IDENTIFYING THE MECHANICAL DAMAGE LIMIT OF THE RABBIT SUBSYNOVIAL CONNECTIVE TISSUE

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ACKNOWLEDGEMENTS:
This study was supported by grants from NIH (NIAMS AR49823) and Mayo Foundation.

INTRODUCTION:
Carpal tunnel syndrome (CTS), a compression neuropathy of the median nerve, is the most common peripheral neuropathy. While the direct cause of CTS is unknown in most cases, one hypothesis is that microtears in the sub synovial connective tissue (SSCT) surrounding the tendon and the median nerve in the carpal tunnel initiate fibrosis of the SSCT and thereby create CTS.

The SSCT is composed of layered bundles of collagen running parallel to the tendon. These layers are interconnected by smaller vertical fibers. A hypothesis of SSCT function is that during finger movement the loose fibers between adjacent layers are stretched and the fibrous bundles move layer by layer, pulled by the interconnections, more or less as an arm would move within layers of sleeves. At some level of strain, these interconnections will rupture.

Rabbit SSCT is organized within the carpal canal very similar to humans, and has been used as an experimental model of CTS. In order to test the hypothesis that microtears can occur in the SSCT beyond a strain threshold in this model, we analyzed the mechanical response of the rabbit SSCT subjected to various levels of displacement comparing the data acquired from two repeated displacement-relaxation tests. An interval for viscoelastic recovery was allowed between tests to identify permanent damage.

MATERIALS AND METHODS:
Specimen Preparation and Setup: We used 24 forepaws obtained at necropsy from rabbits (body weight 3.89±0.47 kg) sacrificed for other, IACUC approved, studies. All paws were cut at the mid-forearm. The flexor digitorum superficialis (FDS) tendons were exposed at the antebrachial level with the carpal tunnel intact. While all digits were held in full extension, the third FDS tendon was transected 5 mm proximal to the proximal edge of the carpal tunnel and was used for testing. The excursion of the third FDS tendon from full middle finger extension to full flexion was measured using a digital caliper. The third FDS tendon was also exposed distal to the carpal tunnel and cut at the level of the A1 pulley, and the both ends of the tendon were sutured with a single suture of 6-0 polypropylene (Prolene, Ethicon, Somerville, NJ). Then, the whole specimen was mounted on a custom specimen holder on a mechanical actuator. The other digits were pinned to the holder in the fully extended position. The proximal part of the other FDS tendons and the FDP muscle were also pinned to the holder to maintain their position while testing. The wrist joint was fixed in the neutral position using a 1.0 mm Kirschner wire. The proximal end of the third FDS tendon was connected to a 500-g load cell (Transducer Techniques, Temecula CA). After pre-tensioning, the distal end was connected to a 0.4 N weight to maintain tension in the tendon during testing. Motion of the actuator with the specimen mounted generated relative motion between the tendon and carpal tunnel. The specimen was maintained at room temperature (20 ºC) and kept moist by a continuous saline drip for the duration of all testing.

First Relaxation Test: The tendon was moved by the actuator to simulate a flexion motion at a speed of 0.5 mm/s. The tendons were moved to excursions of 2 mm, 3.5 mm, 5 mm, or 8 mm. Six randomly chosen specimens were tested at each excursion level. The readings from the load transducer and the corresponding excursions were recorded with a data acquisition system at a sample rate of 10 Hz to obtain the following values:
- Peak Force (F): The highest force observed while testing.
- Stiffness (S): Slope of linear region of the force-displacement curve.
- Decay time (Tp): The point in time when the decline in force across a 30 minute time interval was less than 1% of the peak force.
- Plateau force (Fp): The force at the decay time.

The first relaxation test was stopped when the decay time limit was achieved, as measured by a simultaneous analysis using a MATLAB (Mathworks, Natick MA) search algorithm. After the first relaxation test, the tendon was moved in the extension direction to the original position and maintained in that position for an additional amount of time equal to the measured decay time.

Second Relaxation Test: After recovery from the first test, the tendon was moved in the flexion direction again for the same excursion distance as the first test. The readings from the load transducer and the corresponding excursions were recorded in the same manner.

Statistical Considerations: Allowing sufficient relaxation time for viscoelastic recovery, parameter differences between the first and second tests indicate permanent damage (Figure 1). The ratio of “F2 to F1”, “S2 to S1”, “F2p to F1”, and “T2p to T1p” in each group were compared using one-way ANOVA with a Tukey-Kramer post-hoc test for the difference among the different excursion groups. All statistical tests were two-sided and p-values less than 0.05 were considered significant.

DISCUSSION:
We found that 3.5 mm of displacement, motion within the physiological range, causes an irreversible change in the strength (decreased “F2 to F1” ratio) of the rabbit SSCT. Interestingly, with further displacement these ratios decreased by similar proportions, which is consistent with the hypothesis that the SSCT functions as a series of interconnected layers, which fail sequentially by microtears. Future studies might look at the effect of relaxation time, cycle time, strain rate, and other parameters on these properties, and the propensity of the SSCT to injury, both in vitro and in vivo.

SIGNIFICANCE:
Our findings support the hypothesis that SSCT damage from everyday activity can occur. Such damage may be a factor in the etiology of CTS.

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