ADAPTATION OF THE MYOTENDINOUS JUNCTION TO TENDON RETRACTION. AN EXPERIMENTAL STUDY IN SHEEP.

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Introduction: After rotator cuff tendon tear, the corresponding musculotendinous unit may retract massively, accompanied by severe fatty infiltration of the muscle tissue. The retraction of the tendon and muscle is the main reason for the irreparability of long-standing rotator cuff tendon tears. The amount of retraction of the musculotendinous unit is usually estimated by the size of the gap between tendon and bone, but the contribution of muscle and tendon to this retraction has hitherto not been analyzed experimentally. It was the goal of this study to investigate the adaptation of the myotendinous unit architecture to tendon release in an animal model for chronic rotator cuff tendon tears.

Methods: For all experiments, approval of the institutional review board has previously been obtained. In 6 sheep, the infraspinatus tendon was released with a portion of the greater tuberosity and repaired after 40 weeks. The bone chip at the tendon end allowed for accurate identification of the original tendon end in CT and during operation. Structural muscle changes, retraction of the tendon end and of the myotendinous unit were monitored by CT, electron- and light-microscopy at 0, 16, 40, 46 52 and 75 weeks. The sheep were sacrificed after 75 weeks and pennation angle and mean muscle fiber length were assessed in MRI and by dissection. The distribution pattern of the intramuscular fat was analyzed after sacrifice.

Results: Within 40 weeks after release, tendon retraction of 4.8cm and marked fatty muscle infiltration had developed. Unexpectedly, tendon retraction exceeded retraction of the muscle by 2.7cm at that time-point. Histologically, disorganization of the muscle fibrils had occurred within intact muscle fibers. Tendon repair to the humeral head was only possible by bridging the existing gap with sutures, leaving a gap of 4.2cm. 35 weeks after repair, at sacrifice, tendon-like scar tissue had developed along the sutures, bridging the gap entirely. The muscle was only retracted by 1.7cm compared to the control side and inserting distally on the newly formed tendon tissue, while the original tendon end remained retracted by 4.2cm as after tendon repair (p<0.001). During the entire experiment, the muscle pennation angle increased from 22° (control side) to 38° (experimental side), while the mean fiber length decreased by 50% (3cm to 1.5cm), p< 0.001 for both values. The interfibrous fat tissue showed a planar distribution, forming small septa along the muscle fibers, oriented perpendicularly to the main line of muscle action.

Discussion: In this animal model, tendon tissue showed strong tendency to spontaneous regeneration despite massive retraction. The myotendinous junction compensated for the tendon retraction in an unexpected and hitherto unknown fashion by adapting its architecture. Thereby, the original muscle architecture was partly restored, while the original tendon end remained irreversibly retracted into the muscle. Without bone chip on the tendon end, these alterations would have been impossible to observe. It therefore seems, that from apparent tendon retraction, it may not be concluded to the retraction of the corresponding muscle. In the future, alternative procedures other than vigorous mobilization of the musculotendinous unit may have to be evaluated experimentally to improve rotator cuff tendon repair. A geometrical explanation for fatty muscle infiltration has been found, according to the fat distribution. The pennation angle increase (+72%) with shortening of the mean fiber length (-50%) results in interfibrous cavities in a planar distribution, oriented perpendicularly to the line of muscle action. The function of the interfibrous fat septa in fatty infiltrated retracted muscle may therefore be best explained as void filling material.