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Perspective Article

Recent progress in intrasynovial flexor tendon repair and rehabilitation

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Significant advances in the understanding of intrasynovial flexor tendon repair and rehabilitation have been made since the early 1970s[1,2] when reports first demonstrated that flexor tendon lacerations within the fibroosseous digital sheath could be repaired primarily and rehabilitated successfully without tendon excision and delayed grafting[3]. The concept of adhesion-free, or intrinsic tendon healing – that tendons could heal primarily without the ingrowth of fibrous adhesions from the surrounding sheath has been validated both experimentally and clinically in studies over the past 20 years[4-13]. Recent attempts to understand and improve the results of intrasynovial flexor tendon repair have focused upon restoration of the gliding surface[11,14-23], repair site biomechanics[24-37] and on the molecular biology of early tendon healing[38-47]. The goals of the surgical treatment of intrasynovial flexor tendon lacerations remain unchanged, however: to achieve a primary tendon repair of sufficient tensile strength to allow application of a post-operative passive motion rehabilitation protocol such that formation of intrasynovial adhesions is inhibited, restoration of the gliding surface is stimulated, and healing of the repair site is facilitated[48]. I will focus upon two studies published recently from our tendon group at Washington University in St Louis that have evaluated intrasynovial repair site excursion and proximal musculotendinous load as independent variables, with the objective of determining whether or not rehabilitation programs that emphasize active motion are biologically advantageous to the healing tendon.

In an effort to improve the results of intrasynovial flexor tendon repair, authors have advocated rehabilitation methods that generate increased levels of applied *in vivo* force and tendon excursion[2,5,49,50]. The clinical success of early passive motion rehabilitation has encouraged surgeons to prescribe rehabilitation protocols that further increase the "motion stress" on the repair site in order to stimulate healing[48]. However, there are no scientific data to support the concept that more aggressive rehabilitation accelerates tendon healing.

To address the issue of rehabilitation, we quantified, in an *in vivo* canine study, the flexor digitorum profundus (FDP) tendon force and excursion produced by five clinically relevant passive motion protocols[51,52]. Force was measured using a Z-transducer placed on the FDP tendon and excursion was assessed using a video technique. The combined excursion and force data indicate three distinct mechanical paradigms ($p < 0.05$). First, with the wrist flexed and either one or four digits flexed and extended (1F, 4F), low excursion (1.7 mm) and low force (5 N) were produced. Second, with synergistic flexion of the digits as the wrist was extended (SYN), high excursion (3.5 mm) but low force were produced. Third, with the wrist and digits flexed and extended simultaneously (1E, 4E), high excursion and high force (17 N) were produced. These data described, for the first time, the combined excursion and force variables that operate on the canine FDP tendon during joint manipulations and allowed a set of experiments to be conducted in which excursion and force were varied independently of each other.

We first examined the effects of increased *in vivo* tendon excursion on digital range of motion and tendon strength following FDP tendon transection and repair[53]. Ninety-six FDP tendons from 48 dogs were injured and repaired. The affected forelimbs were treated by passive mobilization during two five-minute rehabilitation sessions, performed five days a week starting on the first post-operative day. Rehabilitation for one group (4F; low-force/low-excursion) was performed after removing the volar flexion block and consisted of passive flexion and extension of the four digits with the wrist maintained in the flexed position. For the second group (4E, low-force/high-excursion) both the volar flexion and dorsal extension blocks were removed and the wrist and digits flexed and extended simultaneously. Both protocols were

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performed at a rate of approximately 1 Hz, resulting in 600 cycles of loading each day. Dogs received rehabilitation daily until sacrifice at 10, 21 or 42 days.

Our results indicated that the use of rehabilitation that produced increased tendon excursion did not influence range-of-motion or tensile properties. Joint rotation and tendon excursion in digits from the low-force/low-excursion and low-force/high-excursion groups were not significantly different ($p > 0.05$), with both groups not different from unoperated controls. Tensile structural properties (ultimate force, rigidity, strain at 20 N, strain at failure) were not significantly affected by increased excursion ($p > 0.05$). We conclude that a threshold of 1.7 mm of tendon excursion is sufficient to inhibit adhesion formation and to allow excellent recovery of functional properties following sharp transection of the canine FDP tendon. Additional excursion, at the same low force level, provides little added benefit.

The objective of our next study was to assess the effects of variations in applied *in vivo* force on biomechanical properties. We compared low-force/high-excursion versus high-force/high-excursion rehabilitation, using 246 tendons from 123 dogs[25]. Because we hypothesized that there might be an interaction between the stiffness of the repair and the level of applied rehabilitation force, we used both 4- and 8-strand repairs in this study.

Our results indicated that tensile properties were not different between low- and high-force rehabilitation groups, regardless of repair technique. Rehabilitation method did not significantly affect ultimate force ($p=0.48$), repair-site rigidity ($p=0.96$), strain at 20 N ($p=0.29$) or strain at failure ($p=0.22$). For example, in the 42-day, 4-strand group, tendons treated with high-force rehabilitation had an average ultimate force of 96 N, which was not significantly different from the average of 102 N for the low-force rehabilitation group. Moreover, method of rehabilitation had no significant effect on distal or proximal interphalangeal joint flexion (distal, $p = 0.91$; proximal, $p = 0.87$) and only a slight effect on tendon displacement (9% less in the high-force group; $p = 0.024$). Operated digits had approximately the same range of motion as contralateral control digits, with proximal interphalangeal joint flexion ($p = 0.20$) and tendon displacement ($p = 0.58$) not significantly different and distal interphalangeal joint flexion reduced by only 11% compared to control ($p < 0.001$).

Based on these results, we conclude that increasing the level of force within the range that can be applied using passive mobilization (i.e., 5 to 17 N), does not accelerate the time-dependent accrual of stiffness and strength in the canine model. Taken together with the previous finding that increased tendon excursion did not enhance tendon healing[53], our findings suggest that there be a re-examination of the widely held concept that increases in force and motion produced by more vigorous mobilization protocols are beneficial to tendon healing. While more vigorous rehabilitation may help improve overall hand function, we found no evidence that it enhances tissue healing and strength in the context of a modern suture repair.

Several conclusions can be drawn from our recent findings using the clean laceration canine model with multistrand suture repair and early passive motion rehabilitation. First, suture technique is of primary importance in providing a stiff and strong repair throughout the early healing interval, and the benefits of a multistrand repair are observed regardless of the level of rehabilitation force. Second, increased tendon excursion beyond the amount produced by passive digital flexion-extension (with the wrist flexed) does not enhance digital motion or tendon healing. Third, application of increased levels of passive force during the early post-operative period is not supported as a means to accelerate tendon healing, since the time-dependent accrual of stiffness and strength does not appear to be enhanced by increased force levels within a clinically relevant range. In order to increase the sensitivity of the repair site to increased force levels, it may be necessary to alter the biochemical environment to increase expression of integrins or other force-sensitive molecules. Future strategies to accelerate tissue healing may therefore require manipulation of both biochemical and rehabilitation variables.

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