Behavior of chronically retracted tendon after single stage or staged repair with continuous musculotendinous re-lengthening: An experimental study

INTRODUCTION:
Repair of the retracted musculotendinous unit after rotator cuff tendon tear is limited by musculotendinous retraction and is not always possible. Healing after repair is not always achieved and usually limited by the quality of the deteriorated musculotendinous unit[1]. We have shown that continuous musculotendinous traction can restore original muscle architecture[2]. However, the changes in chronically retracted tendons undergoing single stage repair or repair after continuous traction are unknown. The purpose of this study was to investigate the quantitative and qualitative alterations of tendons which underwent chronic retraction followed by either single stage or staged repair using continuous musculotendinous traction as preparation of repair.

METHODS:
The infraspinatus tendon of 17 sheep was released and allowed to retract while protected against spontaneous healing by encasement with a silicone tube, for 10 months (group I n=5) and 4 months (group II, n=8, group III, n=4). In group I direct repair was performed after 10 months. In group II repair was performed successfully after the retracted musculotendinous unit had been progressively re-lengthened to its original length. Animals for which the re-lengthening failed and tendon retracted were embedded into a group III. Tendon structure was assessed at multiple levels of hierarchy, by MRI, histology and electron microscopy. Data were statistically analyzed using Student’s paired t-test for intra-group comparison and Spearman-correlation at a significance level of p<0.05. Values denoted in percentage (%) have been referred to the corresponding contralateral shoulder.

RESULTS:
Compared to normal controls, tendon thickness decreased in group I to 65%±22% (p<0.05) and normal thickness was seen in group II (116%±25% p=0.118). Tendons in group III showed increased thickness of 129%±11% (p<0.05), but with substantial shortness. Thickness was positively correlated with the muscular pennation angle in all groups. Enhanced collagen fiber crimping (Figure 1) and disorganization (Figure II) was found in groups I and III, whereas in group II the changes were partially restored; advanced alignment of fibrils, tendency toward a bimodal distribution of fibril diameter (Figure 2) and a decrease of the enhanced collagen fiber crimping was found in continuously re-lengthened tendons.

DISCUSSION:
The retraction and degeneration of the musculotendinous unit represents the limiting factor for the repair of tendon tears, an unsolved and common clinical problem. Nonetheless, morphological changes of chronically retracted tendons and their adaptations to direct or staged repair have hitherto not been investigated to our knowledge. We hypothesized that besides the muscle, which has been the focus of most related research to date, the tendon itself may contribute to the retraction and degeneration process.

Following direct or staged repair (after continuous re-lengthening), there were significant and important differences between the groups. While those animals with direct or failed repair (groups I and III) did show signs of severe tendon degeneration (decreased total tendon length/volume, decreased tendon thickness (group I), enhanced crimping pattern, and atrophy and disorganization of the collagen fibrils), tendons of group II with staged and successful repair after musculotendinous re-lengthening to the original length did in great part recover from degeneration.

There are limitations to the interpretation of our results. Tendons in group I had longer time for retraction. However, we were able to show previously, that there is no significant further retraction after 16 weeks [3], where spontaneous scar formation mimics a surgical repair in situ.

Torn/released tendons, from its intramuscular origin to the bony insertion, should be considered as a tissue that changes considerably during retraction, and retracts even more than the muscle[4]. It appears that the contribution of the central tendon atrophy to difficulties in surgical repair of chronically retracted musculotendinous units such as the rotator cuff or Achilles tendon have been largely underestimated and will need further investigation.

REFERENCES: