades, experimental studies have demonstrated conclusively that early, protected postoperative digital motion is a critical factor in achieving adhesion-free intrasynovial tendon repair and improved functional outcomes. Tempering the enthusiasm for early motion, however, is the observation that repair site deformation occurs regularly between the tendon stumps in the first 3 to 4 weeks after

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repair, and that gap formation greater than 3 mm can prevent healing tendons from accruing tensile strength, contributing to catastrophic repair site failure.[1–4] Multistrand repairs combined with early postoperative mobilization protocols markedly improve the tensile properties of repair, with 6- and 8-strand constructs providing substantially higher values for ultimate failure load compared with 2- or 4-strand ones.[5] Increasing the caliber of core suture[6] and the depth and purchase of the epitendinous suture[7,8] has been shown to have a substantial positive effect on the tensile properties of the repair in *ex vivo* models.

Over the past 15 years, we have consistently used a clinically relevant canine *in vivo* model for flexor tendon repair research.[5,9–15] During this time, technical advances associated with greater capacity to reduce gap formation have been identified, including increasing the number of strands crossing the repair site, the length of core suture purchase, the suture caliber, and the configuration of the epitendinous suture.[15] In response to these advances, we have modified our tendon repair techniques and now have 6 years of experience with an 8-strand core suture method with a 1.2-cm core suture purchase technique and a modified 2-mm deep epitendinous suture in a series of intrasynovial flexor tendon repairs.[9–12,15] Because most prior studies demonstrating the biomechanical advantages of these modifications were performed in *ex vivo* models,[2,6,7,16–18] we recognized the need to evaluate the effect of such modifications *in vivo*. Therefore, we performed the current investigation to evaluate the gap formation and rupture rates of tendons repaired by this current suture technique in an established animal model treated with early postoperative controlled mobilization.

Our primary objective was to determine whether the implementation of modified suture repair methods had a positive effect on gap formation and repair rupture rates *in vivo*. To accomplish this aim, we compared gap formation and rupture using current repair methods with an internal historical control set of tendons from the same animal model repaired with an 8-strand core suture method that used a shallower core and epitendinous suture. Our null hypothesis was that gap formation and rupture rate would remain consistent across repair techniques.

MATERIALS AND METHODS

We examined 75 canine flexor tendons from 75 adult mongrel dogs. We repaired these tendons using our current suture technique described below. Gap formation and rupture rates of these tendons were compared with a group of internal historical controls that used shallower core and epitendinous suture techniques. We used the same postoperative rehabilitation protocol for both the current and historical control tendon repairs. The Washington University School of Medicine Animal Studies Committee approved all studies. The historical comparisons consisted of 76 tendons reported previously in studies examining the effect of postoperative mobilization protocols on tendon repair tensile strength.[13] The 75 tendons examined in this study all served as control specimens for experimental tendon repairs that investigated the effect of repair site augmentation with growth factors.[9–12] As control specimens, the tendons had no growth factor treatment.

Animal model and current surgical technique

Since 1995, we have used a canine digital flexor tendon model because of the similarity of the canine flexor tendon structures to those of humans.[8] We used adult mongrel dogs (Covance, Denver, PA) weighing 20 to 30 kg in all cases. Animals are maintained in a licensed animal care facility. For the surgical procedure, we anesthetized the animals with an initial intravenous dose of thiopental sodium (0.5 mL/kg) supplemented by intermittent injections of atropine (0.5 mL) and acepromazine (0.2 mL). We intubated them and maintained them on 1% halothane or isoflurane anesthesia. We shaved the forelimb and washed it with povidone-iodine. Then we exsanguinated the forelimb and applied a tourniquet to the forelimb.

Each forelimb (n = 75) underwent a digital flexor tendon repair in the second and fifth digits. We selected the second and fifth digits for ease and reproducibility of the surgical approach to the border digits. One repair served as the control, and the other as the experimental repair. The control repairs were the specimens reported in the present study. We chose the control tendon by consecutively alternating between the second and fifth digits to ensure an equal distribution. We approached the sheath of the second or fifth digit through a midlateral incision in the region between the annular pulleys proximal and distal to the proximal interphalangeal joint. We cut the tendon sharply in a transverse fashion using a scalpel blade at the level of the proximal interphalangeal joint. In 59 of 75 cases, because of standardization of the control tendon with the experimental tendon, we created a small defect, oriented transversely to the tendon and within the free tendon stumps, using the entire width of a 64-blade scalpel for standardization with the repair taking place in the other tendon of the same forelimb.[11] We created no trough in the other 16 tendons. We then immediately performed tendon repair using Winters

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