**In Vivo Response of a Nitinol Based Flexor Tendon Repair Device in a Rabbit Model**

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**Introduction:** The digital flexor tendons of the hand, including the flexor digitorum profundus (FDP), are responsible for enabling finger flexion. Injury involving a laceration to the flexor tendons are common and associated with a high incidence of morbidity [1]. The state of the art for flexor tendon repair is the use of two or more core sutures in combination with an epitendinous circumferential suture. There are inherent limitations to suture based methods, including a high level of skill required to perform the repair, increased surgical time and the tendency for sutures to strangulate the tissue [2]. Current techniques often result in sub-optimal clinical outcomes, with reported failure rates ranging from 4%-10% [3]. To address these limitations, a nitinol, non-suture based repair device has been developed. The objective of this study was to determine the primary repair strength and in vivo behavior of the device, relative to a suture control, in a rabbit model at a five week time point.

**Methods:** The device consists of two nitinol sleeves attached at the center with fine gauge nitinol wire (Fig 1). Each sleeve has a lattice structure and numerous micro tines that grip the tissue. To manufacture the device, a lattice pattern with tines at the vertices was laser cut into a 150 µm thick superelastic nitinol tube (OD=4 mm). The laser cut tube was heat set at 515°C into a three dimensional shape with an elliptical cross-section and inward facing tines and chemically etched and electro polished. The device was assembled and pre-stretched onto a hollow applicator made from tubular stainless steel. To use the device, the tendon stumps were pulled into the applicator using suture loops, the applicators were removed and the device contracted over the tendon stumps. The two ends were then pulled together by tightening the connecting wires and secured using crimped stainless steel swages, with excess wire being trimmed. To determine the in vivo behavior of the device, a rabbit model was utilized. The study consisted of a simulated laceration and surgical repair of the FDP tendon of 18 rabbit hind limbs. These were divided into non-surgical and surgical groups. In the non-surgical group, six repairs were made on excised rabbit flexor tendons (3 device and 3 suture) and then subjected to biomechanical testing. This yielded the primary repair strength. The surgical group consisted of two cohorts of six. For each cohort of six, a surgical procedure was performed in which a simulated laceration was repaired either with suture (N=3, each cohort) or the nitinol repair device (N=3, each cohort) and allowed to heal for five weeks. The surgeries of the first and second cohort were staggered by three weeks as a safeguard against unforeseen complications. All animals were sacrificed at five weeks and the FDP tendons were excised. Within the surgical group, tendons from the first cohort were subjected to biomechanical testing, while tendons from the second cohort were subjected to histological analysis. For the surgical procedure, animals were anesthetized and a posterolateral skin incision was made over the right FDP tendon and the flexor sheath was opened in a Z fashion. In the suture group, the tendon was cut 100% and repaired with a Kessler two-strand suture repair using 4.0 Ethibond. In the device group, the tendon was cut 100% and then reattached with a sterile nitinol repair device. In all groups, the tendon sheath was repaired and the skin was re-approximated with a running sub-q nylon suture. A splint was applied to the hind limb and e-collars (plastic neck collars) were used to minimize chewing at the repair site. For biomechanical testing, the ends of the tendon were gripped using freeze clamps and subjected to a constant strain rate uniaxial tensile test (0.5%/s) until failure using a custom tensile testing apparatus consisting of a stepper motor, a lead screw and a 50 lb. load cell. Data acquisition was controlled using LabVIEW. The samples were kept moist with saline during testing. Tensile testing was performed on the non-surgical group and the first cohort of the surgical group. Histology was performed on the second cohort of the surgical group. Excised tendons were fixed in formaldehyde and then embedded in hard resin [4]. The specimens were cut longitudinally with a diamond point saw into 2 mm thick sections. From each large section, a sub-section was ground to a 30 µm thickness. Each section was stained with haematoxylin-eosin (HE) and observed under a transmission light microscope. Histology was performed on the second cohort of the surgical group.

**Results:** In the non-surgical group, the primary repair strength of the device repair (37.1 ±0.4 N) exceeded that of the suture repair (22.1±4.1 N). This difference was statistically significant (two sided t-test, p<0.05) (Fig 2). Biomechanical testing of the first cohort of the surgical group revealed that at five weeks, the strength of the device repaired tendon (86.5±28.7 N) and the suture repaired tendon (99.7±42.4 N) both exceeded the primary repair strengths of the non-surgical group, but were less than the undamaged contralateral tendon (252.8±33.8 N) (Fig 2). The difference between the strength of the device and suture repairs at five weeks was not significant. Observations during the necropsy of the first cohort within the surgical group revealed the presence of adhesions in all tendons, which was a result of the immobilization from the splinting. Necropsy of the second cohort revealed that all repairs within this group had failed. Animal facility records indicated that during an early bandage change the splints were not properly replaced, resulting in ruptures of the repairs. Failure was manifested as suture pullout for the suture repairs and fracturing of the center connecting wires in the device repair. The device was still in place on the separated tendon...
stumps, thus histology was still performed. HE stained sections at 2X (Fig 3), 10X and 40X (not shown) revealed that at five weeks, no appreciable tissue necrosis was observed either in the device or suture groups. No inflammatory cells were observed in either group. In all tendons repaired with the device, fibroblasts were observed to be proliferating on the surface of the nitinol, which was encapsulated in collagen.

**Discussion:** In a rabbit model, the primary strength of the nitinol repair device exceeded that of a two strand Kessler repair. This suggests that the repair device may provide the necessary primary strength to enable early mobilization. In the first cohort of the surgical group, the nitinol repair device was found to facilitate the repair of a full FDP laceration, with a five week repair strength that was over double that of the primary repair strength and similar to that of the suture control. Histology revealed that the device did not elicit a sustained inflammatory or foreign body response and facilitated the growth of fibroblasts on the device surface. This data suggests the feasibility of utilizing the nitinol repair device for the fixation of flexor tendon lacerations. However, the small sample size, the failure of all repairs in the second cohort and the single time point of five weeks severely limits the conclusions of this study. Considerable future work will need to be conducted, including fatigue testing, work of flexion testing and further animal testing with both early and late time points. Additionally, an animal model that displays a lessened tendency for adhesion formation (e.g. Canine) may be necessary in order to further investigate the formation of adhesions [6].

**Significance:** The current state of the art for flexor tendon repair is the use of sutures. The purpose of this study was to investigate a novel device that may offer an alternative to sutures for the repair of flexor tendon lacerations.

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