Structure and function of the rabbit's supraspinatus muscle after resection of its tendon

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The tendinous part of the supraspinatus muscle was removed in 12 rabbits in order to simulate a major rotator cuff tear. Changes in the contractile response and the development of atrophy of the supraspinatus muscle were significant. These changes may indicate that the modest results obtained in reconstruction of major rotator cuff tears may be due to muscular insufficiency.

I have studied changes in function and histology in the supraspinatus muscle in rabbits after the tendon had been severed.

Materials and methods

Twelve adult rabbits (average weight 3.6 kg) were anesthetized using Hypnorm® 1 mL/kg body weight. Because the subcutaneous fat is very thin, the deltoid and the biceps muscles were easily identifiable. The preparation proceeded proximally following the biceps tendon in the intertubercular groove. The supraspinatus tendon was sharply dissected and detached from its bony insertion. The supraspinatus muscle was released from the surrounding tissues without damage to the neurovascular bundle, and the muscle itself was allowed to retract freely. Postoperative immobilization was not used.

The animals were killed 4, 6, 9, and 12 weeks after surgery. The scapula with the glenohumeral joint and the proximal third of the humerus were dissected bilaterally en bloc each. The samples were wrapped in wet gauze bandages, and contraction experiments were immediately performed. In order to measure the contractile properties of the supraspinatus muscle, a long muscle fiber unit was removed atraumatically and secured to a tension device. The muscle fiber unit was prestretched to a standardized pretension by a weight corresponding to 7 grams. A pair of silver/silver-chloride electrodes were inserted in the muscle unit. The preparation was placed in a bath of Krebbs-Ringer buffered (pH = 7.4) solution maintained at the temperature of 38 °C ±2° and bubbled with 5 percent CO_2 in O_2.

Square pulses (10V, 10 seconds) were delivered at a rate of 1 Hz. The isometric contraction of the electrically stimulated muscle fiber unit was recorded on the paper recorder. The first twitch of the muscle fiber unit was chosen as a peak response (reference point). Then, the preparation with maintained length was repeatedly stimulated until it no longer responded, and a complete loss of tension was observed (maximum 100 sec). Fatigue was determined as the percentage difference between the control peak response and the response measured during the continuous stimulation at 25-second intervals. After embedding the samples in paraffin and staining them with hematoxylin-eosin (HE) and van Gieson, light microscopy was used to examine the amount of degeneration and diminution of the muscle fibers, and the amount of intramuscular fat tissue in the supraspinatus muscle.

The muscle belly of the supraspinatus was divided in the middle, and the histoplanimetry of the muscle-fiber areas was performed by aid of a computer program (Revell 1983) to estimate the degree of muscle atrophy. This was expressed as the percentage share of the muscle. In each experiment the left supraspinatus muscle serves as a control.

The mean ±SE were calculated. The nonparametric Wilcoxon signed rank test was used to determine the significance of the differences between two or more groups. $P > 0.05$ was considered not significant.

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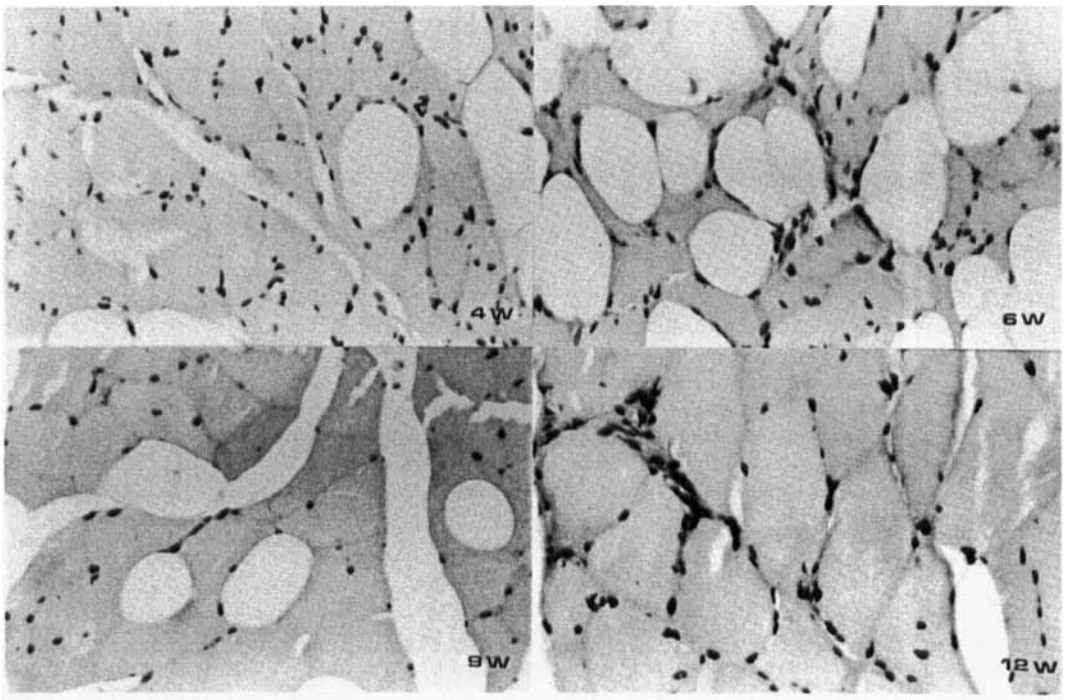

Figure 1. Photomicrographs of the retracted supraspinatus muscle during each time point showing the amount of fatty degeneration and muscle atrophy. HE, x40.

Results

The contractile response of the prestretched muscle-fiber unit, measured from the operated on shoulders up to 12 weeks postoperatively, ceased with 100 seconds of repeated stimulation, whereas the nonoperated on shoulders still showed a contractile activity of 20 percent of the peak response value. The slope of the cessation in operated on specimens was not prominent at 6 weeks, where the difference between the operated on and nonoperated on specimens reached a significant level already at 50 seconds. The contractility was decreased after repeated simulation of 75 seconds in all the operated on shoulders, although some reversibility of the fatigue could be observed in the 9- and 12-week specimens (15–20 percent of the maximal contraction, $P > 0.05$) when compared with that at 6 weeks (5 percent, $P > 0.01$; Table 1).

Fatty degeneration of the interspace and atrophy of the supraspinatus muscle fibers were observed as early as 4 weeks postoperatively. These histologic changes were most prominent at 6 weeks, and diminished towards the end of the experiment (Figure 1).

Table 1. The contractile activity of the control group and the group of removed tendinous part of the supraspinatus muscle. Mean as percentages

Duration of stimulus in seconds	Maximal contraction				Control group
	4	6	9	12 weeks	
25	76	76	76	75	77
50	48	30 *	42	48	52
75	15 *	5 **	15 *	20 *	32
100	–	–	–	–	20

* $P < 0.05$.
** $P < 0.01$.

A decrease in the percentage share of the muscle-fiber area within the supraspinatus muscle was observed at 4 ($P < 0.05$), 6 ($P < 0.05$) and at 9 ($P < 0.05$) weeks when compared with the normal side. At 12 weeks, the difference had disappeared.

Discussion

Theoretically, muscle fatigue can be due to central and peripheral factors, such as reduced muscle contractility, a rapid fall in local energy supply, failure of neuromuscular transmission, and impairment of excitation-contraction coupling (Huxley 1969). In the present experiment, muscle atrophy with simultaneously decreased contractile activity constituted the main structural and functional changes in the supraspinatus muscle after removal of its tendinous part. Because innervation of the supraspinatus muscle was intact, it is unlikely that fatigue could be due to failure of the neuromuscular transmission or impaired excitation-contraction coupling. Similarly, because the blood supply to the muscle was intact, a rapid fall in energy supply can be ruled out. Therefore, the muscle fatigue was most evidently based on reduced muscle contraction activity. The supraspinatus muscle atrophy was partly recovered at 12 weeks, and the slope had declined at 12 weeks, although the contractile response still was decreased after repeated stimulation for 75 seconds. This can be due to secondary attachment of the supraspinatus muscle to the surrounding tissues, with a partial restoration of the contractile activity. Similar observations have been reported by Smith (1983).

References

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