INTRODUCTION: Recent in vivo studies of flexor tendon midsubstance healing have indicated that during the first 10 days after injury and repair there is no increase in ultimate tensile force [1]. In contrast, there is an increase of approximately 170% in the rigidity (stiffness) of the repair site and a decrease of more than 45% in the repair-site strain at 20 N force. The basis for the increase in rigidity during the early stages of tendon healing is not known, but may be due to either biological or mechanical factors. In particular, cyclic loading of the tendon repair site during post-operative rehabilitation may have a mechanical conditioning effect that increases the stiffness of the repair site independent of repair-site healing. Our objective was to determine whether or not cyclic loading of repaired flexor tendons causes an increase in repair-site rigidity and whether or not this increase depends on the level of applied force. We simulated 10 days of passive motion rehabilitation by applying 6000 loading cycles to repaired flexor tendons ex vivo at force levels generated during passive motion rehabilitation. We then evaluated the tensile mechanical properties. We hypothesized that cyclic loading causes an increase in repair-site rigidity and a decrease in repair-site strain at 20 N.

MATERIALS AND METHODS: Forty-eight flexor digitorum profundus (FDP) tendons with attached distal phalanges were obtained from twelve hindlimbs of six adult mongrel dogs. All procedures were approved by our institutional animal studies committee. Specimens were randomly assigned to one of three groups: control (no loading), low-force (5 N) cyclic loading or high force (17 N) cyclic loading. Tendons were transected in Zone II, 4 cm proximal to the insertion site. They were repaired using a four-strand modified Kessler technique with double-stranded 4-0 Prolanid suture (S. Jackson) supplemented with a running peripheral suture of 6-0 Prolene (Ethicon). For cyclic loading, the distal phalanx and proximal tendon stump were placed in custom clamps attached to a materials testing machine (Instron 8500R). The tendon was loosely wrapped in plastic and kept moist throughout the testing period using phosphate buffered saline. Tendons in the low-force and high-force groups were loaded for 6000 cycles at 2 Hz from 0.5 N to peak forces of 5 and 17 N, respectively. The force levels were chosen to match the peak tendon forces that were measured in vivo for low- and high-force passive motion rehabilitation protocols of the canine forelimb [2]; 6000 cycles were used to simulate the number of cycles applied during a 10-day period of rehabilitation (600 cycles/day) [3]. Specimens in the control group were sham loaded for an equivalent period of time (50 minutes). After cyclic loading, specimens were allowed to recover for 1 hour and then tested to failure in tension. Repair-site strain was determined during tensile testing by tracking the position of two reflective markers placed an initial distance of 1.5 cm apart, centered about the repair site. From plots of force versus repair-site strain we determined ultimate (maximum) force (N), repair-site rigidity (N/(mm/mm)), repair-site strain (%) at 20 N force and repair-site strain (%) at failure [3]. One way analysis of variance was used to determine the effect of loading on tensile properties.

RESULTS: Specimens subjected to high-force cyclic loading had significantly increased rigidity compared to specimens in the low-force and control groups (p < 0.001; Figure). Rigidity in the high-force group was increased by 100% compared to control. Similarly, repair-site strain at 20 N was decreased by 50% in the high-force group compared to control group (p < 0.001; Table). Ultimate force was increased in the high force group compared to control (p = 0.02; Table), but the magnitude of the increase was only 13%. There were no significant differences between the low-force and control groups (p > 0.05).

DISCUSSION: Our objective was to determine whether or not ex vivo cyclic loading of repaired flexor tendons causes an increase in repair-site rigidity and a decrease in repair-site strain similar to those observed previously after 10 days of in vivo rehabilitation in a canine model [1]. Based on our findings, we conclude: 1) Repair-site rigidity is increased by 100% and repair-site strain decreased by 50% following 6000 cycles of high-force (17 N) loading. In contrast, ultimate force is changed only slightly (13%) by high-force loading. 2) Mechanical conditioning of the repair site by repetitive forces applied during rehabilitation may have a consequence that has not been noted previously, i.e. it leads to increases in rigidity and decreases in strain. This effect may explain in part the changes in tensile properties observed after only 10 days of healing in vivo. 3) The lack of effect of low-force (5 N) loading on repair-site rigidity and strain is in contrast to the previous in vivo findings in which rigidity increased in the low-force rehabilitation group. This discrepancy suggests that the observed in vivo changes can not be explained entirely by mechanical conditioning effects. 4) Therefore, both biological and mechanical factors are likely to play an important role in the rapid changes in repair-site mechanical properties that occur in vivo following flexor tendon injury and repair.


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