SCIENTIFIC ARTICLE

Surgical Mobilization of Skeletal Muscles Changes Functional Properties—Implications for Tendon Transfers

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Purpose Tendon transfer surgery restores function by rerouting working muscle–tendon units to replace the function of injured or paralyzed muscles. This procedure requires mobilizing a donor muscle relative to its surrounding myofascial connections, which improves the muscle's new line of action and increases excursion. However, the biomechanical effect of mobilization on a donor muscle's force-generating function has not been previously studied under *in vivo* conditions. The purpose of this study was to quantify the effect of surgical mobilization on active and passive biomechanical properties of 3 large rabbit hind limb muscles.

Methods Myofascial connections were mobilized stepwise from the distal end to the proximal end of muscles (0%, 25%, 50%, and 75% of muscle length) and their active and passive length-tension curves were measured after each degree of mobilization.

Results Second toe extensor, a short-fibered muscle, exhibited a 30% decline in peak stress and 70% decline in passive stress, whereas extensor digitorum longus, a short-fibered muscle, and tibialis anterior, a long-fibered muscle, both exhibited similar smaller declines in active (about 18%) and passive stress (about 65%).

Conclusions The results highlight 3 important points: (1) a trade-off exists between increasing muscle mobility and decreasing force-generating capacity; (2) intermuscular force transmission is important, especially in second toe extensor, because it was able to generate 70% of its premobilization active force although most fibers were freed from their native origin; and (3) muscle architecture is not the major influence on mobilization-induced force impairment.

Clinical relevance These data demonstrate that surgical mobilization itself alters the passive and active force-generating capacity of skeletal muscles. Thus, surgical mobilization should not be viewed simply as a method to redirect the line of action of a donor muscle because this procedure has an impact on the functional properties of the donor muscle itself. *(J Hand Surg Am. 2021;46(4):341.e1-e10. Copyright © 2021 by the American Society for Surgery of the Hand. All rights reserved.)*

Key words Length–tension relationship, muscle architecture, muscle function, muscle mobilization, myofascial force transmission.

Online Exclusive

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Received for publication December 30, 2019; accepted in revised form September 10, 2020.

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

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0363-5023/21/4604-0017$36.00/0
https://doi.org/10.1016/j.jhsa.2020.09.017

Tendon transfer surgery restores lost joint function by transferring the distal tendon of a functional muscle from its native position into the tendon of a muscle with impaired function.[1–3] Lost function results from spinal cord injury, brachial plexus or peripheral nerve injury, cerebral palsy, stroke, and even direct injury to muscle–tendon units. Although advances have been made in tendon transfer surgery, the reproducibility of consistent and favorable outcomes in patients remains elusive.[4–8] One potential source of outcome variability is the lack of objective data that quantify the functional effects of mobilization on muscle function. Donor muscle mobilization is one important step in performing a tendon transfer because it optimizes the line of muscle action and increases muscle passive stretch potential, therefore improving donor muscle function.[9] However, muscle mobilization also risks disrupting neurovascular structures and changes the amount of internal shortening allowed in the muscle, both of which affect force production. Thus, mobilization must be used judiciously.[10–12]

Skeletal muscle architecture (the most important parameters of which are fiber length (L_f) and physiological cross-sectional area) is the most critical determinant of whole muscle function[13,14] and thus has been emphasized in the selection of donor muscles.[6,9] In general, surgeons try to select donor muscles with similar architectural properties to the impaired muscle to optimize post-transfer function. However, short-fibered muscles (for example, the flexor carpi ulnaris [FCU]) may be particularly susceptible to postmobilization changes in functional properties compared with long-fibered muscles (for example, the extensor carpi radialis longus [ECRL]) based on the fact that a greater amount of the fiber origin is disrupted in a short-fibered muscle with increasing mobilization. Although the effects of fasciotomy and mobilization have been examined for a single muscle,[12,15,16] the effect of surgical mobilization on short- and long-fibered muscles has not been tested experimentally. Therefore, the purpose of this study was to measure the biomechanical effects of mobilization on 3 muscles of varying architecture in an *in vivo* setting. We hypothesized that muscle architecture would determine the magnitude of the force-generating impairment effected by mobilization, specifically that short-fibered muscles would experience the greatest change in force as a result of to the greater number of fibers progressively disrupted by the mobilization procedure.

MATERIALS AND METHODS

Experimental procedure

Twelve male New Zealand White rabbits (mass, 2.7 ± 0.5 kg) were used in this experiment. Sample size varied among muscles because during surgical isolation and testing, some muscles (especially extensor digitorum longus [EDL]) were damaged and thus were unable to be physiologically activated. We chose tibialis anterior (TA) (n = 9), EDL (n = 5), and extensor digitorum of the second toe (EDII) (n = 12) muscles because of TA's long fibers (L_f = 38.08 mm) and the EDL's and EDII's short fibers (15.34 and 10.71 mm, respectively).[17,18] These muscles also have unique designs regarding the anatomical origin of their fibers and physiological function. Contractile properties were determined as previously described.[18,19] Anesthesia was induced with 5% isoflurane and maintained on 2% isoflurane. An anterior midline incision was made from mid-thigh to the ankle. The leg was immobilized with the hip and knee at 90° by Steinmann pins drilled through the femoral condyles and the tibial malleoli. After muscle isolation, 2 suture markers were placed at the distal and proximal myotendinous junctions to define muscle length (L_m) and define it at the neutral ankle joint. The distal tendon was transected and clamped to a servomotor at the distal myotendinous junction and aligned with the force-generating axis of the motor (Fig. 1). Muscles were then activated by either peroneal (TA and EDL) or tibial nerve (EDII) stimulation using a cuff electrode. Testing began at the L_m at which the hip and knee were at 90° and the ankle was at 0° extension; then, to create the isometric length–tension curve, L_m ranged from −25% L_f to +20% L_f in increments of 5% L_f. Passive force at each length was defined as the preactivation baseline force before stimulating to tetanic tension. Then, each muscle was mobilized relative to adjacent connective tissue, muscles, and bones from the distal end of the muscle at 25%, 50%, and 75% of the muscle's L_m (Fig. 1). Thus, 10 isometric contractions were elicited for each muscle at each level of mobilization, resulting in 40 contractile measurements per muscle. To mobilize the muscle from 25% to 75% of the L_m, surrounding connective tissue adjacent to muscles and bone was sharply divided.

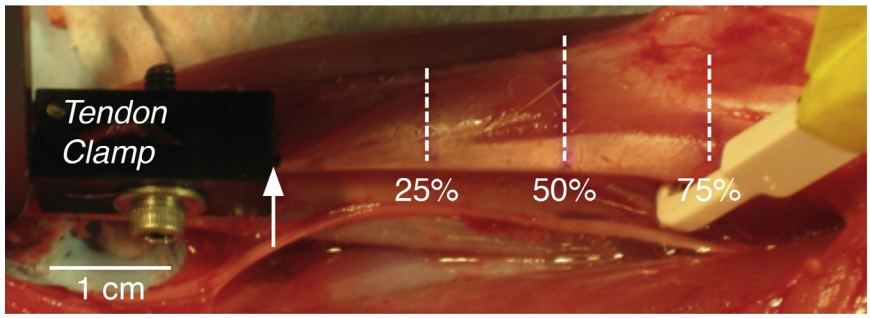

FIGURE 1: Experimental apparatus for EDII mobilization. Suture markers (arrows) were placed at the distal and proximal muscle–tendon junctions to define muscle length. After an initial nonmobilized length–tension curve was generated, myofascial connections to the tibia were sharply released to 25% of muscle length and a length–tension curve was re-measured. This process was then repeated for the 50% and 75% release distances.

Data analysis

Absolute active and passive force were converted to relative force by dividing measured force by peak active force achieved while maximally stimulating the nonmobilized muscle. This permitted quantitative comparison across the 3 muscles of different sizes. Similarly, L_m was normalized to optimal L_m (L_0) at 0% release to yield relative L_ms (Fig. 2 provides definitions). The passive length–tension curve was fit with a third-order polynomial, and passive force was defined arbitrarily as the force at $L_0 + 20\%$ relative to active force. Passive stiffness was obtained from the slope of the passive force–length relationship (Table E1). Contractile parameters for all 3 muscles were compared across degrees of mobilization using 2-way repeated-measures analyses of variance (release distance and muscle as grouping factors) with least significant difference post hoc tests. Sample size was chosen to achieve sufficient statistical power to detect a 10% change in maximum tetanic tension for the TA, the muscle least sensitive to release. Statistical power calculations were based on coefficients of variation in TA force reported previously using the same methods.[17–20] Significance was set to $P < 0.05$; data are presented as mean ± standard error of the mean.

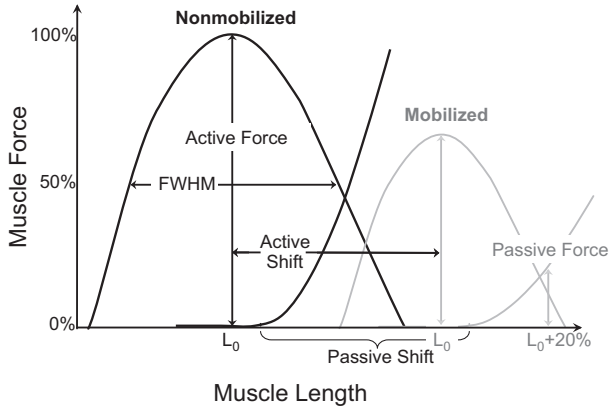

FIGURE 2: Schematic definition of variables used to quantify shape of the length–tension curve. Peak active force was measured at optimal length (L_0, the length at which peak force was developed) for both nonmobilized (black line) and mobilized (gray line) states. Passive force was quantified as the relative force measured at $L_0 + 20\%$ L_0 for all degrees of mobilization. Active shift was defined as the offset between L_0 at a given release distance and L_0. Passive shift was defined as the offset between slack length (defined as passive force just measurable above the noise level of the force transducer) of the nonmobilized and mobilized states. We chose FWHM as a measure of active muscle excursion and defined it as the width of the length–tension curve at 50% of the peak force at a given release distance.

RESULTS

All muscles demonstrated changes in functional properties as they were mobilized relative to their surrounding tissues. Mobilization clearly weakened all of the muscles, because they developed less active force after mobilization ($P < .05$). This is easily appreciated by viewing the averaged length–tension curves for each muscle plotted on a relative scale, because the curves decreased in height and increased in width as mobilization progressed (Fig. 3). Measured peak force decline was different among muscles ($P < .05$); TA and EDL showed a decline only after 75% release (about a 20% decrease in force; $P < .05$) (Fig. 4A), whereas the EDII showed a decline at 25% release that continued to decline at 50% and 75% release (total of about a 30% decrease in force; $P < .05$) (Fig. 4A). Statistical analysis by 2-

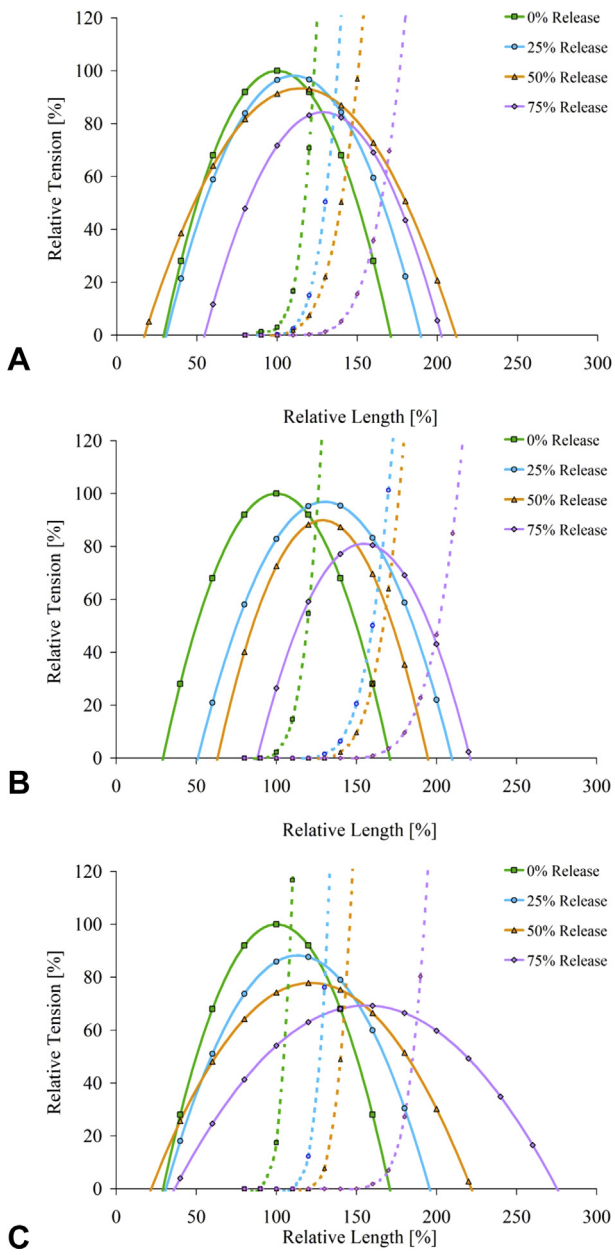

FIGURE 3: Averaged active and passive length−tension curves for the **A** TA, **B** EDL, and **C** EDII muscles. With increasing mobilization, peak active force declines, the slope of the passive length−tension curve declines, optimal and slack lengths increase, and the width of the length−tension curve increases. Data are from 5 to 12 independent experiments for each muscle. Error bars at each release distance are not shown, for clarity.

way analysis of variance revealed a muscle × mobilization distance interaction term ($P < .05$) explicitly demonstrating a muscle-dependent effect of mobilization.

Similar to active force, passive force declined with mobilization ($P < .05$) (Fig. 4B), but the magnitude of the effect was much greater compared with active force (about a 70% decline in passive force compared with a 20% decline in active force). This reduction was muscle-dependent ($P < .05$) and the relative decline in magnitude of passive force differed among muscles ($P < .05$) (Table E1) again with interaction ($P < .05$). The EDII displayed the highest premobilization passive force, which also declined to the greatest extent (Table E1).

The length at which active force was maximum (optimal L_m) shifted to longer L_m as each muscle was mobilized ($P < .05$); this length increase was muscle-specific ($P < .05$) (Fig. 4C). In addition, muscle slack length (beginning of the passive force curve) shifted to a longer length as mobilization progressed ($P < .05$), but this effect was not muscle-specific ($P = .22$) (Fig. 4D). For both active and passive shift, the magnitude of the length change was similar for all 3 muscles ($P = .13$ and $P = .59$, respectively; statistical power = 95%) (Table E1).

With increasing mobilization, the width of the length−tension curves quantified as full width at half maximum (FWHM) increased ($P < .05$) (Fig. 4E). This effect was muscle- and distance-specific (both $P < .05$).

As a positive control to confirm that our experimental methods did not damage either muscle or peripheral nerve, absolute peak stress for TA, EDL, and EDII was measured as 268.0 ± 11.2, 234.2 ± 33.4, and 229.4 ± 54.2 kPa, respectively, typical of mammalian muscle, and did not differ among muscles ($P = .82$; statistical power = 85%). Optimal L_m for TA, EDL, and EDII was 62.3 ± 8.1, 71.6 ± 8.9, and 58.4 ± 5.0 mm, respectively. The FWHM values for TA, EDL, and EDII were 22.6 ± 1.3, 10.5 ± 1.6, and 8.3 ± 0.5 mm, respectively, all of which agree with values reported previously by our laboratory as well as others.[20]

DISCUSSION

The purpose of this study was to determine the biomechanical effects of mobilizing a muscle relative to its surrounding bony and fascial investments. This approach is applicable to virtually any surgical approach or procedure in which a muscle is mobilized, divided, released, or transposed, such as tendon transfer, fasciotomy, step-cut lengthening, aponeurotomy, and rerouting after arthroplasty. These data demonstrate several important concepts. First, active and passive force-generating capacities decline, and force generation shifts to longer lengths as a muscle is increasingly mobilized relative to surrounding tissue (Figs. 3, 4).

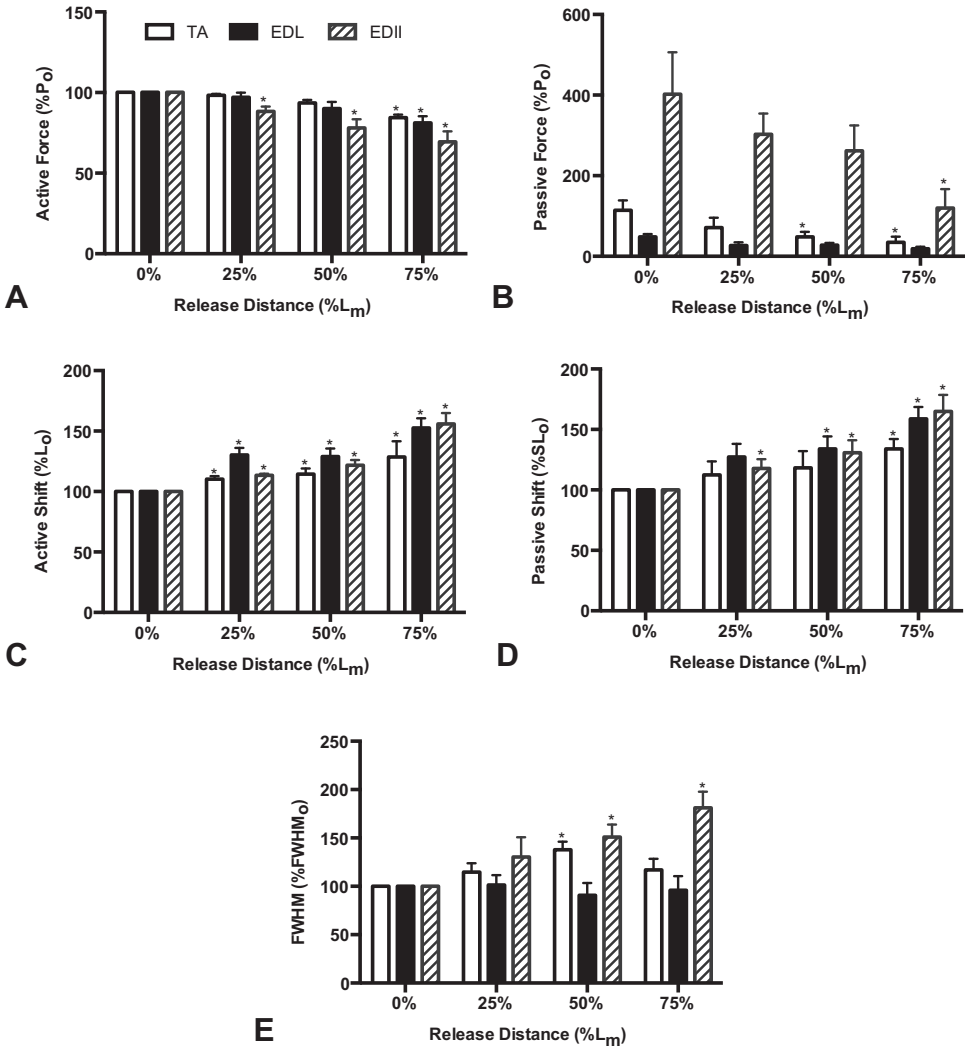

FIGURE 4: With mobilization, active and passive force declines, optimal and slack length shifts to longer lengths, and the width of the length−tension curve increases. **A** Peak active force, measured at L_0, and **B** passive force, measured at L_0 + 20% L_0, declined with release distance, with passive declining at a greater rate than active. The EDII experienced the largest decline in active and passive force; however, the decline in active force for TA and EDL was not different despite their architectural differences. **C** Optimal length (active shift) and **D** slack length (passive shift) increased with mobilization. **E** Width of the length−tension relation, quantified by FWHM, increased with release distance, highlighting the trade-off between excursion and muscle mobility. Data represent mean ± standard error of the mean (n = 5−12). Asterisks indicate difference from the nonmobilized state ($P < .05$).

Second, intermuscular force transmission in whole muscle is functionally important, as has been noted by others.[10,21−24] The EDII had a larger fraction of fiber attachments disrupted, which we believe explains the resulting greater reduction in active force-generating capacity relative to the other 2 muscles. However, the EDII was still able to generate more than 70% of its premobilized active force despite having 75% of its L_m freed from the bone (Fig. 4A), which demonstrates that force can still be transmitted from the fibers to the insertion tendon in this mobilized muscle.

Third, muscle architecture was not a major determinant of sensitivity to mobilization-induced impairment. Despite clear differences in the architecture between TA and EDL, their decline in active force was similar (Fig. 4A). Furthermore, the relative passive force decline did not correlate with architecture because all muscles lost more than 50% of their passive force when fully mobilized (Fig. 4B). Clinically, these data suggest that muscle mobilization impairs active force-generating capacity slightly (about 20%), but greatly reduces passive load-bearing properties of a muscle (about 70%). The implication

of this finding is that surgical methods that rely only on premeasurement and post-measurement of absolute whole L_m to determine the appropriate length at which to suture the distal tendon to the recipient site may be incorrect because the actual biomechanical properties of the muscle are altered by the surgical mobilization.

The 3 muscles chosen in the current study highlight the impact of different mechanisms of force transmission.[11] To initiate movement, forces generated through actin−myosin interactions must be transmitted outside the cell.[25] Two important pathways rely on the transmission of force from the muscle fiber to the tendon (myotendinous transmission) and from the muscle fiber to extracellular connective tissue (myofascial transmission). At a 75% release distance, most EDII fibers are no longer connected to their native origin, yet active force declines only 30%, which indicates that the force from freed fibers that are floating within the muscle tissue are still transmitted laterally through the connective tissue matrix to the distal tendon. This result is in agreement with previous reports and suggests a strong role of interfiber connections in force transmission for some muscles[11,21,22,24,26] and proposed force transmission pathways for muscles with fibers that do not span the length of the muscle.[22−24,27]

On the basis of muscle architectural differences alone (L_f/L_m ratio: TA = 0.67, EDL = 0.23, and EDII = 0.19),[17,18] we estimate that muscle mobilization relative to its attachments must exceed 67%, 23%, and 19% of L_m for TA, EDL, and EDII, respectively (ie, 1 L_f) to begin disrupting individual muscle fiber origins. Therefore, EDII would have the largest fraction of its muscle fibers disrupted at a given release distance compared with TA; the consequence is a greater decline in both active and passive force EDII with progressive mobilization. However, actively, EDII had a larger magnitude of decline over the range of release distances compared with either TA or EDL (Fig. 4A). In addition, EDII passive force declined precipitously at 50%, resulting in a difference between 0% and 75% release distance. The TA and EDL both demonstrated differences between 0% and 25% release distance, indicating an effect on passive force with minor mobilization (Fig. 4B). This is the opposite of our initial hypothesis and demonstrates that architecture alone is not the major predictor of force-generating impairment of a muscle after mobilization.

Human forearm architecture has been studied extensively.[3,14] The human equivalents to the EDII studied here are FCU and pronator teres, because both muscles have fairly short fibers.[14] However, we would posit that FCU, with its extensive longitudinal association with the ulna, would render it even more vulnerable to mobilization-induced functional changes because, like the EDII, the release distance may be much longer than the L_f in the distal two-thirds of the FCU (about 4 cm). At the other extreme, the human equivalents to the TA are brachioradialis (BR) and ECRL, because both the BR and ECRL have long L_f, (12 and 8 cm, respectively).[14] However, the BR is tightly associated with the radius and sufficient mobilization is required to reroute the muscle into a thumb flexor.[8] We thus suspect that BR may be more sensitive to mobilization-induced functional changes than ECRL.

The current study suggests that proximal muscle fiber attachments may be the most important predictor of force generation impairments observed after mobilization. A shaded polygon overlays the outline of the fiber origins for the 3 muscles studied in Figure 5. As EDII is mobilized relative to the tibia, the fiber origin is freed to a greater extent than the other 2 muscles, and thus apparently results in the greater decline in both active and passive force. Conversely, fiber origins for TA and EDL are only starting to be freed at 75% release distance, and thus most of their fibers are still attached to their fibrous origin. Recent 3-dimensional descriptions of muscle hold the promise of identifying the structural basis for these important concepts.[28,29]

Within the human forearm, mobilization of the BR muscle has been studied extensively,[7,8,30−32] but its active and passive biomechanical properties were not studied before and after mobilization. Increased width of a length−tension curve is associated with increased sarcomere length heterogeneity.[33] When the integrity of intramuscular connective tissue is damaged, muscles exhibit reduced forces and increases in the length range at which active force can be generated.[11,34] In this manner, disrupted connections may result in an increased distribution of mean sarcomere lengths across different fibers of the muscle, which results in a smoothing and widening of the length−tension curve.[12,16] In the current experiment, as EDII was mobilized, more of its fibers were mobilized compared with TA and EDL. Thus, we speculate that both the increase in the width of the length−tension curve and the reduction in peak force result from increased sarcomere length heterogeneity. This idea requires experimental verification.

Clinically, the greater passive force impairment compared with active force has implications for tendon transfer surgeries. Currently, methods

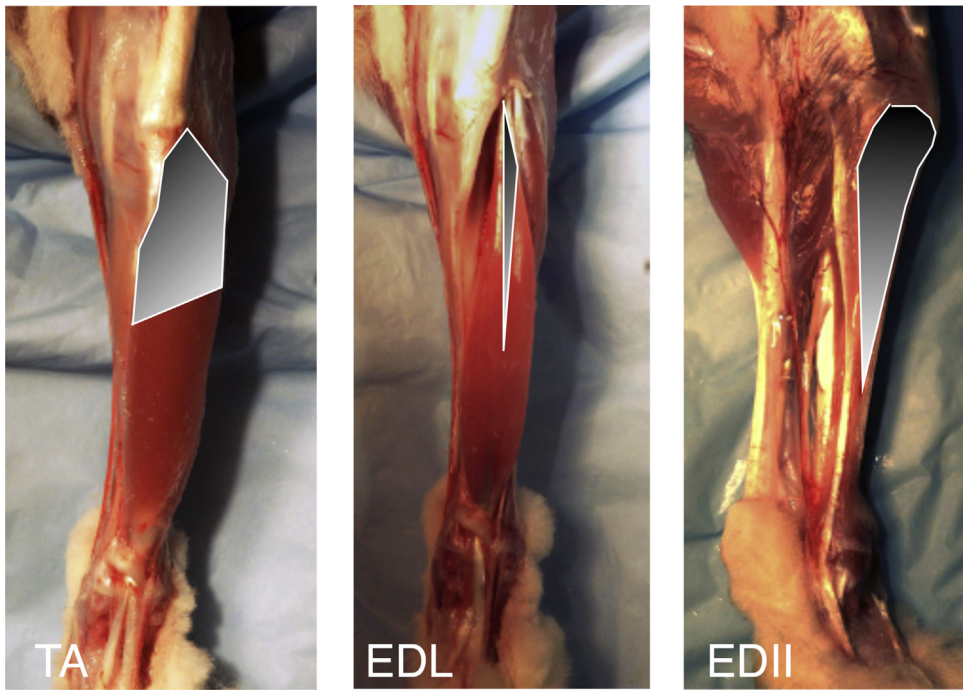

FIGURE 5: Fiber origin morphology influences passive force impairment. The TA does not have a clear proximal tendon, but rather a short broad origin deep on the lateral tibial condyle, the proximal lateral surface of the tibia, the interosseous membrane, and the proximal fibula. The EDL has a well-defined proximal tendon that arises from the femoral lateral epicondyle, crosses the knee to join with the muscle belly, and has a narrow strip that partially extends along the muscle length where the fibers originate from the tendon. Finally, the EDII is tightly secured to the medial aspect of the tibia through a well-defined fascial sheath, and the fiber origin spans most of the muscle length. Based on the experimental results, a quantitative measure of the number of fibers originating from each region of a muscle's origin may better predict the force generation impairments seen with mobilization for the 3 muscles studied compared to their muscle architecture.

clinicians use to choose the length at which the transferred muscle is set are relatively vague; surgeons often rely on the feel of a muscle to tension a transferred muscle appropriately.[5] If mobilization drastically reduces muscle passive force (Fig. 4B), the muscle may easily be stretched to supraphysiological lengths during tensioning. At these lengths, muscles generate less force (eg, FCU muscles placed at a relatively high passive force produced approximately 25% of the maximum active force[5]). Thus, the conventional wisdom that transferred muscles lose at least one strength grade[35,36] may be partially explained by the concurrent effects of muscle overstretching and a mobilization-based force decrease. After mobilization, adhesions are likely to occur, and increased passive force would be expected in addition to the acute loss of active muscle force. To avoid these 2 potentially negative effects of mobilization in tendon transfer surgery, unnecessary muscle manipulation and mobilization should be avoided. Surgeons are thus recommended to perform sufficient, but not excessive, muscle mobilization. By definition, sufficient mobilization is the point at which the desired excursion is obtained to perform the function of the muscle needing to be replaced. Our recommendation is to tailor each donor for sufficient excursion, maintaining fascial connections when necessary, sacrificing fascial connections only when sufficient muscle force is present, such as when transferring extensor digiti minimi to abductor pollicis brevis. In this case, little force is needed for this transfer, but motor positioning is crucial. Moreover, to maintain this function, early postoperative active and passive training of transferred muscles are required to prevent adhesion formation and secure a more predictable surgical outcome.[37]

This study had several limitations. First, to expose the EDL muscle for testing, the TA was removed, and thus EDL was not tested in its true *in vivo* state. However, EDL stresses were normal at L_0, which suggests that the muscle's force-generating capacity was not substantially altered before the experiment. Second, intramuscular and intermuscular connectivity was not quantified. These types of data would provide insight into the importance of the fiber origins and within- or between-muscle connections.

Physically defining the number and size of the fibers originating from the bone will be paramount to understanding how the origin of a muscle affects the force-generating impairment caused by mobilization. Third, despite differences in the origins and architecture of the current muscles, they do not follow the complex 3-dimensional trajectory that is common in some muscles, especially those with broad origins, such as the pectoralis major. Thus, the relationships presented in this study may not apply to more architecturally complex muscles. Fourth, this study was performed under isometric conditions. Extrapolation to dynamic conditions must be made with caution. Finally, these changes in force−length properties were measured acutely and may change over time with scarring or architectural adaptations such as sarcomere number change. No such changes were incorporated into the current study.

These data demonstrate that as a muscle is mobilized relative to surrounding connective tissue, active and passive force-generating capacity is dramatically altered, which highlights a trade-off between mobilizing a muscle to improve its excursion and line of action and resulting alterations in force-generating capacity. We found that the architecture of a muscle was not a strong predictor of muscle functional decline after mobilization. The anatomical topographical design of the muscle fiber origins may better predict the force decline a muscle exhibits after mobilization, but this idea requires further testing.

ACKNOWLEDGMENTS

We acknowledge grant support by the Department Veterans Affairs and National Institutes of Health/National Institute of Child Health and Human Development Grants HD048501 and HD050837. This work was supported in part by Research Career Scientist Award No. IK6 RX003351 from the US Department of Veterans Affairs Rehabilitation R&D Service. We thank Shannon Bremner for her expertise developing the LabView program, Alan Kwan, and Genaro Sepulveda for technical assistance.

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TABLE E1. Change in Biomechanical Variables as a Function of Release Distance[§]

Muscle	Dependent Variable	Slope (% per % Release)	Intercept (%)	Coefficient of Determination (R^2)
		Mean ± SEM	Mean ± SEM	Mean ± SEM
TA*	Active force	−0.18 ± 0.02	100 ± 0	0.86 ± 0.03
	Passive force	−1.05 ± 0.24	106.26 ± 30.53	0.83 ± 0.05
	Active shift	0.36 ± 0.05	100 ± 0	0.81 ± 0.04
	Passive shift	0.42 ± 0.05	100 ± 0	0.83 ± 0.07
	FWHM	0.40 ± 0.14	100 ± 0	0.44 ± 0.11
EDL†	Active force	−0.23 ± 0.04	100 ± 0	0.81 ± 0.06
	Passive force	−0.35 ± 0.11	43.47 ± 6.57	0.86 ± 0.07
	Active shift	0.72 ± 0.16	100 ± 0	0.81 ± 0.05
	Passive shift	0.77 ± 0.14	100 ± 0	0.80 ± 0.09
	FWHM	−0.08 ± 0.22	100 ± 0	0.70 ± 0.13
EDII‡	Active force	−0.42 ± 0.03	100 ± 0	0.83 ± 0.04
	Passive force	−3.56 ± 0.67	405.08 ± 61.05	0.82 ± 0.04
	Active shift	0.64 ± 0.09	100 ± 0	0.84 ± 0.04
	Passive shift	0.78 ± 0.10	100 ± 0	0.76 ± 0.07
	FWHM	1.07 ± 0.22	100 ± 0	0.62 ± 0.08

SEM, standard error of the mean.
§Values represent mean ± standard error for
*n = 9,
†n = 5, and
‡n = 12 animal subjects. Units for force are percent decline per percent release; units for shift are millimeters per percent release; and units for FWHM are percent unreleased FWHM per percent release.