TENDON BIOMECHANICAL PROPERTIES ENHANCE HUMAN WRIST MUSCLE SPECIALIZATION

Gregory J. Loren and Richard L. Lieber
Department of Orthopaedics and AMFS/Bioengineering, Biomedical Sciences Graduate Group, University of California and Veterans Administration Medical Centers, San Diego, U.S.A.

Abstract—Biomechanical properties of human wrist tendons were measured under loads predicted to be experienced by those tendons under physiological conditions. This was accomplished by measuring the architectural properties of the five prime wrist movers—extensors carpi radialis brevis (ECRB), extensor carpi radialis longus (ECRL), extensor carpi ulnaris (ECU), flexor carpi radialis (FCR), flexor carpi ulnaris (FCU)—and predicting their maximum tension (P₀) using a specific tension value of 22.5 N cm⁻². Loading the corresponding tendons to P₀ resulted in significantly different strain among tendons (p < 0.01) with the largest strain observed in the FCU (3.68 ± 0.31%) and the smallest strain observed in the ECRL (1.78 ± 0.14%). Further, strain magnitude was significantly positively correlated with the tendon length-to-fiber length ratio of the muscle-tendon unit, a measure of the intrinsic compliance of the muscle-tendon unit. Theoretical modeling of the magnitude of muscle sarcomere shortening expected based on the measured biomechanical properties revealed a maximum sarcomere length decrease of about 0.6 µm for the FCU to a minimum of about 0.2 µm for the ECRB at P₀. Thus, tendon compliance may, but does not necessarily, result in significant modification of muscle force generation. The significant variation in tendon biomechanical properties was not observed using traditional elongation-to-failure methods on the same specimens. Thus, the use of elongation-to-failure experiments for determination of tendon properties may not be reasonable when the purpose of such studies is to infer physiological function. These data indicate that muscle-tendon units show remarkable specialization and that tendon intrinsic properties accentuate the muscle architectural specialization already present.

Keywords: Tendons; Biomechanics; Muscle architecture; Motor control.

INTRODUCTION

Skeletal muscles exert their actions on bones via tendons. While the biomechanical properties of isolated tendons have been studied in detail (Butler et al., 1979, for review), only recently have muscle–tendon interactions been characterized. Hoffer et al. (1989) demonstrated in cats, during normal gait, as the paw contacted the ground and the muscle–tendon unit was forced to lengthen (i.e. during the yield phase of the step cycle), the tendon lengthened, while the muscle fibers actually shortened. Thus, muscle–tendon interactions may convey distinct functional properties on the muscle–tendon unit that may not be predicted based on knowledge of isolated muscle or tendon attributes. In a theoretical model, Zajac (1989) demonstrated tendon compliance increases the operating range of a muscle, suggesting that tendon is not merely an inert link between muscles and bones, and further that tendon compliance imparts specific properties to the muscle–tendon unit.

Skeletal muscles are highly specialized with regard to force generating properties. Muscles generate significantly different maximum tensions and act through significantly different ranges based primarily on different architectural design (Lieber et al., 1990; Powell et al., 1984; Sacks and Roy, 1982; Zajac, 1989). Given, then, that tendons can modify muscle characteristics beyond architecture alone, the extent to which contractile properties of skeletal muscles are modified by tendon compliance is unclear. Furthermore, whether architectural specialization is correlated in some way with tendon biomechanical properties is unknown.

It is not possible to address this question by simply applying published modulous and strain values obtained from traditional elongation-to-failure experiments. This is because tendons have a safety factor of from 5 to 30 (Ker et al., 1988) and are highly nonlinear in their biomechanical behavior (Butler et al., 1979). Extrapolation from such published ultimate stress and modulous values is thus not feasible.

In this report, we have studied the relationship between muscle architectural design and tendon biomechanical properties by loading the tendons over the range of physiological forces predicted to be generated by the muscles.

METHODS

Skeletal muscle architecture

Muscle architecture was determined according to the methods developed by Sacks and Roy (1982) as...
previously implemented (Lieber et al., 1990). Five cadaveric specimens were used, intact from the mid-humeral level and free of obvious musculoskeletal defects. The prime wrist movers—extensors carpi radialis brevis (ECRB), extensor carpi radialis longus (ECRL), extensor carpi ulnaris (ECU), flexor carpi radialis (FCR), flexor carpi ulnaris (FCU)—were isolated by dissection of the forearm. The muscle–tendon units \((n = 25)\) were removed and weighed. Muscle length \((L_m)\) was measured as the distance from the origin of the most proximal muscle fibers to the insertion of the most distal muscle fibers. Then, utilizing the \(L_d:L_m\) ratios previously measured in our laboratory, we calculated muscle fiber length \((L_d)\). Surface pennation angle \((\theta)\) with the muscle under zero tension was also taken from the previous study. Muscle length and fiber length were normalized to a sarcomere length of 2.5 \(\mu\)m using laser diffraction of fiber specimens in order to compensate for variations in limb position during fixation.

Muscle physiological cross-sectional area (PCSA) was determined according to the following equation (Sacks and Roy, 1982):

\[
PCSA (cm^2) = \frac{\text{Muscle mass (g) \cos \theta}}{\rho (g \cdot cm^{-3}) \text{ Fiber length (cm)}} \tag{1}
\]

where \(\rho = \text{muscle density (1.056 g cm}^{-3}, \text{Mendez and Keys, 1960)}\) and \(\theta = \text{surface pennation angle. Based on the fact that muscle PCSA is correlated with muscle maximum tetanic tension (\(P_{tet}\)), \(P_{tet}\) for each muscle was predicted by multiplying PCSA by a muscle specific tension of 22.5 N cm\(^{-1}\) (Close, 1972; Powell et al., 1974).

**Physiological tendon loading**

Muscle–tendon units were stored fresh-frozen. The muscle–tendon unit was divided at the junction of the aponeurosis and external tendon and the respective lengths measured with digital calipers to the nearest 0.1 mm. A 40 mm segment of external tendon was sectioned beginning 10 mm from the distal insertion. Care was taken to keep the specimen moist by bathing it in saline at all times. Using elastin stain, transverse dye lines were applied to the unloaded central tendon sectioned beginning 10 mm from the distal insertion. The tendon was placed in a 37°C saline bath, and clamped to the arm of a dual-mode servo-motor (Model 310B, Cambridge Technology, Inc., Watertown, MA) permitting controlled loading. The free end of tendon was secured to a stationary clamp yielding about 30 mm of exposed tendons between clamps.

A digital function generator (Model 3314A, Hewlett-Packard Co., Everett, WA) was programmed to drive the motor in five linear load–unload cycles to the \(P_b\) of the musculotendinous actuator tested. To minimize strain rate effects, load was imparted over a 30-s interval (0.017 Hz) and released over a consecutive period, with actual strain rates ranging from 0.05 to 0.14% s\(^{-1}\). Simultaneous force–time records were obtained at 0.1 s intervals via the servo-motor interfaced with a Macintosh IIx computer (Apple Computer, Inc., Cupertino, CA) using SuperScope software (version 1.0, GW Instruments, Inc., Somerville, MA). The experiment was video-recorded for subsequent strain analysis. Because of the 100 N mechanical load limitation of the motor, the FCU of arm specimen 5 with a predicted maximum force of 120 N was excluded from biomechanical examination.

The fourth force–deformation cycle was utilized for strain analysis using a video dimension analyzer (VDA; Model 303, PIM, Inc., San Diego, CA; Woo et al., 1980). The VDA signal was amplified by a factor of 100 and low-pass filtered at 10 Hz (Universal Amplifier 13–4615–58, Gould, Inc., Cleveland, OH) prior to computer acquisition. Each specimen was strain tracked three times from parallel regions of the tendon specimen with corresponding records averaged over time. From corresponding points on the load–time relationship and strain–time relationship, the load–strain curve was constructed (Woo et al., 1980).

**Tensile failure of tendons**

After physiological testing, the tendon specimen was mounted on a MMED systems apparatus with a 3000 lb load cell (Materials Technology Corporation, La Cañada, CA) and secured to a stationary platform using modified Cryo-Jaw clamp fixation and liquid carbon dioxide (Riermersa and Schamhardt, 1982); the interface strength was further augmented with cyanoacrylate adhesive. The tendons were preconditioned by cyclical deformation, 10 times at 3% strain as described by Woo et al. (1980); note that this was generally outside of the physiological range and deformed to failure at a strain rate of about 0.5% s\(^{-1}\).

**Tendon cross-sectional area determination**

Tendon area dimension was assessed using three methods: manual digital micrometry, saline displacement, and computed microscopic analysis following standard histological preparation. First, prior to specimen biomechanical testing, a digital caliper was used to determine the major \((a)\) and minor \((b)\) tendon diameters in three distinct and representative regions. Tendon cross-sectional area (CSA) was calculated as the average area of the three regions using the equation:

\[
CSA = \pi ab, \tag{2}
\]

where \(a\) and \(b\) represent the major and minor radii respectively. Tendon CSA was calculated again using approximately 3 ml of 0.9% NaCl solution at room temperature placed in a 5 ml graduated cylinder etched at 0.1 ml increments. Initial volume was recorded at the base of the meniscus to the nearest 0.05 ml. The tendon specimen was submerged in the cylinder after gentle blotting with gauze and the final volume recorded. Three displacement measurements were made, divided by specimen length, and averaged
to yield mean tendon CSA. Lastly, after transection of the tendon to experimental length, the distal fragment was fixed in 10% buffered formalin for 24–48 h at room temperature, serially dehydrated in alcohols, cleared, and infiltrated with paraffin under vacuum. The specimen was subsequently embedded in SurgiPath medium and sectioned to 6 μm perpendicular to the longitudinal axis. Three representative sections were selected for microscopic analysis using image 1 software (Universal Imaging Corporation, West Chester, PA) to calculate average tendon CSA. Pilot studies on fixed vs. frozen human wrist tendons documented an approximate 30% area shrinkage with histological preparation. Thus tendon microscopic CSA calculations were corrected by this factor.

The three procedures yielded comparable results. Variability was noted, however, in interval microscopy measurements likely reflecting the inherent variability of the biological tissue. Moreover, although image analysis was repeated in three histological sections, such sections only represented the region adjacent to the specimen to undergo biomechanical examination and thus may not have accurately represented the variability along the entire tendon length. Consequently, tendon CSA determined by saline displacement normalized to specimen length was used for stress calculations.

**Biochemical assay**

Following failure testing, the fractured tendon specimen was rehydrated in saline. Three repeat wet masses were obtained on an analytical balance after gentle blotting of the specimen and the mean hydrated mass for each tendon calculated. Specimens were subsequently quenched in isopentane cooled in liquid nitrogen and lyophilized at −20°C for 24 h. Three repeat dry weights were similarly obtained and the mean percent hydration determined.

A central 1 cm fragment was sectioned from the tendon specimen and then divided longitudinally for duplicate percent collagen assays by a modification of the automated method of Blumenkranz and Asboe-Hanson (1974).

**Muscle–tendon modeling**

To predict the magnitude of wrist muscle sarcomere shortening at the expense of tendon lengthening for a maximal muscle contraction, the theoretical model described by Lieber et al. (1992) was used. This model assumes that sarcomere length–tension and force–velocity properties are identical between muscles and uniform across the entire muscle. The model also assumes a finite time-course of cross-bridge attachment (Huxley, 1957), an ideal sarcomere length–tension relationship (Gordon et al., 1966) and an ideal force–velocity relationship (Edman et al., 1979; Katz, 1939) and was implemented as previously described with the following modifications:

1. The isometric sarcomere length–tension relationship was scaled to the filament lengths measured in human wrist muscles (Lieber et al., 1994). Average myosin filament length was 1.66 μm while average actin filament length was 1.30 μm. Using these filament lengths to construct a hypothetical length–tension curve yielded an optimal sarcomere length of 2.80 μm and a maximum sarcomere length for active tension development of 4.26 μm.

2. The sarcomere length–passive tension relationship was modified from the relationship obtained by Lieber et al. (1991) for frog muscle by shifting the sarcomere length at which tension is zero from 2.2 to 2.8 μm so as to align sarcomere length at zero tension with optimal sarcomere length (see also Zajac, 1989).

3. Muscle fiber length, tendon and aponeurosis lengths (external and internal tendon in Zajac's (1989) terminology) for each muscle (Table 1) as well as the specific compliance function for each muscle (Fig 1) were substituted for the analogous values in the frog model.

The logical program flow proceeded as follows: Following initialization of experimental parameters such as connective tissue material properties, sarcomere length and muscle–dimensional quantities, resting tendon and muscle fiber lengths were calculated using experimentally obtained load–strain values (Fig. 1). The muscle was then “activated” according to the data of Huxley (1957). As the muscle developed tension appropriate to the sarcomere length specified, a certain amount of tendon tension resisted sarcomere shortening according to the measured load–strain values. This tension was compared to the isometric tension for that sarcomere length and level of activation to determine the relative isometric tension. Using the force–velocity relationship (Katz, 1939), sarcomere shortening velocity was then calculated and the new sarcomere length calculated.
Table 1. Measured properties of muscles and tendons*

<table>
<thead>
<tr>
<th>Parameter</th>
<th>ECRB</th>
<th>ECRL</th>
<th>ECU</th>
<th>FCR</th>
<th>FCU</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Muscle properties</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Muscle length (mm)</td>
<td>186.4</td>
<td>155.3</td>
<td>209.9</td>
<td>192.8</td>
<td>220.6</td>
</tr>
<tr>
<td>Fiber length (mm)</td>
<td>70.8</td>
<td>127.3</td>
<td>58.8</td>
<td>59.8</td>
<td>41.9</td>
</tr>
<tr>
<td>Physiological CSA (mm²)</td>
<td>240.1</td>
<td>130.0</td>
<td>210.0</td>
<td>211.9</td>
<td>363.6</td>
</tr>
<tr>
<td>Predicted maximum tetanic tension (N)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tendon properties</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aponeurosis length (mm)</td>
<td>101.3</td>
<td>182.1</td>
<td>264.1</td>
<td>215.1</td>
<td>153.7</td>
</tr>
<tr>
<td>External tendon length (mm)</td>
<td>102.7</td>
<td>61.4</td>
<td>201.6</td>
<td>230.3</td>
<td>126.5</td>
</tr>
<tr>
<td>Total tendon length (mm)</td>
<td>204.0</td>
<td>153.7</td>
<td>336.1</td>
<td>363.6</td>
<td>160.6</td>
</tr>
<tr>
<td>Tendon length : fiber length ratio</td>
<td>2.89</td>
<td>3.89</td>
<td>3.86</td>
<td>4.96</td>
<td></td>
</tr>
<tr>
<td>Tendon CSA (mm²)</td>
<td>14.6</td>
<td>14.2</td>
<td>15.7</td>
<td>17.7</td>
<td></td>
</tr>
<tr>
<td>Tendon stress at P₀ (MPa)</td>
<td>4.06</td>
<td>2.30</td>
<td>3.36</td>
<td>3.06</td>
<td></td>
</tr>
<tr>
<td>Tendon strain at P₀ (%)</td>
<td>1.99</td>
<td>1.78</td>
<td>2.35</td>
<td>2.48</td>
<td></td>
</tr>
<tr>
<td>Modulus at P₀ (MPa)</td>
<td>726.1</td>
<td>438.1</td>
<td>595.4</td>
<td>548.0</td>
<td></td>
</tr>
<tr>
<td>Ultimate stress (MPa)</td>
<td>71.3</td>
<td>67.9</td>
<td>74.0</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tangent modulus (MPa)</td>
<td>904.7</td>
<td>604.1</td>
<td>857.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Safety factor (× P₀)</td>
<td>18.0</td>
<td>31.8</td>
<td>21.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Biochemical properties</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hydration (% dry mass)</td>
<td>77.0</td>
<td>74.4</td>
<td>80.3</td>
<td>79.3</td>
<td></td>
</tr>
<tr>
<td>Collagen (% dry mass)</td>
<td>77.0</td>
<td>78.4</td>
<td>79.6</td>
<td>74.0</td>
<td></td>
</tr>
</tbody>
</table>

*Values shown are mean ± standard error of n = 5 independent measurements; *significance level from one-way ANOVA.
†Signifies n = 4.

Abbreviations: ECRB, extensor carpi radialis brevis; ECRL, extensor carpi radialis longus; ECU, extensor carpi ulnaris; FCR, flexor carpi radialis; FCU, flexor carpi ulnaris; CSA, cross-sectional area; P₀, muscle maximum tetanic tension. *p < 0.05; **p < 0.01; ***p < 0.001; ****p < 0.0001.
Physiological behavior of human wrist tendons

795

(based on the sarcomere shortening velocity and time interval of 0.01 ms). This process was repeated in 0.01 ms increments and sarcomeres continued to shorten until muscle contractile force was equivalent to the resistive force of the connective tissue.

This program was written in FORTRAN in the Macintosh Programming Workshop (MPW) programming environment using the MPW SADe symbolic debugger program. (MPW 3.2.3, Apple Computer, Inc., Cupertino, CA.)

Statistical analysis

Biomechanical parameters for individual tendon samples were analyzed by one-way analysis of variance (ANOVA). Multiple paired comparisons between tendons were performed post-hoc using Fisher's protected least-squares difference method. In addition, tendons were grouped as flexor or extensor and as radial deviator or ulnar deviator; such groups were compared by two-way ANOVA. Simple regression was used to determine the significant interrelationships between morphological and functional characteristics. Stepwise regression was used to distinguish which of the many independent variables measured (Table 1) had the greatest influence on sarcomere shortening (the dependent variable). F-to-enter was set to 4.000 and F-to-remove 3.996. Data were analyzed using StatView 4.0 software (Abacus Concepts, Inc., Berkeley, CA). Significance level (α) was selected as 0.05; statistical power (1-β) exceeded 65% for all parameters evaluated. Data are expressed as mean ± SE unless otherwise noted.

Exponential curves for the individual load-strain and stress-strain relationships were obtained in the form \( y = ae^{bx} \) with correlation coefficients exceeding 0.76 for all data sets.

RESULTS

Muscle architecture and actuator morphology

Musculotendinous actuator properties were similar to those reported previously (Lieber et al., 1990, Table 1). Maximum tetanic tension for all muscles was predicted based on the relationship established between PSCA and \( P_0 \) (Powell et al., 1984). Predicted maximum tetanic tension (\( P_0 \)) of FCU (89.1 ± 8.4 N) was significantly greater than \( P_0 \) of other wrist muscles (p < 0.0005) while that of ECRL (31.9 ± 2.7 N) was significantly less (p < 0.05). Moreover, the FCU tendon CSA was significantly larger (27.4 ± 3.6 mm²) than other wrist tendons (range 14.2 ± 0.5–17.7 ± 1.6 mm²), in effect normalizing the maximum physiological stress imposed on tendon groups (wrist flexors 3.31 ± 0.40 MPa, extensors 3.74 ± 0.24 MPa).

Wrist extensor muscle fibers were significantly longer than the fibers of flexors (p < 0.0005), while the radial muscles had significantly longer fibers than the ulnar deviators (p < 0.0005) as previously reported (Lieber et al., 1990). Total tendon length (\( L_t \)) was comparable among tendon groups, though ECRL had a significantly longer tendon than others (p < 0.05, Table 1). Given the inherent tendon material compliance, both the relative tendon stiffness and the tendon length:fiber length ratio (\( L_t: L_f \)) of a muscle–tendon unit influence the compliance of a musculotendinous actuator. A greater \( L_t: L_f \) ratio results in an increased intrinsic compliance of the system. Wrist flexor units and ulnar deviator units were noted to have a greater intrinsic compliance than the extensor and radial groups based on this ratio (p < 0.0001).

Physiological load–strain relationships of prime wrist movers

Under physiological loads, the relationship between load and strain was characterized by a curve with progressively increasing slope and no clear demarcation between toe and linear regions as is evident in deformation-to-failure experiments (Fig. 1). At \( P_0 \), wrist flexor tendons strained significantly more than extensor tendons (p < 0.005) while tendons acting to deviate the wrist ulnarly strained more than those acting radially (p < 0.01; Fig. 2). Specifically, FCU strained 3.68 ± 0.31% at \( P_0 \), more than any other wrist tendon (p < 0.05).

Normalization of load to stress and determination of tendon tangent modulus at \( P_0 \) from individual exponential curve fits revealed no significant difference among tendons (p > 0.2). Thus, the slopes of the stress–strain relationships of the tendons were comparable at maximum tetanic tensions, yet notable variability was apparent at tensions less than \( P_0 \) (Fig. 1). Normalization of the load–strain relationship to tendon CSA did not appreciably diminish the variability noted in physiological tendon behavior, suggesting materials dissimilarities between tendons at low forces.

Physiological strain was significantly positively correlated with tendon CSA (p < 0.01, \( r^2 = 0.3 \)).

Fig. 2. Average strain at muscle \( P_0 \) for tendons grouped by function. Open bars represent radial muscles while filled bars represent ulnar muscles. Data were obtained from load-strain relationships similar to those shown in Fig. 1. Two-way ANOVA revealed a significant difference between strain in flexors vs extensors (p < 0.005) and ulnar- vs radial deviators (p < 0.01) with no significant interaction (p > 0.2).
These data indicate that the larger tendons strain to a greater extent in vivo and are in contrast to the premise of Ker et al. (1988) that increased tendon CSA protects against increased strain in musculotendinous actuators which generate high forces. As previously noted by An et al. (1991), muscle PCSA correlated significantly with tendon CSA (p < 0.0005, r² = 0.46).

Tensile failure relationships

No significant differences were noted in biomechanical properties of human wrist tendons deformed to failure. Ultimate stress (range 51.6 ± 9.3–74.0 ± 13.5 MPa, p > 0.4), ultimate deformation (range 11.4 ± 1.0–16.6 ± 1.7%, p > 0.3), and tangent modulus in the linear region (i.e. forces greater than P₀ and less than ultimate force; range (54.0 ± 15.7–107.6 ± 13.1 MPa, p > 0.1) did not vary significantly between tendons. Such findings support a common structure–function relationship of tendons at supramaximal muscle forces.

The fact that physiological strain averaged approximately 2.5% for the different tendons but ultimate deformations corresponded to strains of about 14% indicates that, at in vivo loads, tendons of the human wrist operate within the toe region of this curve. Indeed, the tangent moduli of individual tendons at P₀ were significantly less than the moduli in the linear region of the stress–strain curve (at higher forces; p > 0.005), lending further support to this conclusion. In addition, the safety factors did not vary significantly among flexors and extensors or radial and ulnar deviators; ECRL, though, was noted to have a greater safety factor (31.8 ± 4.4 × P₀) than other tendons (range 16.8 ± 5.2 × P₀–23.7 ± 2.7 × P₀), likely due to the relatively low maximum muscle tension.

Biochemical composition of tendons

Wrist flexor tendons were composed of a significantly lower percentage of collagen than wrist extensors (p < 0.05; Table 1), while no appreciable difference was noted among radial and ulnar deviators. This finding was anticipated based on the higher strain in flexor compared to extensor tendons. The significantly greater tendon CSA of wrist flexor tendons (p < 0.0005) thus cannot be attributed solely to an augmented collagen component. In fact, collagen percentage negatively correlated with tendon CSA (p < 0.01, r² = 0.28). Furthermore, no correlation between percent collagen and strain was evident (p > 0.9, r² < 0.0001). Since in vivo tendons operate in the toe region of the stress–strain relationship, presumably the geometrical configuration of collagen fibrils rather than the relative collagen content determines tendon behavior at physiological loads.

Tendon hydration may be useful as an index of ground substance given the hydrophilic nature of proteoglycan. FCU had a significantly greater water content than both radial extensor tendons (p < 0.05), Physiological strain, moreover, varied significantly as percent hydration (p < 0.05, r² = 0.2). Consequently, variations in tendon hydration or possibly matrix composition may contribute to the individual biomechanical behaviors of tendons at physiological loads.

Muscle–tendon model simulation

The variable considered to have the greatest physiological importance resulting from tendon compliance was maximum sarcomere shortening. Because of the differing biomechanical properties and tendon and fiber lengths, the magnitude of sarcomere shortening permitted was significantly different between muscle–tendon units (p < 0.0001; Fig. 3). Sarcomere shortening between all pairs of muscle–tendon units was significantly different (p < 0.05) with the exception of the ECRB and ECRL (p > 0.4) and the FCU and FCR (p > 0.1). Sarcomere shortening magnitude ranged from about 0.2 μm for the ECRL to about 0.6 μm for the FCU. Given sarcomere lengths of myosin and actin filaments of 1.66 and 1.3 μm respectively (Lieber et al., 1994), this magnitude of sarcomere shortening could result in muscle force changes of 10–50% P₀, depending on the range over which the sarcomere shortened.

The best predictor of sarcomere shortening was tendon strain at P₀ (F-to-enter = 100.7) which accounted for 81% of the experimental data variability. The only other variable to enter the stepwise regression equation was Lₙ; Lₐ ratio (initial F-to-enter = 44.7; F-to-enter after step 1 = 24.3) which then accounted for an additional 11% of the experimental variability. Thus, the multiple regression equation resulting from statistical analysis was

Maximum sarcomere shortening (μm) =

- 0.149 (% strain at P₀)
  + 0.088(Lₙ; Lₐ)
  + 0.182,

which had a multiple correlation coefficient of 0.92 (p < 0.0001).

Fig. 3. Average sarcomere shortening predicted given muscle–tendon unit architectural properties and tendon biomechanical properties. The best predictor of sarcomere shortening was tendon strain at P₀.
DISCUSSION

The purpose of this investigation was to define the biomechanical properties of the prime wrist tendons under physiological loads predicted based on muscle architectural properties and a theoretical model of muscle–tendon activation (see methods). Our goal was to extend our previous study of muscle architecture to an understanding of muscle–tendon interaction in the wrist.

The advantages of tendon straining under physiological loads are not intuitively apparent. Indeed, tendon deformation may impair the ability of a muscle–tendon unit to displace a joint, theoretically requiring an increased muscle fiber length to restore mechanical capability. However, by incorporating tendon compliance, the operating range of the system may be increased if that muscle is operating on its ascending limb. Conversely, if the muscle operates on its descending limb, tendon compliance will decrease its operating range. Currently, there are no data available which describe the normal operating range of the human wrist muscles and thus the underlying consequences of tendon compliance cannot be unambiguously determined.

Tendon serves as an elastic component in series with a contractile component or muscle fiber. As a muscle develops force, then, a segment of length change may be taken up by the tendon rather than the muscle fiber. This in effect skews the sarcomere–length tension relationship, allowing sarcomeres on the ascending limb to shorten onto the plateau of the curve; we predict muscle force changes of up to 50% $P_0$ depending on the range over which the sarcomeres shorten. Tendon strain as well as the relative tendon length determines the magnitude of increase in the operating range of the muscle–tendon unit.

A second consequence of tendon compliance is to act as a length buffer—preventing joint rotations imposed on the muscle–tendon unit from being directly transduced as length changes by the skeletal muscle. The FCU tendon, for example, when subjected to a load equivalent to its maximum isometric tension (9 kg; Table 1) will extend 7.4 mm which corresponds to a wrist rotation of 29° (Jacobson et al., 1993). Similarly, FCR tendon stretch at peak isometric tension accounts for a 20° joint rotation, while equivalent deformations of ECRB, ECRL, and ECU tendons correspond to 11°, 16°, and 36° of wrist rotation, respectively. This buffering effect is emphasized in compliant actuators, improving the neuromuscular system's ability to maintain constant force despite external length perturbations, as discussed by Rack and Ross (1984). In contrast, relatively stiff actuators have improved sensitivity to extrinsic joint rotations providing precise control of joint position.

We found that tendon properties do not simply scale with muscle architectural properties. Rather, they act to add further specialization to the muscle–tendon unit. For example, the FCU muscle, with its short fibers ($L_f = 41.9$ mm) is arranged in series with a long length of tendon and aponeurosis ($L_t = 207.6$ mm) which renders it the most compliant of the wrist joint actuators ($L_f: L_t = 4.96$; Table 1). The FCU tendon is also the most materially compliant (strain at $P_0 = 3.68\%$) which indicates that the biomechanical properties of the tendon accentuate the already compliant nature of the musculotendinous actuator. In contrast, ECRL has long fibers ($L_f = 127.3$ mm) and, though in series with a long tendon and aponeurosis ($L_t = 264.1$ mm), is the most intrinsically noncompliant of the wrist muscle–tendon units ($L_f : L_t = 2.10$; Table 1). Under physiological loads, however, the ECRL tendon is quite stiff (strain at $P_0 = 1.78\%$); thus the intrinsic noncompliance of the musculotendinous actuator is enhanced by the incorporation of a stiff tendon. Such findings were unexpected based either on our previous frog studies (Lieber et al., 1991; Trestik and Lieber, 1993) or based on reviews by Zajac (1989) and Ker et al. (1988). This general relationship is the rule for the wrist actuators since $L_f : L_t$ ratio was positively correlated with tendon strain at $P_0$ (Fig. 4).

The best predictor of sarcomere shortening was tendon strain at $P_0$ (Fig. 2). We initially expected that $L_f : L_t$ ratio would be the best predictor since the relative amount of tendon in series with a given number of sarcomeres is directly related to the absolute sarcomere shortening magnitude. However, in this study, because tendon compliance accentuates the intrinsic muscle–tendon actuator properties, strain at $P_0$ is actually a better predictor of sarcomere shortening. It should be noted, as pointed out by Zajac (1989), that $L_f : L_t$ ratio is a good predictor of sarcomere shortening, but not the best.

In muscles such as FCU with short fibers and thus low amplitude contractions and a limited active range, augmenting the intrinsic compliance by incorporation of a compliant tendon serves to maximize
the operating range. In contrast, muscles with long fibers like ECIR and ECRL maintain an ample active contractile range and thus may not benefit from a compliant tendon. Most power activities of the human wrist are performed in the flexed and ulnar position; by design, then, muscle force and operating range appear to be emphasized. Precision and control of motion are accentuated in the radial extensors perhaps for manipulative tasks.

Interestingly, physiological strains did not correlate with collagen percentage which may indicate that the structural basis for the increased compliance is the geometrical configuration of collagen fibrils. The wavy nature of collagen fibers at low tension and the alignment of fibers at greater force have been described (see Vidik, 1973, for review). Consequently, the toe region relevant to physiological forces corresponds to the gradual straightening of the collagen fibers and thus is influenced by fiber shape and surrounding matrix. Conversely, at higher loads, the linear region of the stress–strain relationship and ultimate tensile strength reflect the number of collagen fibrils and are thus influenced by relative collagen content.

Lanir (1978) modeled fiber–matrix interactions affecting tendon behavior at low tensions. Such relations are dominated by the bulk tissue properties of the matrix and the amplitude and periodicity of collagen fibers. With consideration of our data, specifically the variability in load–strain relationships at submaximal tensions, it is likely that such parameters have a greater influence on physiological behavior than relative collagen concentration.

These data also argue against the notion that the connective tissue complex remodels itself like bone, which regulates trabecular orientation and density to maintain constant strain despite force variation. Ker et al. (1988) proposed that increased tendon cross-sectional area protects against increased strain in musculotendinous actuators which generate high forces; we, however, have demonstrated the converse with increased physiological strain noted in larger tendons. Tendon cross-sectional area was found to vary with muscle physiological cross-sectional area as demonstrated by An et al. (1991). This relationship may be inherent to efficient force coupling with each contractile unit connected to one tendon fiber.

Finally, the varying biomechanical behavior of the prime human wrist tendons were apparent only under physiological loads and were not manifest at supramaximal muscle forces. Therefore, the use of traditional deformation-to-failure experiments for determination of tendon properties may not be appropriate when the purpose of such studies is to infer physiological function.

Acknowledgements—The authors thank Stanley Lau for technical assistance. We also thank Dr Cy Frank and Donna Mc Donald (University of Calgary) for performing the percent collagen assays. This work was supported by the Veterans Administration and NIH grant AR35192.

REFERENCES

