

An Analysis of Factors Associated With Failure of Tendon Repair in the Canine Model

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Purpose: The canine model is commonly used for flexor tendon repair research. The purpose of this study was to analyze the factors, including laceration mode (partial and complete), suture techniques, therapy methods, and weight-bearing status, associated with tendon repair rupture or gap formation in the canine model *in vivo*.

Methods: We reviewed the factors associated with repair failure among 624 flexor tendon repairs in zone II from 242 dogs reported previously from our institution, including both partial and complete lacerations.

Results: We found that weight-bearing due to failure of postoperative immobilization was the most important factor influencing tendon repair rupture or gap formation.

Conclusions: As has been noted clinically, in our canine model failure and gapping of a flexor tendon repair was primarily the result of uncontrolled loading. Rehabilitation strategies that reduce the risk of catastrophic loading of the repair are critical to reducing the experimental failure rate when using dogs for flexor tendon research. Similar strategies may also reduce such failures in humans. (*J Hand Surg* 2007;32A:518–525. Copyright © 2007 by the American Society for Surgery of the Hand.)

Key words: Flexor tendon, canine, tendon rupture, tendon repair.

Since 1941, when Mason and Allen¹ used an *in vivo* canine model to perform a flexor tendon injury and repair experimental study, the canine model has been widely accepted as an appropriate animal model for finger flexor tendon research.^{2–6} The canine flexor tendon and its pulley system are very similar to those of humans in anatomy, structure, biology, and function—especially in zone II, in where 2 flexor tendons are tightly wrapped by the flexor sheath and pulley system, leading to a high risk of adhesion formation after injury (Amadio et al, presented at the 35th Annual Meeting of the Orthopaedic Research Society, 1989).⁷

The canine digit, however, is a weight-bearing structure. Controlling weight-bearing after flexor repair is critical in the canine model to avoid gap formation or rupture. Most commonly, casts are used for this purpose^{5,8–10}; more recently radial neurectomy has been proposed as an alternative, with or without splinting.^{11–13}

Because most of the studies using dog models for flexor tendon research have focused on new repair

techniques,^{14–17} therapy comparisons,^{11,12,18–21} or other treatments to improve healing,^{22–24} the evaluations of functional outcomes and healing status have traditionally been the main outcome measures. Less attention has been paid to rupture and gap complications; some studies simply eliminate ruptured tendons from their analysis.^{25–27} We believe that this is a lost opportunity to identify factors that contribute to tendon repair rupture or gap formation. The purpose of this study was to investigate the factors associated with failure or gapping of the tendon repair in a series of studies of canine flexor tendon repair.^{12,13,21,28}

Materials and Methods

A total of 624 flexor digitorum profundus (FDP) tendon repairs in 242 dogs were performed in our laboratory between 1998 and 2004 to study the effects of postoperative therapy and repair techniques on partial and complete flexor tendon laceration. Although the results of these studies have been published,^{12,13,16,21,24,28} all repair failures were excluded

from detailed analysis; it is these failures that we report in this article.

All protocols were approved by our Institute of Animal Care and Use Committee. All dogs were adults, 1 to 2 years old, with an average weight of 22 kg (range, 19–27 kg). A total of 204 tendons from 102 dogs had a partial laceration and repair. A total of 420 tendons from 140 dogs had a complete laceration and repair. All surgical procedures were performed under general anesthesia. All tendon repairs were performed by the authors.

As noted earlier, the surgical methods are well documented elsewhere.^{12,13,16,21,24,28} In each case, the FDP tendon was approached through a lateral incision of the digit, opening the flexor sheath between the proximal and distal pulleys. The FDP tendons were sharply lacerated and repaired 5 mm distal to the distal edge of the proximal (A2 equivalent) pulley. Before tendon repair, all dogs had a radial neurectomy at the axilla to prevent active wrist and elbow extension. After surgery, the treated paw was additionally protected either by a cast, splint, or sling. All animals were observed at least twice daily, 7 days per week, during which time the cast, splint, or sling was removed for therapy. The status of the cast, splint, or sling and any evidence of weight-bearing on the surgically treated limb were recorded twice daily as well. When the dogs were killed the repaired tendons were evaluated; ruptures (complete separation of the 2 tendon ends with no intervening connection of tendon or suture) were noted and any gap formation (range, 1–4 mm) was measured with calipers. For this report, all gaps greater than or equal to 5 mm were considered to be ruptures.

Partial Laceration Model

Two hundred four FDP tendons of the second and fifth digits from 102 dogs were lacerated to 80% of their cross sections according to the method of Dobyns et al.²⁹ The FDP tendon at the proximal interphalangeal level has 2 longitudinal collagen bundles, with an intervening fibrous raphe. The 80% laceration involves cutting all of one bundle and half of the other, plus all of the raphe. One hundred thirty eight tendons were subsequently repaired with a modified Kessler (MK) technique with 5-0 suture (Ticron; Davis & Geck, Wayne, NJ), and 66 tendons were repaired with a Massachusetts General Hospital (MGH) modified Becker technique using 5-0 nylon suture. A circumferential epitenon simple running suture of 6-0 nylon was used in both types of repair. The core suture bite was 1 cm proximal and 1 cm

distal to the laceration site in each case. After tendon repair, the skin was closed in layers without flexor sheath closure.

The dogs were evenly divided into groups with different therapies. One group had a more traditional canine therapy, which was performed with passive digit joint (metacarpophalangeal, proximal interphalangeal, distal interphalangeal) flexion/extension with the wrist maintained in flexion. The other group was treated with synergistic wrist motion—that is, wrist extension with digit flexion and wrist flexion with digit extension.³⁰ The dogs were killed at 1, 3, and 6 weeks, and the repaired tendons were evaluated for rupture and gap formation.

Full Laceration Model

A total of 420 FDP tendons from the second, third, fourth, and fifth digits from 140 dogs were completely lacerated and then repaired with an MK technique using 4-0 looped suture (Supramid; S. Jackson, Inc., Alexandria, VA). A simple running suture (6-0 nylon; Ethicon, Inc., Somerville, NJ) was used in the epitenon. The core suture bite was 1 cm proximal and 1 cm distal to the laceration site in each case. Dogs were killed at various time points depending on the specific protocol. The dogs killed on days 1, 3, 5, and 7 were immobilized after surgery in a nonremovable cast with elbow, wrist, and digit flexion. These dogs had no postoperative therapy. Other dogs were immobilized with a removable cast, splint, or sling to perform postoperative therapy, which began on postoperative day 5. Therapy was performed twice daily with passive motion of the elbow, wrist, and digit joints according to the various protocols, as reported previously.

Statistical Analysis

In the partial laceration group, the dataset included 102 dogs with 204 tendon repairs. In the full laceration group, the dataset included 140 dogs with 420 tendon repairs. The risk factors for rupture or gap that were analyzed included unplanned postoperative weight-bearing, timing of mobilization, time of death, laceration mode (full, partial), suture type (MK vs MGH in partial laceration model only), type of immobilization (removable, nonremovable), and type of therapy. We used statistical software (SAS/STAT GENMOD Procedure; SAS Institute Inc., Cary, NC) and generalized estimating equations (GEE) modeling to analyze the data. The dog was identified as the repeat subject. The *z* statistic was used to calculate the *p* value for each factor. We started with a univariate analysis using software

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