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Plasticity of Muscle Architecture After Supraspinatus Tears

The shoulder is the most mobile joint in the body and a common site for clinical pathology. Rotator cuff tears are common, affecting approximately 7% of elderly patients.[11] These tears frequently lead to debilitating instability and pain. The supraspinatus tendon is the most commonly damaged structure in rotator cuff tears.[5,18]

• **STUDY DESIGN:** Controlled laboratory study.

• **OBJECTIVES:** To measure the architectural properties of rat supraspinatus muscle after a complete detachment of its distal tendon.

• **METHODS:** Supraspinatus muscles were released from the left humerus of 29 Sprague-Dawley rats (mass, 400-450 g), and the animals were returned to cage activity for 2 weeks (n = 12), 4 weeks (n = 9), or 9 weeks (n = 8), before eutha-nasia. Measurements of muscle mass, pennation angle, fiber bundle length (sarcomere number), and sarcomere length permitted calculation of normalized fiber length, serial sarcomere number, and physiological cross-sectional area.

• **RESULTS:** Coronal oblique sections of the supraspinatus confirmed surgical transection of the supraspinatus muscle at 2 weeks, with reat-tachment by 4 weeks. Muscle mass and length were significantly lower in released muscles at 2 weeks, 4 weeks, and 9 weeks. Sarcomere lengths in released muscles were significantly shorter at 2 weeks but not different by 4 weeks. Sarcomere number was significantly reduced at

2 and 4 weeks, but returned to control values by 9 weeks. The opposing effects of smaller mass and shorter fibers produced significantly smaller physiological cross-sectional area at 2 weeks, but physiological cross-sectional area returned to control levels by 4 weeks.

• **CONCLUSIONS:** Release of the supraspinatus muscle produced early radial and longitudinal atrophy of the muscle. The functional implications of these adaptations would be most profound at early time points (particularly relevant for rehabilitation), when the muscle remains smaller in cross-sectional area and, due to reduced sarcomere number, would be forced to operate over a wider range of the length-tension curve and at higher velocities, all adaptations resulting in compromised force-generating capacity. These data are relevant to physical therapy because they provide tissue-level insights into impaired muscle and shoulder function following rotator cuff injury. *J Orthop Sports Phys Ther 2010;40(11):729-735. doi:10.2519/jospt.2010.3279*

• **KEY WORDS:** *muscle plasticity, rotator cuff, shoulder, tendinopathy*

There have been a number of reports documenting skeletal muscle adaptation subsequent to rotator cuff tears. In humans, imaging[10,13,17,22,26,31] and biopsies[25] have been used to document atrophy of whole muscle and muscle fibers in response to tendon tear. In animal models, a number of investigators documented radial atrophy[2,28] in muscle fiber type and size,[2] changes in passive mechanical properties of whole muscle,[14,24] and changes in connective tissue content.[14,24] Although the focus has been on changes in muscle and fiber diameter changes, it is well known that muscle fibers change in the longitudinal dimension in response to tenotomy.[32,33] These changes would be particularly important in the shoulder, as they would affect the passive mechanical properties, length-tension behavior, and force-velocity behavior of the muscle. However, changes in muscle fiber length due to changes in serial sarcomere number have not been documented, in part, because these measurements are highly invasive and require special tools. For this reason, an animal model of rotator cuff tendon tears is necessary.

Skeletal muscle architecture is defined as the arrangement of muscle fibers relative to the axis of force generation,[12,19] and it is known that architectural features primarily determine muscle function.[4,23] Architectural parameters, such as physiological cross-sectional area (PCSA) and normalized muscle fiber length (serial sarcomere number), can predict muscle maximal force,[23] excursion, and velocity.[4] However, they require careful measurement of sarcomere length, which is often ignored. Given that muscle function is a common target of exercise interventions, muscle architectural features are of particular importance to physical therapists. An accurate understanding of these tissue level features would provide a more comprehensive understanding of pathologies affecting motion and allow the rehabilitation professional to design and implement novel treatment approaches to resolve these changes.

The rat is a good model to study rotator cuff muscles because a vast amount of physiological, behavioral, and morphological data already exist for this species, and it is a frequently used model of rotator cuff tears.[2,27] The model permits high-throughput generation of rotator cuff detachments, and comparable skeletal muscle plasticity data are widely available. Therefore, the purpose of this investigation was to define the muscle architecture changes associated with supraspinatus muscle detachments in the rat shoulder. We hypothesized that complete detachment of the supraspinatus tendon would be associated with reductions in PCSA and serial sarcomere number.

METHODS

Twenty-nine adult male Sprague-Dawley rats (400-450 g) were used in this study. The left supraspinatus muscle was released from the humerus before animals were returned to normal cage activity for 2 weeks (n = 12), 4 weeks (n = 9), or 9 weeks (n = 8) before euthanasia. Differences in animal numbers between groups reflect our commitment to minimize the number of animals needed to resolve statistically meaningful differences with fewer animals when possible. Shoulders were skinned and placed in formalin for 48 hours, then rinsed in 0.2 M phosphate buffered saline for further analysis. Right shoulders served as controls. All procedures were approved by the Institutional Animal Care and Use Committees of the University of Pennsylvania and the University of California, San Diego.

Surgical Procedure

Under general anesthesia (2% isoflurane gas), the humerus was held in external rotation, and a 2-cm skin incision was made, followed by blunt dissection down to the supraspinatus tendon. The supraspinatus, which passes under the bony arch created by the acromion, coracoid, and clavicle, was isolated, and a suture was passed under the acromion to apply upward traction for further exposure. The supraspinatus was separated from the other tendons before sharp detachment at its insertion on the greater tuberosity. Any remaining fibrocartilage at the insertion was left intact, and detached tendons were allowed to retract freely without an attempt at repair, creating a gap of approximately 4 mm from their insertion sites. It should be noted that this is distinct from the natural history of human rotator cuff tendon tears, which likely involves tearing of tendons with underlying pathologic changes. The overlying muscle and skin were closed, and the rats were allowed unrestricted cage activity.[16] Animals were not given postoperative analgesics.

Histology

Bilateral shoulders from 2 animals per group were used for histological analysis. Briefly, the scapula, humerus, and soft tissues were harvested en bloc and fixed in Formalin for 48 hours before decalcification in serial dilutions of 30% formic acid. Decalcification was confirmed via plain film radiography before serial coronal oblique sections (5 μm) were made through each paraffin-embedded specimen. Individual sections were mounted and stained with Masson's trichrome and photographed for visualization of the humerus, supraspinatus tendon, and muscle belly.

Skeletal Muscle Architecture

Bilateral shoulders from remaining animals were used for architectural analysis. Briefly, the scapula, humerus, and soft tissues were harvested en bloc and fixed in Formalin for 48 hours before storage in phosphate buffered saline at room temperature for up to 2 weeks. Specimens were then sharply dissected to isolate each supraspinatus muscle and stored in phosphate buffered saline for further analysis. Muscle specimens were removed from buffer, gently blotted dry, and weighed. Muscle length (L_m) was defined as the distance from the origin of the most proximal fibers to the insertion of the most distal fibers, which excludes tendon proper from our measures of muscle length. Fiber length (L_f') was measured from 4 predetermined regions in each muscle, using a digital caliper (accuracy, 0.01 mm).[29] Surface pennation angle was measured in each of these regions with a standard goniometer as the angle between the fibers in each region and the distal muscle tendon (accuracy 5°). Muscle fiber bundles were carefully dissected from the proximal tendon to the distal tendon of each muscle region. Fascicles were then placed in mild sulfuric acid solution (15% v/v) for 30 minutes, to partially digest surrounding connective tissue, and were then rinsed in phosphate buffered saline. Under magnification, 3 small muscle fiber bundles (consisting of approximately 20 single fibers) were isolated from each muscle region and mounted on slides. Bundle sarcomere length (L_s') was determined by laser diffraction using the zero-to-first-order diffraction angle, as previously described.[20] Values for sarcomere number (S_n) or normalized fiber length (L_f) were then calculated for the isolated bundles according to the following equations[24]:

$$S_n = \frac{L_f'}{L_s'} \text{ and } L_f = L_f' \left(\frac{2.4 \ \mu\text{m}}{L_s'} \right)$$

where L_f' is the measured fiber length, L_s' is the measured sarcomere length in each fiber bundle, L_f is normalized muscle fiber length, and 2.4 μm represents the optimum sarcomere length for rat muscle.[25] Physiological cross-sectional area (PCSA) was calculated according to the following equation[23]:

$$PCSA \ (mm^2) = \frac{M \ (g) \times \cos \Theta}{\rho(g/m^3) \times L_f(mm)}$$

where M is muscle mass, Θ is pennation angle, and ρ is muscle density (1.056 g/cm[3]).[30]

Statistical Analysis

Data were screened for normality and homogeneity of variances before parametric statistics were used. Given the differences in animal mass (size) at the time of sacrifice, architectural variables were normalized to the contralateral side and are presented as percent control. One-way analyses of variance were used to test for differences in each dependent variable, and post hoc Tukey tests were used to define individual differences at each time point. No statistical differences in regional fiber length change were noted, so values from each of the 4 regions were averaged to yield 1 fiber length value per muscle. All values are reported as mean ± standard error (SE), unless otherwise noted. Statistical tests were performed using SPSS Version 16.0 (SPSS, Inc, Chicago, IL). Significance was set at the $P<.05$ level.

RESULTS

Histological sections served as important positive controls to insure that the surgical procedure had the intended consequences. In control shoulders at 2 and 4 weeks, supraspinatus tendons were intact between the muscle and humerus (FIGURE 1). Although the supraspinatus tendon was still deta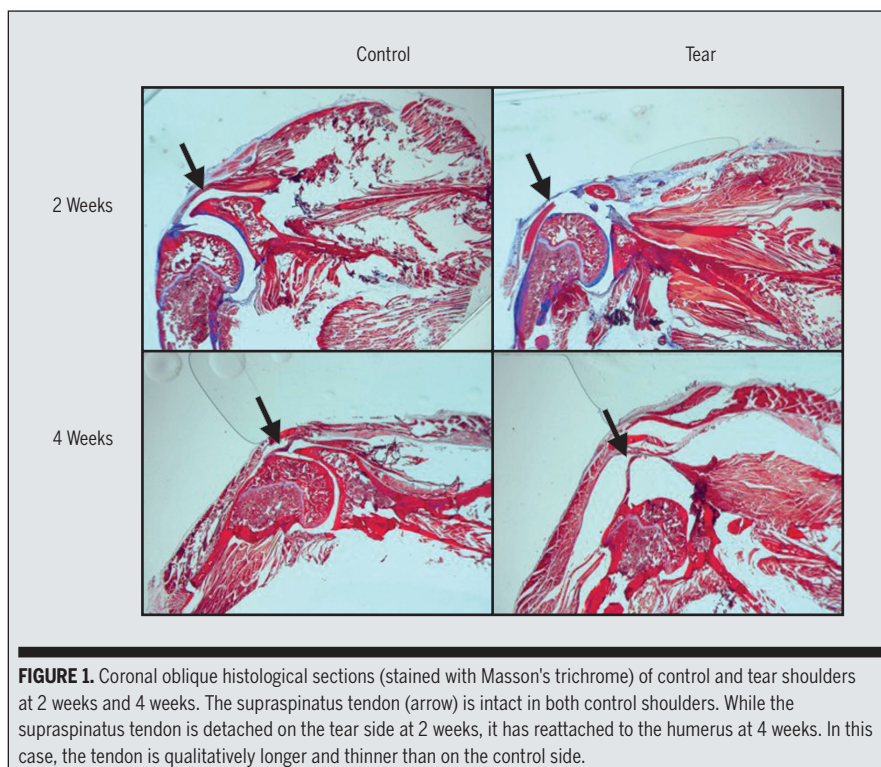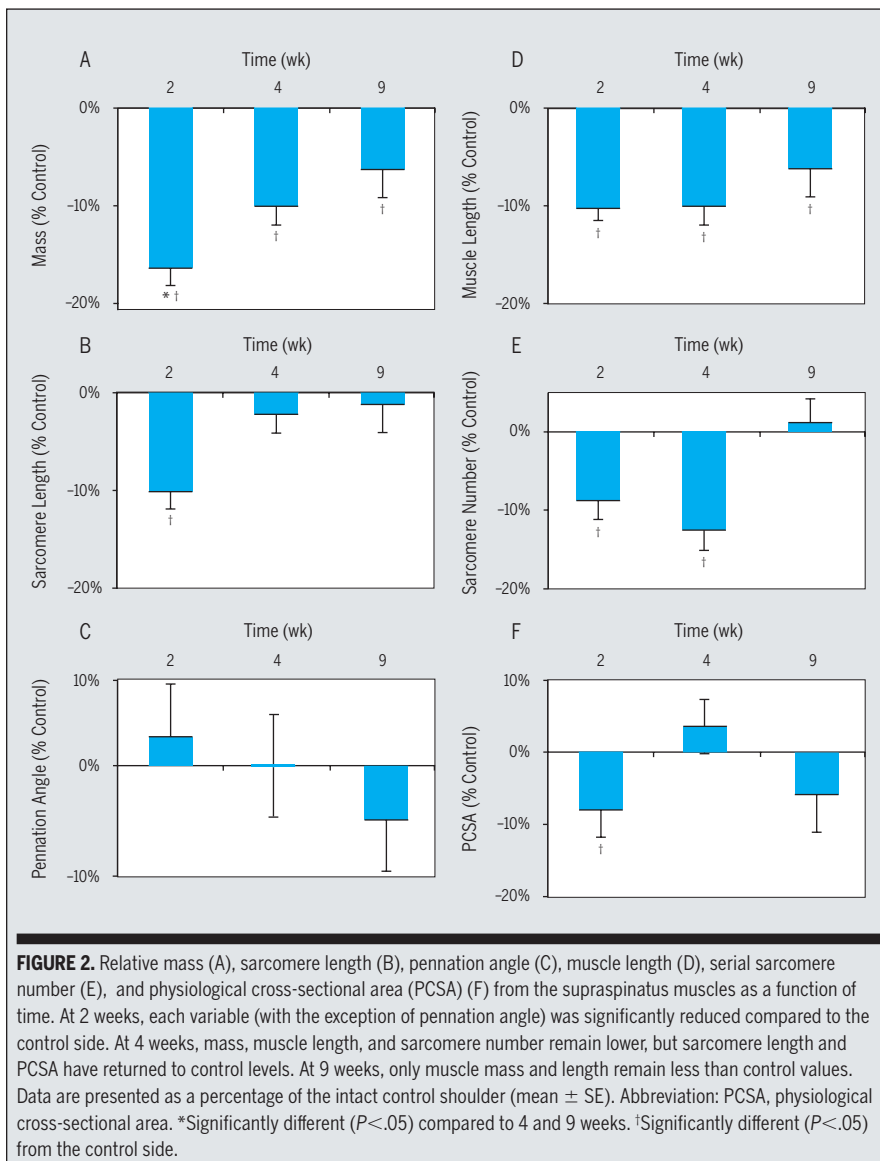ched at 2 weeks, it had reattached to the humerus at 4 weeks. Qualitatively, it appeared that these tendons were longer and thinner compared to control tendons.

Architecturally, the torn and control supraspinatus muscle varied considerably as a function of time. In general, there was evidence of both radial (cross-sectional area) and longitudinal (sarcomere number) atrophy initially and then these values returned towards control by 9 weeks. In terms of the gross parameter mass, there was a significant decline in mass at 2 weeks (–16.5% ± 1.8%), 4 weeks (–10.1% ± 1.8%), and 9 weeks (–6.3% ± 2.9%) in the torn supraspinatus muscles compared to control (FIGURE 2A). These values were returning towards control values at 4 and 9 weeks but remained significantly lower at each time point. Similarly, muscle length was significantly shorter at 2 weeks (–10.2% ± 1.2%), 4 weeks (–10.0% ± 1.8%), and 9 weeks (–6.2% ± 1.3%) in the torn supraspinatus muscles compared to control (FIGURE 2B). Sarcomere length was significantly shorter at 2 weeks (–10.0% ± 16%), which then returned to control values at 4 and 9 weeks (FIGURE 2C). This indicates fiber/muscle retraction. Serial sarcomere number was reduced at 2 weeks (–8.9% ± 2.2%) and 4 weeks (–12.5% ± 2.6%) and then returned to control levels by 9 weeks (FIGURE 2D). Pennation angles did not change significantly at any time point (FIGURE 2E). PCSA was significantly reduced at 2 weeks (–8.0% ± 3.9%) and then returned to control levels by 4 and 9 weeks (FIGURE 2F).

DISCUSSION

The purpose of this study was to define the muscle architectural changes associated with supraspinatus detachment in a rat model. These data demonstrate that rapid radial (PCSA) and longitudinal (fiber length/sarcomere number) atrophy occurs in response to rotator cuff tenotomy. This is reflected in a gross decline in muscle mass and length that does not fully recover by 9 weeks. Our specific measurements of PCSA and fiber length also

FIGURE 1. Coronal oblique histological sections (stained with Masson's trichrome) of control and tear shoulders at 2 weeks and 4 weeks. The supraspinatus tendon (arrow) is intact in both control shoulders. While the supraspinatus tendon is detached on the tear side at 2 weeks, it has reattached to the humerus at 4 weeks. In this case, the tendon is qualitatively longer and thinner than on the control side.

FIGURE 2. Relative mass (A), sarcomere length (B), pennation angle (C), muscle length (D), serial sarcomere number (E), and physiological cross-sectional area (PCSA) (F) from the supraspinatus muscles as a function of time. At 2 weeks, each variable (with the exception of pennation angle) was significantly reduced compared to the control side. At 4 weeks, mass, muscle length, and sarcomere number remain lower, but sarcomere length and PCSA have returned to control levels. At 9 weeks, only muscle mass and length remain less than control values. Data are presented as a percentage of the intact control shoulder (mean ± SE). Abbreviation: PCSA, physiological cross-sectional area. *Significantly different ($P<.05$) compared to 4 and 9 weeks. †Significantly different ($P<.05$) from the control side.

support this fact, although with shorter time courses. Although radial (PCSA) and longitudinal (sarcomere number) atrophy explain muscle mass decline at 2 weeks, longitudinal (sarcomere number) atrophy explains all of the mass decline at 4 weeks. By 9 weeks postinjury, muscle mass declines appear to be the result of longitudinal (muscle only) atrophy as muscle architecture is reorganized. We did not observe changes in surface pennation angle as others have observed in some cases,[21] which perhaps is a product of the fact that fatty infiltration is not observed in this particular model.[2] One ex-

planation for reduced muscle length with normal sarcomere number and length is that serial fiber number is reduced. This is possible because fibers do not span the entire length of the muscle in the rat supraspinatus, which would allow muscle length to be controlled by subtracting the total number of fibers attaching to the tendon.

These structural data suggest the following important series of events that occur after supraspinatus tenotomies in this model. First, when the intact supraspinatus muscle is released, the muscle retracts. This is evident in the 10% decline

in sarcomere length at 2 weeks. During this time the muscle atrophies in both the radial and longitudinal dimensions (**FIGURES 2** and **3**). This has been observed in other tenotomy models.[1,8] Second, between 2 and 4 weeks, the tendon appears to reattach itself to the humerus. This scarring reattachment has been documented previously in the rat[27] and other animal models.[7] During this time, the muscle continues to subtract sarcomeres serially from its muscle fibers, which allows sarcomere length to be restored (**FIGURES 2** and **3**), and may allow tension to be transmitted through the muscle because PCSA is restored as well. By 9 weeks, the architectural features of the muscle are nearly restored, albeit in a shorter muscle (fewer serial muscle fibers).

In humans, it is well known that full-thickness, complete supraspinatus tears do not heal spontaneously.[5] This is a known limitation of all animal models of rotator cuff tendon tears.[7,27] However, the early response of the detached muscle is likely representative of the human condition, as rat muscle adaptation has consistently represented the human condition in other models.[3,9] Importantly, it demonstrates the capacity of rotator cuff muscles to adapt in the radial and longitudinal dimensions, which has been previously underappreciated. This is important, because longitudinal atrophy (fewer sarcomeres in series in a fiber) helps explain the increases in passive tension observed in animal and human whole-muscle preparations.[15,24] Passive tension is important in rotator cuff tendon tears, because it must be overcome when surgically reattaching the tendon to the humerus, and large tension is associated with poor functional outcome.[6] Additionally, these changes would be expected to have a profound influence on function. Although it is well known that radial atrophy reduces the maximum force-generating capacity of muscle, longitudinal fiber atrophy (as seen in the first 4 weeks) would reduce force-generating capacity for 2 reasons. First, if joint range of motion is pre-
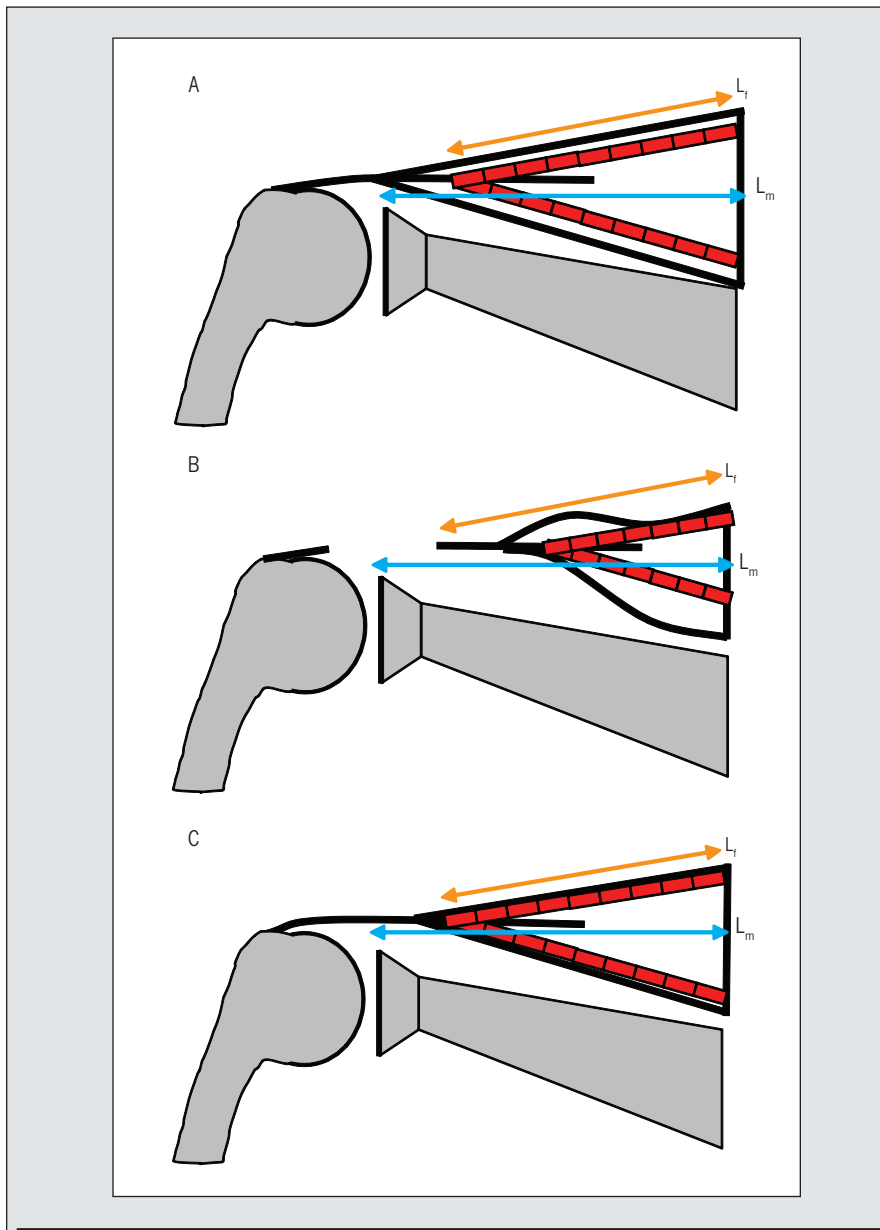
FIGURE 3. Schematic representation of the time course of architectural changes in the detached supraspinatus muscle. When the supraspinatus muscle is acutely detached (A and B), the muscle retracts, muscle mass declines, sarcomere length shortens as the muscle recoils, sarcomere number declines, and physiological cross-sectional area declines. When the supraspinatus muscle reattaches itself (B and C) in this model, the tendon becomes longer because the muscle cannot lengthen itself before reattaching. At this point, the muscle has the capacity to generate resisted tension, which may explain why sarcomere length, sarcomere number, and PCSA return to control levels. The reduction in muscle length at this time point is likely due to a reduction in serial fiber number.

served after the tear, muscle fibers (and, therefore, sarcomeres) would be forced to operate over a wider range of lengths and therefore at potentially less advantageous positions on the length-tension curve at some joint angles. Second, if joint angular velocities remain constant, muscles fibers would be forced to operate at higher velocities, further compromising force-generating capacity when the muscle is shortening.[4]

These data have important clinical implications. First, it appears the supraspinatus muscle seeks a predefined sarcomere length. When tendon morphology is compromised, the muscle adapts by reducing sarcomere number to restore this predefined value. This is important clinically because alterations in sarcomere number will likely affect muscle function. Second, the fact that fiber lengths return to control values, while the overall muscle remains shortened, suggests that there is a reduction in serial muscle fiber number and there is some change in the interaction between muscle fibers and the extracellular matrix, which is an important area of future research and is consistent with previous findings.[21] Third, and less surprising, is radial atrophy in response to tenotomy. However, all of these changes would be expected to impair muscle function and would need to be restored to achieve normal muscle function. This highlights the need to precisely restore muscle length and/or fiber length intraoperatively when reattaching the tendon to the humerus. If muscle length is restored, muscle architecture may be restored through hypertrophy, and postoperative muscle function would be optimized.

It is reasonable to assume that the early changes in muscle architecture (prior to tendon scarring) observed in this model would apply to tendon tears in humans. From a rehabilitation perspective, this suggests that, as a tendon retracts, radial and longitudinal atrophy of the muscle will occur. These tissue-level changes would then explain, at least in part, impairments in muscle function. At the time of repair, these changes are likely to persist for some period, and it is the responsibility of the physical therapist to challenge that muscle in a way that stimulates radial and longitudinal hypertrophy while protecting the repair site. The ideal exercise intervention for resolving these tissue-level changes should, of course, be a focus of future work.

The idea of restoring muscle length intraoperatively, although logical, is difficult to implement clinically for several

reasons. As tendon tears in humans are often related to mechanical wear over time, there is often tendon degeneration present at the time of repair. Secondly, if a muscle is retracted, these data suggest that there may also be reductions in fiber length and muscle length that are present at the time of repair. Both of these adaptations, shorter fibers/muscle and a degenerated segment of tendon, would make it difficult to make an anatomical repair without imparting significant tension on the muscle-tendon unit. As mentioned previously, overtensioning has been associated with poor functional outcomes.[6] One possible solution to this problem would be to reattach the supraspinatus tendon to the native position on the humerus and then to control adduction postoperatively. Progressively lowering the humerus into neutral abduction would limit tension on the muscle-tendon unit, thereby protecting the repair site, and it would allow the muscle to adapt to its new mechanical environment (perhaps by increasing fiber length and muscle length). Of course, during this period of motion restriction it would be important to maintain joint mobility through restricted range-of-motion activities and joint mobilization techniques. Following restoration of range of motion, controlled loading of the muscle will be needed to generate radial and longitudinal hypertrophy. Although lengthening contractions are typically the most effective strategy for this type of adaptation, the timing and intensity of such exercises are likely to be tissue quality specific and should be the focus of future empirical experiments.

CONCLUSION

TENOTOMY OF THE SUPRASPINATUS muscle results in early radial and longitudinal atrophy of the muscle. These changes are expected to impair muscle function. In this animal model, the tenotomized muscle scars to the humerus over time, which allows some architectural features to be restored.

This scarring capacity suggests that this animal model deviates from the human condition at later time points, but it further highlights the adaptability of the skeletal muscle to its mechanical environment. In the human condition we would expect permanent radial and longitudinal atrophy of the muscle if the tendon is completely torn. If the tendon is surgically reattached, it is important to expose the muscle to controlled loading to facilitate hypertrophy while protecting the repair site during healing. The optimal loading (rehabilitation) strategy will be the focus of future work but these data provide a tissue-level explanation for muscle performance impairments after supraspinatus tendon tears. ◉

■ KEY POINTS

FINDINGS: Acute supraspinatus tendon tenotomy results in both longitudinal and radial atrophy of the supraspinatus muscle.

IMPLICATION: These tissue-level changes would impact muscle function by reducing the maximum force-generating capacity of the muscle and by forcing the muscle to operate over a wider range of sarcomere lengths, further reducing force-generating capacity.

CAUTION: The rat shoulder model has different healing properties than that of humans, so the timing and magnitude of these tissue level changes may differ from humans.

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