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We released the infraspinatus tendons of six sheep, allowed retraction of the musculotendinous unit over a period of 40 weeks and then performed a repair. We studied retraction of the musculotendinous unit 35 weeks later using CT, MRI and macroscopic dissection.

The tendon was retracted by a mean of 4.7 cm (3.8 to 5.1) 40 weeks after release and remained at a mean of 4.2 cm (3.3 to 4.7) 35 weeks after the repair. Retraction of the muscle was only a mean of 2.7 cm (2.0 to 3.3) and 1.7 cm (1.1 to 2.2) respectively at these two points. Thus, the musculotendinous junction had shifted distally by a mean of 2.5 cm (2.0 to 2.8) relative to the tendon. Sheep muscle showed an ability to compensate for approximately 60% of the tendon retraction in a hitherto unknown fashion. Such retraction may not be a quantitatively reliable indicator of retraction of the muscle and may overestimate the need for elongation of the musculotendinous unit during repair.

Repair of long-standing tendon ruptures with advanced musculotendinous retraction, as is commonly seen after ruptures of the tendons of the rotator cuff, can be difficult owing to decreased elasticity of the muscle and severe shortening of the musculotendinous unit. In current clinical practice, a tear of the rotator cuff may be repaired by direct reinsertion of the stump of the tendon into the head of the humerus. If this is not feasible, reconstructive procedures such as tendon interposition, tendon transfer, or mobilisation and shift of the entire rotator muscle may be necessary.

In a previous study by Gerber et al, the infraspinatus muscle of the sheep was shown to undergo fatty infiltration to stages 2 to 3, according to Goutallier et al, within 40 weeks after experimental release of the tendon. This was associated with substantial, irreversible architectural changes in the muscle, such as an increase in the pennation angle and shortening of the muscle fibres.

The purpose of our study was to analyse the retraction of both the muscle and the tendon separately in order to determine whether the musculotendinous unit has the potential to undergo internal remodelling during musculotendinous retraction.

Materials and Methods

The present study is part of an experiment in which six healthy female Swiss Alpine sheep were used. The mean age of the sheep at the beginning of the study was 3.75 years (3 to 5). The mean weight was 48.4 kg (45 to 54) initially and 53.7 kg (42 to 64) at death. All experiments were carried out according to the local laws of animal welfare and use of animals for experiments and approved by the Investigative Review Board (permission number ZH 160/99). The key steps of the basic experiment have already been published.

Computed tomography. Computed tomography (CT) (Siemens Somatom AR; Siemens Medical Solutions, Erlangen, Germany) was performed under general anaesthesia at intervals of 0, 16, 40, 46, 52 and 75 weeks to obtain transverse sections through the scapula at the level of the infraspinatus. Positioning of the animals for CT or MRI was carefully controlled in the scout view and corrected if necessary with a tolerance of approximately 5° from the desired position. All sections were orientated parallel to the spine of the scapula with the most central slice through the glenoid and head of the humerus used for the measurements.

For the purpose of this study, retraction of the osteotomised bone chip, representing tendon retraction (Rtendon), and retraction of the most distal part of the infraspinatus muscle, medial and lateral to the central tendon plate, representing the retraction of the musculotendinous junction (Rmuscle), were measured on the CT images (Fig. 1).
All measurements on CT and MRI were performed using a consensus readout of two observers (DCM, GL), who were blinded to the animal number or time point of measurement, using the analysis software of the CT unit.\(^8,^{10}\)

**Tendon release and repair.** Release of the tendon was performed by osteotomy of the greater tuberosity, leaving a bone chip approximately 1 cm long and 0.5 cm thick on the tendon;\(^8\) 40 weeks after release, the retracted tendon was surgically repaired using two sutures under moderate tension. Owing to tissue retraction a gap remained between the end of the tendon and the greater tuberosity.\(^8\) The integrity of the sutures and the repair were monitored during the first two weeks using temporarily implanted sensors.\(^11\)

**Post mortem.** After 75 weeks following the initial tendon release and 35 weeks after repair of the tendon, the sheep were killed and both shoulders (operated and control side) were harvested. MRI was performed on both shoulders, including the tendon and bony insertion, were decalcified using 5% potassium nitrate acid for ten days and embedded in paraffin blocks measuring 5 mm x 10 mm x 15 mm. Longitudinal slices 5 \(\mu\)m to 7 \(\mu\)m thick were cut through the centre of the myotendinous junction and stained with haematoxylin and eosin. As with macroscopic assessment, the position of the muscle insertion relative to tendon and bone chip was identified in the histological sections.

**Statistical analysis.** The differences between the time-points of relative muscle shift on the central tendon and between the pennation angles of the operated and control sides were compared using two-tailed paired \(t\)-tests, with a level of significance defined as \(p < 0.01\).

**Results**

**Gross observations**

**Tendon repair, 40 weeks after release** (Fig. 2). At the time of repair of the tendon, the gap between the humeral head and the end of the retracted tendon was filled with scar tissue. The silicone tendon cover was entirely surrounded by a thin tube of scar, allowing for some mechanical connection between the end of the tendon and the head of the humerus (Fig. 2c). Repair (Fig. 2e) resulted in a mean shortening of the gap between the head of the humerus and the end of the tendon of 0.60 cm (0 to 1.2).

**At death 35 weeks after repair.** Dissection revealed that the gap between the end of the tendon and the head of the humerus was again filled with newly-formed tendinous scar tissue (Fig. 2f). The sutures bridging the head and the tendon were embedded in a neo-tendon.\(^8,^{10}\) Unexpectedly, the musculotendinous junction was far less retracted than the end of the tendon (Figs 2f and 3). The muscle was retracted a mean of 1.7 cm (1.0 to 2.3) on the lateral side and 1.8 cm (1.1 to 2.3) on the medial. In all sheep the retracted original tendon end, including the bone chip, was found to be partly covered by infraspinatus muscle fibres. The mean length of the entire muscle of the control side was 18.2 cm (15.5 to 20.0), whereas the released musculotendinous unit was shortened by the amount of retraction of the tendon (Fig. 3).

**Retraction of muscle and tendon over 75 weeks**

**Findings on CT and MRI** (Figs 4 and 5). Tendon. One hour after release of the tendon the retraction distance measured a mean of 3 cm (2.6 to 3.2) (Fig. 3). Over T2-weighted fat-saturated and conventional fast spin-echo (TR 3660 ms/TE 85 ms) images. In the axial images the mean angle of the fibres of the infraspinatus muscle relative to the central tendon was measured. This represents the pennation angle and was measured for the medial and lateral aspects of the muscle \((\alpha \text{ and } \beta)\). The measurements were performed 4 cm proximal to the distal end of the muscle. The mean length of the fibres of the infraspinatus muscle was measured at the same location.

**Histological examination of the myotendinous junction.** After fixation, the distal 8 cm of the infraspinatus muscles, including the tendon and bony insertion, were decalcified with 5% potassium nitrate acid for ten days and embedded in paraffin blocks measuring 5 mm x 10 mm x 15 mm. The silicone tendon cover was entirely surrounded by a thin tube of scar, allowing for some mechanical connection between the end of the tendon and the head of the humerus (Fig. 2c). Repair (Fig. 2e) resulted in a mean shortening of the gap between the head of the humerus and the end of the tendon of 0.60 cm (0 to 1.2).

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Diagram of sequence of muscle insertion shift. Transverse sectional drawing through the infraspinatus myotendinous junction, scapula and humeral head. Figure 2a – Intact muscle at the start of the experiments. The deltoid muscle is drawn with a dotted line. The infraspinatus muscle consists of a central tendon plate and lateral and medial sections. Figure 2b – Tendon release. The greater tuberosity is osteotomised and covered with a silicone tube to prevent spontaneous healing. Figure 2c – After 40 weeks, before repair. Despite the tendon cover, scar tissue has developed between greater tuberosity and tendon end. The scar is surrounding the tendon cover. The bone chip seems to be slightly retracted into the muscle. Figure 2d – The situation as would be expected geometrically after tendon retraction. If the muscle insertion had retracted by the same amount as the tendon end, then the pennation angle would be greater than 90° on the lateral and medial sides. Figure 2e – After repair. The neo-tendon and the tendon cover have been removed. The tendon end is repaired by screws in the greater tuberosity using two United States Pharmacopoeia #6 sutures, forming a figure-of-eight. The repair is only bridged by sutures, which strain the muscle by approximately 6 mm. Figure 2f – 35 weeks after repair. New tendon has formed between humeral head and tendon end. The insertion point of the lateral muscle fibres has advanced another 5 mm and covers the bone chip by > 50%.
the following 40 weeks the retraction increased by a mean of 1.7 cm (1.5 to 2.2). Repair resulted in a mean shortening of the existing gap of 0.6 cm (0 to 1.2) which remained stable until death.\(^8,10\)

**Muscle.** The most distal part of the infraspinatus muscle, the musculotendinous junction, retracted synchronously with the tendon by a mean of 3 cm (2.6 to 3.2) immediately after release. Some further retraction of 0.4 cm (3.0 to 4.2) occurred during the following 16 weeks. Thereafter, the musculotendinous junction appeared to shift its insertion on the tendon distally towards the head (Fig. 2c, f). The relative movement of the most lateral site of insertion of the muscle on the tendon relative to it, obtained by subtraction of the muscle retraction from that of the tendon, varied between 2.0 cm and 2.8 cm (mean 2.4) (Fig. 3) until death.

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**Fig. 4** CT before tendon repair. Transverse section through a sheep 40 weeks after tendon release. The intact shoulder with control infraspinatus (ISP) muscle is displayed below, the operated side (ISP) above. The white arrowhead points to the retracted bone chip, the black arrowhead to the original position of the bone chip on the greater tuberosity.

**Fig. 5a – MR of the healthy control side.** MRI sections through the musculotendinous junction, scapula, glenoid and humeral head. ‘ISP lat’ represents the lateral infraspinatus portion, ‘ISP med’ the medial side. The separating fasciae of lateral ISP and deltoid (D) and medial ISP with teres major (Tm) are indicated with black arrowheads.

**Fig. 5b – MRI of the operated side.** Operated muscle at euthanasia, with the retracted bone chip (white arrowhead) representing the original tendon end, neo-tendon between bone chip and humeral head (white arrows). The bone chip is partly covered by the edge of the ISP (black arrowheads).
Pennation angle and fibre lengths at 75 weeks (35 weeks after repair and post mortem)

Findings of MRI and histology (Fig. 5). A general increase in the pennation angle of the muscle has previously been described in the context of fatty muscle infiltration. Here we describe the separate measurements of the medial and lateral portions of the muscle which are necessary for individual analysis of the medial and lateral retraction of the muscle. The mean pennation angle against the central line of muscle action was increased on the lateral side, where the fibres originate from the fascia, from 23° (20° to 27°) to 52° (32° to 78°), and on the medial side, with fibres originating from the scapula, from 20° (18° to 22°) to 51° (36° to 59°). The mean fibre lengths were reduced by approximately 50% from 32 mm (28 to 34) to 18 mm (9 to 24) for the lateral and from 33 mm (27 to 39) to 14 mm (9 to 21) for the medial aspect of the muscle. The differences between the operated and the control sides were statistically significant (p < 0.005) for all four comparisons.

The preparation of the histological sections was challenging because of the presence of the bone chip within the substance of the tendon even after decalcification. Therefore, quantitative measurements were made on MRI. However, on the longitudinal histological sections it could be confirmed that the bone chip, which was initially released with the tendon end from the greater tuberosity, had retracted far towards the muscle. The chip was covered with a layer of fibrous tissue on which muscle fibres could be identified. Fatty infiltration was also present in the most distal parts of the muscle.

Discussion

The development of fresh tendon tears may rarely be followed in humans, as surgical reinsertion of acute tears is indicated. However, when retraction can be followed radiologically, it is impossible to differentiate whether a torn and shortened tendon is absorbed at the distal end or whether it is retracted into the muscle. Possible enlargement of the tendons by apposition of scar tissue at the site of rupture while the tendon has retracted into the muscle may further obscure radiological diagnosis. Therefore, an existing animal model of chronic tendon tear and delayed repair was used to investigate the development of tendon retraction and the subsequent changes in the corresponding muscle.

In this study, the muscle retracted within the first hour after experimental release by the same distance as the tendon, but over time the muscle apparently shifted its insertion on the tendon towards the head of the humerus in a hitherto unknown fashion. At the end of the experiment we found that the retracted original tendon end was covered by the distal end of the infraspinatus muscle, as if retracted into it.

If the muscle were to have maintained its site of insertion on the central tendon plate, then with retraction a theoretical mean increase of the pennation angle from the original 22° to roughly 135° for the most distal muscle fibres would geometrically have been expected, as depicted in Figure 2d. In such a position, the muscle fibres would paradoxically be pulling in the opposite direction to the main muscle action. Instead, the pennation angle increased to only a mean of 51° medially and 52° laterally, resulting in known shortening of the fibre of approximately 50%.

If tears of the rotator cuff are chronic in humans, the tendon end may retract by several centimetres. Direct reinsertion may then be impossible, owing to this and to the diminished elasticity of the musculotendinous unit. This loss of elasticity has been attributed mainly to scar formation, the formation of intramuscular fibrous tissue and recently to massive shortening of the muscle fibres. However, a massive tendon retraction that exceeds the original fibre length by almost 150% cannot be explained by shortening of the muscle fibre alone. It must result from profound alterations in muscle architecture, such as are described in this study.

With a shift of muscle relative to the central tendon, combined with the shortening of the fibres by 50%, bringing the tendon back to its original site would result in extension of the muscle fibres by approximately a factor of three, leading to disruption of their cells.

A possible explanation for the relative shift of muscle and tendon may be that the central fibres of the tendon glide relative to the peripheral fibres and are pulled into the muscle along with the bone chip. Such a relative movement would eventually result in retraction of the original tendon end into the muscle. The presence of fibrous connective tissue between the retracted bone chip and the muscle tissue supports, but does not prove, this hypothesis.

Tendon tears tend to heal spontaneously in both animals and humans by the formation of scar tissue, resulting in enlargement. The observed mechanism of shift of the insertion of the fibres partially compensates for the enlargement of the tendon in length and avoids paradoxical orientation of the muscle. Therefore, a further possible explanation for our findings may be that the muscle actively adapts the fibre orientation according to the direction of loading, in analogy to Wolff's law. It could also be speculated that new muscle tissue is produced and grows in the direction of the original insertion. This seems unlikely because the most distal portion of the muscle undergoes fatty degeneration, as does the rest of the muscle tissue on both macroscopic and MRI evaluation. Newly-generated muscle would be expected to be of normal quality. Furthermore, if neo-growth of distal muscle were to have occurred, such homogeneous orientation of the fibres as found in our study would not be expected. None the less, neo-growth or re-orientation of muscles cannot be formally excluded.

A limitation of this study is that the origin of the muscle fibres was not marked at the start of the experiment and had to be identified after dissection. CT data may underestimate the size of the muscle in the longitudinal direction, owing to difficulty in differentiating the distal infraspinatus from the deltoid muscle. Furthermore, development of the
three-dimensional orientation could not be assessed over time as the single fibre bundles are not visible in CT. However, the macroscopic findings during muscle dissection did confirm the CT and MRI measurements. The study was performed in relatively young, skeletally mature animals. The tear was acute and allowed the tendon to retract entirely and at once. Therefore, this model may not fully represent the changes associated with degenerative tears of the tendon in elderly humans.

Following experimental release in sheep the infraspinatus tendon retracts far more than the muscle. This results in an apparently shortened tendon. Massive retraction of the tendon is also known in humans, but to our knowledge, has never been attributed to changing musculotendinous architecture, rather simply to substance loss. According to our observations, muscle retraction cannot be judged reliably by measuring the retraction of the tendon end. Instead, the retraction of the myotendinous junction should be considered. If the tendon of such a remodelled muscle could be surgically reinserted, muscle fibres would theoretically be stretched by approximately 300% after retraction and consequently be damaged.

This study was supported by grants from the Swiss National Research Foundation, number 32-59553.99 and by the ResOrtho foundation, Zürich. The authors are grateful to Margarete Akens PhD, Alexander El-Warrak DVM, Rebecca Schneider DVM, Annette Kutter, med.vet., for helping with the animal experiments, Nicole Känzig for the CT measurements, Katalin Zlinszky for preparing the histology sections and Franziska Graber for preparing the electron microscopy sections. The help of Burkhart Seifert PhD with statistical analysis is gratefully acknowledged.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

References