

ABSTRACT: Rabbit extensor digitorum longus (EDL) tendons were cut with the muscle active (active tenotomy, AT) or with the EDL at rest (passive tenotomy, PT). One, 7, and 21 days after tenotomy, contractile testing was performed. A second experiment was performed in which EDL tendons underwent PT and, after a 7-day delay, muscle-tendon units were restored to their original length. Maximum isometric tension dropped precipitously 1 day after either AT or PT to approximately 50% of normal and continued to decline by day 7. In contrast to PT, where peak tension (P_o) decreased further by 21 days, after AT, P_o partially recovered. Differences in muscle mass, cross-sectional area, fiber type, and sarcomere number did not explain the differential response. One day after length restoration of muscles, P_o rapidly increased by approximately 40%. These observations have implications for understanding the outcome of muscle-tendon unit injury and surgical repair.

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SKELETAL MUSCLE RECOVERY AFTER TENOTOMY AND 7-DAY DELAYED MUSCLE LENGTH RESTORATION

REID A. ABRAMS, MD,¹ ALBERT M. TSAI, MD,¹ BRANDI WATSON, MD,¹
AMIR JAMALI, MD,¹ and RICHARD L. LIEBER, PhD^{2,3}

¹ Department of Orthopedic Surgery, University of California, San Diego, 200 West Arbor St., San Diego, California 92103, USA

² Departments of Orthopedics and Bioengineering, University of California, San Diego Medical Center, San Diego, California, USA

³ Department of Veterans Affairs Medical Center, San Diego, California, USA

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Tenotomy is a common phenomenon that may result from trauma (e.g., tendon laceration or insertion avulsion), degenerative processes (e.g., rotator cuff, distal biceps, or Achilles rupture), rheumatoid arthritis (e.g., tendon rupture), or surgical manipulation (e.g., tendon transfer or lengthening). Considerable emphasis has been given to the mechanisms of tendon healing that occur after laceration and repair, and the study of postoperative rehabilitation programs which are aimed at optimizing healing while limiting adhesion formation.^{6,7,20,22,24} It has tacitly been assumed that the main requirement for a successful surgical outcome is a healed tendon that glides well.

Almost no attention has been given to muscle structural and functional changes that occur secondary to tendon injury. It is possible that the clinical

outcome of tendon repair is affected by muscle changes occurring secondary to tenotomy. Reported muscle changes after tenotomy have included increased intramuscular connective tissue, decreased capillary density, Z-disc streaming, early muscle fiber necrosis, and decreased twitch tension.^{1,3,5,10,11} These changes may reflect injury and repair processes or perhaps an adaptive remodeling process and have not previously been accompanied by measures of muscle functional properties.

In spite of the relatively common occurrence of tenotomy in disease, trauma, and surgery, our lack of understanding of muscular changes makes development of rational treatment protocols difficult. An understanding of muscle changes occurring after tenotomy may influence both the timing and technique of surgical repair, postoperative rehabilitation strategy, and outcome when performing tendon repair, grafting, or tendon transfer. Since tenotomy can occur while the muscle is actively contracting or at rest, the purpose of this study was to compare the effects of active tenotomy (AT) to passive tenotomy (PT) on muscle properties as well as to determine the nature of recovery after muscle length was re-

Abbreviations: AT, active tenotomy; EDL, extensor digitorum longus; L, muscle length; L_f , fiber length; L_o , optimal length; MHC, myosin heavy chain; NCAM, neural cell adhesion molecule; P_o , peak tension; PT, passive tenotomy; TA, tibialis anterior

Key words: EDL; muscle length; muscle-tendon units; tenotomy

Correspondence to: R.A. Abrams; e-mail:rlieber@ucsd.edu

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stored. Brief reports of this work have been presented.^{8,25}

MATERIALS AND METHODS

Since our ultimate goal is to apply the results of these experiments to treatment of human digital flexor and extensor tendon ruptures or lacerations, we chose the rabbit extensor digitorum longus (EDL) muscle-tendon unit as the model, since it has multiple tendons from a common muscle belly, similar architectural features to the human digital flexors and extensors, and available normal muscle architectural and contractile data.^{12,15}

Experimental Design. Sixty-nine male New Zealand white rabbits (mass 2.39 ± 0.3 kg) were separated into active ($n = 33$) and passive tenotomy ($n = 36$) groups. Historical controls were also included in the analysis for comparison purposes. The normative data that we previously collected in our laboratory are actually data from the *same* population of rabbits used in the tenotomy study and were measured using precisely the same apparatus. We believe that it is unethical to sacrifice more animals to perform identical control experiments as were provided over the previous decade in this laboratory for purposes of establishing control values.^{13,14,16,18} Tenotomy of the left EDL tendons was performed as described below. Contractile testing and muscle harvesting were performed 1, 7, or 21 days after tenotomy. To study muscle recovery after tenotomy with delayed muscle length restoration (such as occurs clinically), EDL tendons underwent PT and 1 week later were restored to their original length and sutured to the ankle retinaculum. One, 7, or 21 days after length restoration, contractile testing was performed. Animal care adhered to the *NIH Guide for the Care and Use of Laboratory Animals* and the experimental protocol was approved by the San Diego V.A. Medical Center Animal Use Subcommittee. Following terminal experiments, all animals were euthanized by intravenous injection of pentobarbital sodium via the marginal ear vein.

Active or Passive Tenotomy. Rabbits were maintained on 2–4% isoflurane and positioned supine with the left foot secured to a footplate attached to the arm of a motor (Cambridge Technology Model 6400, Cambridge, Massachusetts) which enabled direct measurement of dorsiflexion torque during muscle activation (Fig. 1). Subcutaneous electrodes were placed near the peroneal nerve at the proximal fibular head and stimulation threshold was recorded using single twitches under computer control (Super-

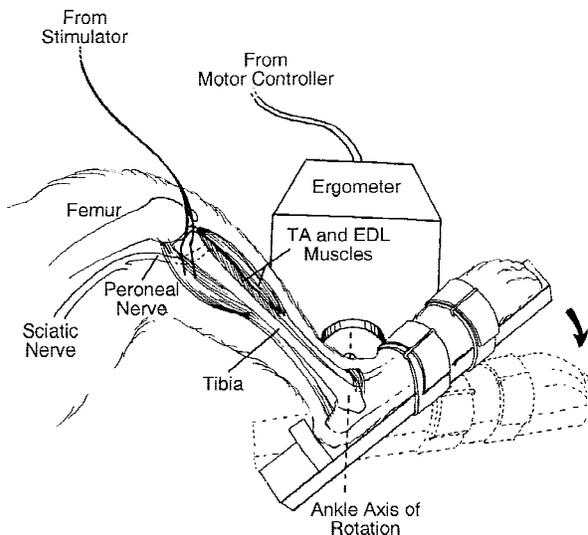


FIGURE 1. Experimental method for controlled, active tenotomy of rabbit dorsiflexor muscles. The peroneal nerve is activated, causing isometric ankle dorsiflexion. Then, the EDL tendons are cut, resulting in active tenotomy and an abrupt torque decrease. (Figure used by permission from Lieber et al.¹⁷)

scope II, G.W. Instruments, Watertown, Massachusetts). Maximal torque was recorded using single twitches at increasing voltage until a plateau was reached.

After determination of maximum torque, a longitudinal incision was made between the proximal and distal anterior ankle retinacula and the four slips of the EDL tendon isolated in the jaws of an iris scissors. For AT, the muscle was stimulated for 1.2 s at supramaximal voltage and optimal frequency and, when maximal force was reached, the tendon was briskly cut, resulting in sudden proximal muscle retraction and drop in joint torque (Fig. 2). For PT, tendons were cut without muscle activation. Incisions were closed with subcuticular 5-0 Dexon and dressed with a Nexaband dressing. Animals were monitored until awake and returned to unrestricted cage activity. No abnormal gait patterns or signs of discomfort were observed in any animal subjects and weight gain during the recovery period was similar to untreated animals of the same age.

Delayed Muscle Length Restoration. Rabbits ($n = 22$) were anesthetized and the left EDL tendons underwent PT using the protocol described above. After a 7-day delay, animals were reanesthetized, the incisions were reopened and the tenotomized and shortened muscle-tendon units were exposed. The proximal tendon stumps were reextended to 1 cm distal to the proximal ankle extensor retinaculum (approximately the length that would be required to

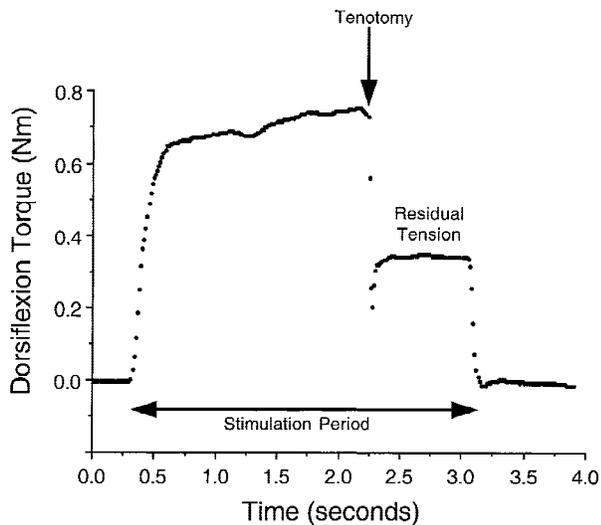


FIGURE 2. Real-time contractile record of dorsiflexion torque during active tenotomy. During AT of the EDL tendons (arrow), ankle dorsiflexion torque dropped by $52.7 \pm 4.2\%$ within 10 ms. The residual tension was due to the torque generated by the intact TA.

repair the tendons to the distal stumps) and secured to the retinaculum with 4-0 Ticron sutures (Davis and Geck, Wayne, New Jersey). One, 7, or 21 days following muscle-tendon length restoration (i.e., 8, 14, and 28 days after PT) animals ($n = 7-8$ per time period) underwent terminal simultaneous contractile testing of the EDL and tibialis anterior (TA) muscles after readjusting the muscle to optimal length. The TA muscles, which had not been treated, were tested as a control for either animal growth or muscle atrophy secondary to the two surgical treatments.

Muscle Isometric Contractile Testing. One, 7, or 21 days after tenotomy, or tenotomy followed by delayed muscle length restoration, animals underwent terminal muscle contractile testing. Anesthesia was induced and an anterior midline incision was made from mid-thigh to the ankle. The peroneal nerve was exposed and the leg was fixed to a rigid frame with 3.2 mm Steinman pins inserted through the distal femur and proximal tibia. The TA was cut distally and reflected. The proximal tendon stumps of the previously cut EDL were identified and in situ muscle length (L_{is}) was measured with dial calipers (Model 505-633, Mitutoyo Corp., Tokyo, Japan) as the distance between the origin of the most proximal muscle fiber to the insertion of the most distal fiber. The distal end of the EDL was securely to a servomotor (Cambridge Technology Model 6400, Cambridge, Massachusetts) maintaining the muscle at in

situ length. Motor output corresponding to L_{is} was recorded. Stimulus threshold and maximum force was recorded and the remainder of the contractile tests were performed at 1-2 times maximal voltage. Muscle temperature was maintained at 37°C during testing with radiant heat, a mineral oil bath, and a servo-controller (Yellow Springs Instrument model 73A, Yellow Springs, Ohio). At L_{is} , contractile properties were measured during twitch and tetanic contractions at stimulation frequencies from 5-200 Hz, of 600-800 ms duration, and a 90-s rest period interposed between successive tetanus. EDL twitch and tetanic tensions were measured at increasing lengths until optimal muscle length (L_0) at which peak tension (P_0) occurred was determined. The motor output corresponding to L_0 was recorded and the change in length (ΔL) from L_{is} to L_0 was calculated, where $\Delta L = L_0 - L_{is}$.

The protocol for measuring contractile properties for the animals undergoing PT and delayed muscle length restoration differed slightly from those undergoing tenotomy alone. First, ΔL was not measured since only the 7-day post-tenotomy time period was studied. Second, P_0 for the TA was also evaluated over time as a control for animal growth. Instead of reflecting the TA, in these groups, the TA tendon was affixed to a second servomotor and tetanic tension was simultaneously measured during the same activation as the EDL. Control experiments in which terminal nerve branches to the two muscles were isolated demonstrated neither an effect of simultaneous TA testing on EDL tension or EDL testing on TA tension.

After contractile testing, the EDL muscle was excised and weighed. Half of each muscle sample split longitudinally was immediately fixed in 10% buffered formalin at L_0 for 24-48 h and then stored in phosphate buffered saline for architectural analysis. The remaining muscle half was frozen at L_0 in isopentane cooled by liquid nitrogen (-159°C) and stored at -80°C for subsequent histochemical processing.

Architectural Analysis. Muscle length was defined as the distance from the origin of the most proximal fibers to the insertion of the distal most fibers as measured on the formalin-fixed specimens. Fiber bundles of 3-50 fibers were dissected from the proximal medial, proximal lateral, and distal portions of each muscle and mounted onto standard microscope slides. Fiber length (L_f) was measured for each fiber using a digital filar eyepiece and XM processor (Lasico, Los Angeles, California). Average sarco-

mere length of each fiber specimen was measured in three locations along each bundle using laser diffraction¹⁹ and measured L_f was normalized to a sarcomere length of 2.5 μm to compensate for positional variations during fixation. Serial sarcomere number for each fiber was calculated by dividing normalized mean L_f for each of the fiber bundles by measured sarcomere length. Physiological cross-sectional area was calculated as described by Sacks and Ro.²¹

Histochemical Analysis. Muscles that had been stored at -80°C were sectioned at -20°C using a cryostat (Reichert-Jung, Model 9500, Leica, Inc., Deerfield, Illinois) and mounted onto glass slides. Serial 10 μm thick transverse sections from the muscle midportion were stained with hematoxylin and eosin (H&E) for observation of general muscle morphology and immunostained with antibodies against fast (MHC_f), slow (MHC_s), or developmental (MHC_d) myosin heavy chain isoforms (Vector Laboratories, Burlingame, California). The end regions of the muscle fibers, which may show necrotic changes secondary to tenotomy,¹ were not examined. Positive controls for the specificity of MHC_d were generated by injecting 1.5 cc of 0.5% Bupivacaine into the TA and EDL muscles of 1 animal. Cross-sectional areas of both fast and slow muscle fibers were measured using the Image 1 analysis program (Universal Imaging Corp., Westchester, Pennsylvania) interfaced to a Nikon Microphot light microscope (Nikon Inc., Troy, New York). Approximately six fields randomly chosen from each of two sections was sampled per muscle resulting in a total of approximately 400 fibers sampled per muscle.

Statistical Analysis. Significance level was set to 0.05 (i.e., $\alpha = 0.05$) and statistical power to at least 80% (i.e., $\beta = 0.20$) for all insignificant changes reported. Contractile properties, architectural parameters, muscle mass, and fiber size areas were compared by two-way ANOVA using time period (1, 7, or 21 days post-tenotomy) and tenotomy type (active vs. passive tenotomy) as grouping factors.²³ In the PT and delayed muscle length restoration experiment, contractile parameters were compared separately for both the TA and EDL as a function of time (1, 7, or 21 days after length restoration, corresponding to 8, 14, or 28 days after the original tenotomy) between time groups by one-way ANOVA. Data were analyzed using StatView 5.0 (Abacus Concepts, Berkeley, California). All data are presented in figures as mean value \pm SEM.

RESULTS

Muscle Contractile Properties after Active and Passive Tenotomy.

During AT of the EDL tendons, ankle dorsiflexion torque dropped by $52.7 \pm 4.2\%$ within 10 ms (Fig. 2). P_0 measured one day after AT decreased significantly by about 40% from 43.7 ± 16.9 N (observed for normal EDL muscles from age- and weight-matched animals¹⁷ to 24.7 ± 1.7 N ($P < 0.001$). P_0 continued to decrease significantly to 15.9 ± 1.3 N on day 7 ($P < 0.01$) and then recovered by day 21 to 22.7 ± 1.11 N, a level not significantly different from day 1 ($P > 0.5$, Fig. 3, filled bars). In contrast, P_0 after PT showed a qualitatively different time course. P_0 decreased by about 45% to 23.5 ± 1.4 N 1 day after tenotomy, and P_0 continued to decline to 18.7 ± 2.4 N by 7 days and 16.9 ± 0.7 N by 21 days after tenotomy (Fig. 3, open bars). We have confidence in the 21-day time points as these experiments were performed on anywhere between 9–12 animals per group and the standard deviations of our data are less than 15% of the experimental mean. Two-way ANOVA of these data revealed the differential effect of tenotomy method (active vs. passive) on P_0 as a function of time, as indicated by the significant interaction term ($P < 0.01$). It is theoretically possible that muscle length changes could affect P_0 values. As muscle fibers shorten, maximum muscle force will decrease. However, after each experiment, P_0 was measured after readjusting the muscle to optimal length. Were this not to be the case, force measurements would be confounded by differences in sarcomere length. Therefore, P_0 decreases were not due to alterations in sarcomere length because sar-

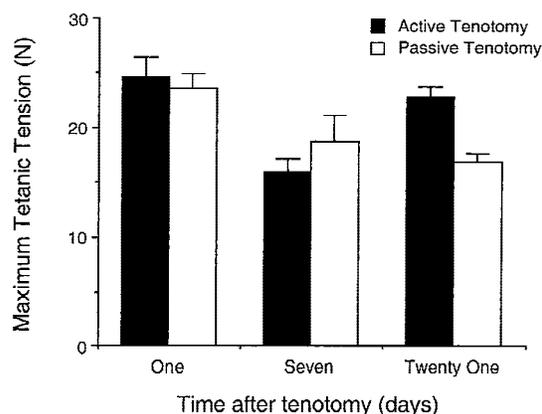


FIGURE 3. Maximum isometric tension generated by EDL muscles after active tenotomy (filled bars) or passive tenotomy (open bars). Two-way ANOVA revealed a differential effect on tenotomy based on the significant interaction term. Data shown are mean \pm SEM ($n = 7-8/\text{group}$).

comere length was readjusted to optimal before each tetanic measurement.

Comparison between in Situ and Optimal Muscle Length. One and 7 days after AT, ΔL was relatively high (5.6 ± 0.4 mm and 6.6 ± 0.8 mm, respectively), but by 21 days, ΔL had decreased to only 3.4 ± 0.5 mm (Fig. 4, filled bars). A similar trend was observed after PT where ΔL at 1 and 7 days was 7.6 ± 0.7 mm and 7.2 ± 0.8 mm, respectively, and at 21 days was 2.7 ± 0.2 mm (Fig. 4, open bars). Two-way ANOVA revealed no significant difference between tenotomy method ($P > 0.5$) but a significant effect of time ($P < 0.01$). Therefore, ΔL varied little between day 1 and day 7 for either tenotomy type but decreased significantly for both tenotomy types by day 21 ($P < 0.0001$).

Architectural Changes after Active and Passive Tenotomy. Two-way ANOVA of serial sarcomere number revealed no significant difference between tenotomy method ($P > 0.6$) but a significant effect of time ($P < 0.05$), demonstrating a decline in sarcomere number by 21 days relative to days 1 and 7 for both tenotomy types (Table 1). After AT, serial sarcomere number declined significantly by about 15% from $6,224 \pm 302$ on day 1 to $5,324 \pm 287$ on day 21 ($P < 0.01$). After PT, sarcomere number decreased significantly from $6,171 \pm 159$ on day 1 to $5,735 \pm 331$ on day 21 ($P < 0.01$). Neither the 1-day nor the 7-day serial sarcomere number from either group was significantly different from control values from animals in which no procedures were performed.¹² The changes in muscle mass (Table 1) are interesting

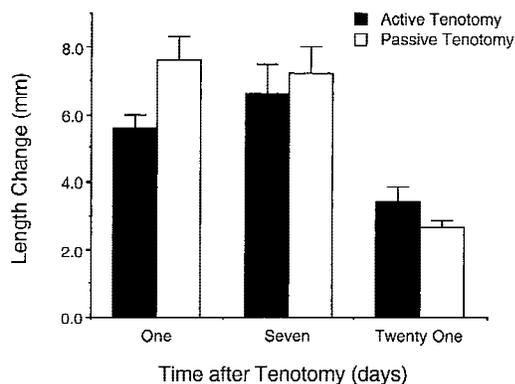


FIGURE 4. Length change required to increase muscle length from in situ length to optimal length after active tenotomy (filled bars) or passive tenotomy (open bars). Note that a smaller length increase is required to lengthen the muscle to optimal length 21 days after tenotomy. Data shown are mean \pm SEM ($n = 6-8$ /group).

and, we believe, real. Based on previous measurements, we think that this change in muscle mass represents a transient shift in water content. While we did not measure water content in these particular muscles, we are unwilling to use mass alone as an index for muscle cross-sectional area. By itself, muscle mass would have no effect on tension measurements, but it would have a dramatic effect on expressing muscle force per unit mass or per unit cross-sectional area. Because there is a question as to whether or not this calculation can be performed, we chose not to present specific tension measurements because we think they are misleading.

In spite of the functional differences observed between groups and at various time periods, no such corresponding structural data were observed. Thus, EDL muscle mass, physiologic cross-sectional area, and fast and slow fiber size at days 1, 7, and 21 did not differ significantly between active and passive tenotomy groups ($P > 0.2$; Table 2). No positive immunostaining for the developmental MHC isoform was observed at any time period although positive controls after Bupivacaine injection indicated the appropriate antibody specificity. There was a tendency for immunostained tissue from 21-day animals to demonstrate an increased coexpression of the fast and slow isoform. This only affected $\sim 10\%$ of the muscle fibers from these groups and no correction was made for coexpression.

Effects of Muscle Length Restoration on Muscle Contractile Properties. P_0 increased significantly and rapidly from 18.7 ± 2.4 N 7 days after tenotomy to 31.2 ± 2.0 N only 1 day after muscle length was restored (i.e., 8 days after PT; Fig. 5, filled bars). Over the ensuing time periods, P_0 continued to increase so that by 21 days after muscle length restoration (i.e., 28 days after tenotomy), it reached 45.4 ± 1.2 N, a value not significantly different from normal EDL muscles from animals of the same age and size¹⁷ ($P > 0.7$). P_0 measured for the TA remained constant at ~ 16 N for all time periods (Fig. 5, open bars), indicating that the increase in P_0 for the EDL was not simply secondary to animal growth or large changes in animal activity.

DISCUSSION

Tendon laceration, rupture, and tendon surgery are common clinical occurrences all of which involve tendon discontinuity and which can be simulated experimentally by tenotomy. Since tenotomy-induced muscle changes can influence the outcome of tendon surgery, the aim of this study was to learn

Table 1. EDL architectural properties.*

Experimental group	Muscle mass (g)	Muscle PCSA (cm ²)	Serial sarcomere no.
Active tenotomy (<i>n</i> = 9–12/group)			
1 day	3.68 ± 0.24	2.54 ± 0.26	6,223 ± 302
7 days	3.32 ± 0.24	2.05 ± 0.14	6,193 ± 190
21 days	3.89 ± 0.13	2.23 ± 0.50	5,323 ± 287
Passive tenotomy (<i>n</i> = 9–11/group)			
1 day	2.96 ± 0.06	1.84 ± 0.05	6,170 ± 159
7 days	3.40 ± 0.31	2.44 ± 0.28	5,939 ± 217
21 days	2.85 ± 0.07	1.92 ± 0.12	5,374 ± 330
Control muscles (<i>n</i> = 6)*	3.56 ± 0.21	1.92 ± 0.20	5,844 ± 185

*Control data from Lieber and Blevins.¹²
PCSA = physiological cross-sectional area.

about the behavior of muscle after tenotomy through evaluation of contractile, histological, and architectural properties.

Tenotomy resulted in a precipitous decrease in P_0 that persisted over the ensuing 7 days independent of whether the tenotomy occurred while the muscle was actively contracting or at rest. However, 21 days after tenotomy, there was a differential effect of tenotomy condition where those muscles that had undergone AT (Fig. 3, filled bars) demonstrated partial recovery of contractile properties whereas those that had undergone PT continued to decrease in force-generating capacity (Fig. 3, open bars).

We were not able to provide a clear structural basis either for the precipitous drop in P_0 or the differential tenotomy effect. Hematoxylin and eosin staining revealed no gross morphological changes that would suggest significant muscle fiber atrophy, necrosis, or inflammation in muscles undergoing AT or PT at any time period. This was confirmed by quantitative analysis of fiber size on a fiber type-specific basis (Table 2). Muscle architectural analysis similarly did not demonstrate any differences in mass, fiber type, length, or cross-sectional area between muscles which had undergone AT or PT. Previous work studying the effects of tenotomy has been

performed primarily in rat and rabbit soleus muscles, and has demonstrated increased intramuscular connective tissue, reduction in twitch tension, decreased capillary density, and muscle fiber necrosis.^{5,10,11} Time-dependent recapillarization was seen after tendon repair, and the muscle necrosis seen early after tenotomy was followed by restoration of normal histology over time.^{1,10} These changes suggest that either the muscle undergoes an injury and repair, or perhaps a remodeling and adaptation process after tenotomy, or both. In contrast to previous reports, we found no evidence of muscle injury to explain the decrease in P_0 in response to tenotomy. Thus, we have concluded that tenotomy (either AT or PT) induced a change in the muscle that interrupted the excitation-contraction coupling apparatus at some level, explaining at least the immediate changes (by day 1) in function we measured.

The mammalian neuromuscular junction is extremely plastic and able to respond to changes in innervation,⁹ development,⁴ and even mechanical injury. Thus, for example, acetylcholine receptor density has been shown to increase in response to mechanical injury to muscle.²⁶ We have preliminary evidence that alterations in neuromuscular interaction occurred in response to tenotomy. Neural cell

Table 2. EDL morphometric properties.

Experimental group	Fast fiber area (μm ²)	No. of fast fibers	Slow fiber area (μm ²)	No. of slow fibers
Active tenotomy (<i>n</i> = 9–12/group)				
1 day	2,715 ± 62	499	1,598 ± 58	95
7 days	2,520 ± 90	222	1,429 ± 30	66
21 days	2,215 ± 60	271	1,451 ± 31	65
Passive tenotomy (<i>n</i> = 9–11/group)				
1 day	2,444 ± 60	378	1,506 ± 36	117
7 days	2,971 ± 62	159	1,544 ± 41	53
21 days	2,384 ± 53	495	1,393 ± 31	134

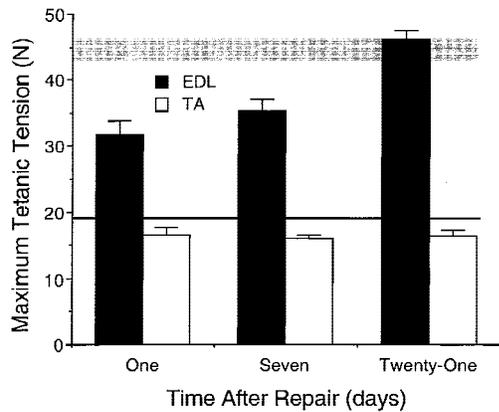


FIGURE 5. Maximum isometric tension generated by EDL muscles (filled bars) and TA muscles (open bars) 1, 7, or 21 days after length restoration of a muscle that had experienced passive tenotomy for 7 days. Thus, the time intervals depicted represent contractile testing 8, 14, and 28 days after the passive tenotomy. Stippled bar represents the mean \pm SEM of EDL maximum isometric tension from a previous study of age- and weight-matched rabbits.¹⁷ Solid line represents mean value for passive tenotomy after 7 days. Data shown are mean \pm SEM ($n = 6-7/\text{group}$).

adhesion molecule (NCAM) is an extracellular matrix protein that is thought to be involved in neuromuscular interaction and is expressed during development, after denervation, and during muscle regeneration. In a preliminary experiment utilizing tissue from this study, NCAM was markedly upregulated in muscles that underwent AT and PT.⁸ This neuromuscular junction interruption/dysfunction may serve a protective role in muscle to prevent activation/contraction if the distal tendon becomes detached. However, the details of such a mechanism remain to be determined.

Sarcomeres have a specific length at which actin and myosin filaments interact optimally to develop maximal tension. Previous work on rat soleus muscle demonstrated that, after tenotomy-induced muscle shortening, resting sarcomere length initially decreased, and subsequently increased by 4 weeks post-tenotomy, whereas the muscle length remained in its shortened state.² This demonstrated that “slackened” fibers can remodel over time by reducing sarcomere number in series. This is consistent with the serial sarcomere number decrease that can occur in muscles that are chronically shortened secondary to immobilization.²⁷ The muscle necrosis observed by other investigators after tenotomy was speculatively related to the remodeling necessary to optimize sarcomere length in the face of a chronically unloaded muscle.³

In our study, optimal muscle length for force generation (L_0) slowly approached the in situ length

(L_{is}) of the tenotomized shortened muscle. Early after tenotomy, sarcomeres were shortened and muscles had to be stretched approximately 7 mm to optimize sarcomere length (Fig. 4), whereas 21 days later, although the muscles remained shortened, the amount of stretch required to reach optimal sarcomere length decreased to only about 3 mm, suggesting muscle fiber remodeling occurred with a reduction of sarcomeres in series, presumably to reset the remaining sarcomeres to their optimal length for force generation. In this regard, the rabbit EDL appears to respond similarly to the rat soleus muscle after tenotomy³ although on a slower time-course. The time-course of this response may influence decisions regarding the optimal timing for performing tendon repairs. If a tendon repair is delayed until after a significant loss of sarcomeres occurs, returning the repaired tendon back to its original length may not be possible. Conversely, if a tendon repair can be performed after a tenotomized muscle has subtracted sarcomeres, it may be possible for the muscle to remodel by adding sarcomeres to optimize sarcomere length. There are certainly circumstances where sarcomere addition must occur, such as during normal growth and with limb lengthening (e.g., Ilizarov distraction techniques).

Muscle length restoration, 7 days after tenotomy (prior to loss of sarcomeres in series), caused a rapid partial recovery of force-generating capacity, followed by full recovery over the next 21 days. This rapid partial recovery as early as 1 day after length restoration is difficult to explain based on any known muscle injury and repair mechanism. As mentioned above, we suggest that neuromuscular junction modulation secondary to altered muscle load caused the early changes observed.

In summary, we conclude that tenotomy affected the associated muscle by causing a decrease in maximal tetanic tension mediated primarily by the excitation-coupling apparatus. There appeared to be a differential effect depending on whether the muscle was actively contracting or at rest during the tenotomy, with the former condition somehow endowing the muscle with a capacity for early partial recovery over the time-periods studied. Furthermore, over time after tenotomy, the muscle length at which maximal tetanic tension occurred (L_0) approached the tenotomized and shortened muscle in situ length (L_{is}), suggesting that prolonged muscle shortening resulted in remodeling by subtracting sarcomeres in series. Lastly, the detrimental effects of tenotomy on force generation were completely reversed by muscle length restoration, even if delayed by 7 days post-tenotomy.

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