J Appl Physiol 94: 1092–1107, 2003.
First published November 27, 2002; 10.1152/japplphysiol.00173.2002.

Myofascial force transmission: muscle relative position and length determine agonist and synergist muscle force

PETER A. HUIJING[1,2] AND GUUS C. BAAN[1]
[1]*Instituut voor Fundamentele en Klinische Bewegingswetenschappen, Faculteit Bewegingswetenschappen, Vrije Universiteit, 1081 BT Amsterdam; and* [2]*Integrated Biomedical Engineering for Restoration of Human Function, Instituut voor Biomedische Technologie, Faculteit Construerende Technische Wetenschappen, Universiteit Twente, 7522 NB Enschede, The Netherlands*

Submitted 4 March 2002; accepted in final form 24 October 2002

Huijing, Peter A., and Guus C. Baan. Myofascial force transmission: muscle relative position and length determine agonist and synergist muscle force. *J Appl Physiol* 94: 1092–1107, 2003. First published November 27, 2002; 10.1152/japplphysiol.00173.2002.—Equal proximal and distal lengthening of rat extensor digitorum longus (EDL) were studied. Tibialis anterior, extensor hallucis longus, and EDL were active maximally. The connective tissues around these muscle bellies were left intact. Proximal EDL forces differed from distal forces, indicating myofascial force transmission to structures other than the tendons. Higher EDL distal force was exerted (ratio ≈118%) after distal than after equal proximal lengthening. For proximal force, the reverse occurred (ratio ≈157%). Passive EDL force exerted at the lengthened end was 7–10 times the force exerted at the nonlengthened end. While kept at constant length, synergists (tibialis anterior + extensor hallucis longus: active muscle force difference ≈ −10%) significantly decreased in force by distal EDL lengthening, but not by proximal EDL lengthening. We conclude that force exerted at the tendon at the lengthened end of a muscle is higher because of the extra load imposed by myofascial force transmission on parts of the muscle belly. This is mediated by changes of the relative position of most parts of the lengthened muscle with respect to neighboring muscles and to compartment connective tissues. As a consequence, muscle relative position is a major codeterminant of muscle force for muscle with connectivity of its belly close to in vivo conditions.

anterior crural compartment; connective tissue; length-force characteristics; muscle proximo-distal force difference

LENGTH IS GENERALLY RECOGNIZED as one of the major determinants of force exerted by muscle. As a consequence, since Blix (4–7) reported his experiments more than a century ago, isometric length-force characteristics have been the subject of extensive research. Work was performed at all levels of organization, i.e., muscle (e.g., Refs. 8, 14, 16, 35, 44, 45, 49, 55, 57, 58, 72), small muscle fascicles (e.g., Ref. 75), and single-muscle fibers (e.g., Refs. 17, 33, 34, 36, 48, 56).

In most of such work, even if performed in situ, the muscular elements of interest were experimented on after substantial dissection to free it as much as possible from surrounding connective tissues and other structures. Force exerted at one end of the fiber, fascicle, or muscle was measured, based on the mostly implicit assumption that force exerted at either end would be equal.

For in situ rat muscle, with most of its connective tissue at its belly intact, we recently showed that this is predominantly not the case (25, 28, 39). The size of any difference in force (ΔF) exerted at proximal and distal tendons of EDL muscle was shown to be quite dependent on the actual length of the muscle tendon complex (25, 28, 39) (i.e., $-22\% < \Delta F < +22.5\%$ and $0\% < \Delta F < +20.0\%$, respectively). In addition, blunt dissection of intermuscular connective tissue, as well as full compartmental fasciotomy, did alter muscle length-force characteristics (27, 59). These effects are explained in terms of myofascial force transmission from muscle i.e., transmission by other paths than the myotendinous ones. In most of the studies referred to in this paragraph, the effects were studied in either proximal lengthening of rat extensor digitorum longus (EDL) muscle or distal lengthening of its synergists, i.e., tibialis anterior (TA) and extensor hallucis longus (EHL) muscles exclusively.

EDL is a unipennate muscle with one proximal aponeurosis from which all of its muscle fibers originate. The insertions of these muscle fibers are divided over four distal aponeuroses. Therefore, EDL is distally a multitendon muscle with connections to digits II–IV (e.g., Refs. 1, 9). EDL extends the toes and is also a dorsal flexor of the ankle. In rat, the unique proximal EDL tendon crosses the knee joint (knee extensor). Such arrangement allows experimental access to the EDL proximal tendon without dissecting the anterior crural compartment. Having access to both proximal and distal tendons allows convenient simultaneous proximal and distal force measurement. It also allows

Address for reprint requests and other correspondence: P. A. Huijing, Faculteit Bewegingswetenschappen, Vrije Universiteit, Van de Boechorststraat 9, 1081 BT Amsterdam, The Netherlands (E-mail: P_A_J_B_M_Huijing@fbw.vu.nl).

The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked "*advertisement*" in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

changes in muscle-tendon complex length to be easily imposed on EDL at either end of the muscle. Short intermuscular connective tissue connects the muscle belly of EDL to that of TA and EHL. TA completely surrounds EDL as well as EHL. TA has a very complex architecture, with muscle fibers originating both from bony origins and from a proximal aponeurosis and inserting on a distal tendon. TA is predominantly an ankle dorsal flexor. All muscle fibers of EHL originate from the anterior intermuscular septum and insert on a distal aponeurosis that is connected to a tendon, which is in very close proximity to the TA tendon, before passing to the big toe. As these muscles do not share aponeuroses, but do have connective tissue links to each other and to the compartment boundaries, they are very useful for studying myofascial force transmission.

In the present work, we studied the effects of symmetric lengthening of rat EDL to identical muscle-tendon complex lengths. Identical target muscle-tendon complex lengths were reached by lengthening EDL at its proximal or at its distal tendon before isometric contraction. The effects on isometric force exerted at each of the proximal and distal EDL tendons and on the expected proximodistal EDL ΔF are studied. In addition, the effects of such symmetric lengthening of EDL to identical muscle-tendon complex lengths on neighboring synergists (TA+EHL) are considered as well.

The principal aims of the present work are to compare the effects of equal proximal and distal lengthening of EDL muscle and test the hypotheses that such effects of equal lengthening on *1*) proximal and distal force exerted at the EDL tendons and on *2*) the expected EDL proximodistal ΔF are equal in magnitude and thus exclusively a function of muscle-tendon complex length. *3*) A similar hypothesis is tested for the effects of changes of EDL muscle-tendon complex length on neighboring synergists.

MATERIALS AND METHODS

Surgical and experimental procedures were in strict agreement with the guidelines and regulations concerning animal welfare and experimentation set forth by Dutch law and were approved by a Committee on Ethics of Animal Experimentation at the Vrije Universiteit, Amsterdam. Immediately after all experiments, double-sided pneumothorax was performed, and animals were killed with an overdose of pentobarbital.

Surgical Procedure and Preparation for Experiment

Male Wistar rats (n = 6, mean ± SD body mass: 301 ± 16.25 g) were anesthetized by intraperitoneal injection of a urethane solution (initial dose, 150 mg/100 g body mass). Supplementary doses of the anesthetic agent (0.62 mg) were injected intraperitoneally (maximally three times), if necessary, to maintain deep anesthesia. The animals were placed on a heated water pad (37°C) during surgery and experimentation.

Femoral compartment. The left femoral compartments were opened to *1*) cut the femur transversely to allow later fixation within the experimental setup; *2*) reach the insertion of the proximal tendon of EDL muscle; and *3*) dissect the sciatic nerve and cut it as proximally as possible. The sural, tibial, and articular branches of the sciatic nerve were cut so that, by stimulating the sciatic nerve during the experiment, the full motor segment of the common peroneal nerve would be excited exclusively.

Anterior crural compartment. In the rat, connective tissue associated with the biceps muscle covers the anterior compartment to reach a very long insertion along the tibia. Removing the skin, parts of the crural fascia, and the biceps femoris muscle exposed the anterior crural compartment of the left leg. The very distal part of the anterior tibial compartment had to be opened (local fasciotomy) to reach the distal tendons of EDL and of the EHL and TA muscles. With the knee joint at ~100° and the angle between the footplate and the tibia at 90° (referred to as the reference position), two sets of distal tendons were tied together: *1*) the four adjacent distal tendons of EDL (using polyester thread); and *2*) the distal EHL tendon, which was tied to the adjacent distal tendon of TA (using polyester thread). The complex of TA and EHL created this way will further be referred to as TA+EHL.

Matching markers were placed on the distal tendons of EDL and of TA and EHL, as well as on a fixed location on the lower leg.

Peroneal compartment. Similar to the anterior crural compartment, the peroneal compartment was opened only distally to reach the distal tendons of the peroneal muscle group. This group consists of the four peroneal muscles (i.e., mm. peroneus longus, brevis, quarti, and quinti), which fill most of the compartment. Their distal tendons were dissected free from surrounding tissues, leaving the compartmental borders and connective tissues around the muscle bellies fully intact. The four adjacent distal peroneal tendons were tied together by using polyester and Kevlar threads.

Further treatment of the tendons of target muscles. The retinaculae at the ankle (i.e., transverse crural ligament and the cruciate ligament) were removed while under observation through a dissection microscope (Wild, magnification ×6–40). Subsequently, tenotomy was performed as distally as possible on the distal EDL and TA+EHL as well as peroneal tendon complexes. The severed tendons were removed from their retinaculae near the ankle joint (transverse crural ligament and cruciate ligament). The proximal tendon of EDL was cut loose from the femur, with a small piece of the lateral femur condyle still attached.

With the use of polyester threads, each of the distal tendon complexes as well as the proximal EDL tendon were sutured to Kevlar threads with a small loop at their end (proximal or distal, respectively).

Subsequently, the left foot of the rat was attached firmly to a plastic footplate by using a Kevlar thread.

If necessary, the tendons were irrigated with an isotonic saline solution to prevent drying.

All Kevlar threads used (Goodfellow) are characterized by a diameter of 0.2 mm and a tensile modulus of 58 GPa. All polyester threads used (Gütermann) had a diameter of 0.3 mm.

Mounting the Animal in the Experimental Apparatus

The cut femur was secured by means of a self-tapping screw (material: electrolytic zinc-plated steel, diameter: 2 mm). This screw was screwed into the femur through the knee toward the tibia and connected with a stiff aluminum rod (diameter: 8 mm) to the experimental setup (for a schematic view, see Fig. 1*A*).

The plate with the left foot attached was manipulated such that the ankle was in extreme plantar flexion. Such a position makes room for passage of distal tendon complexes and

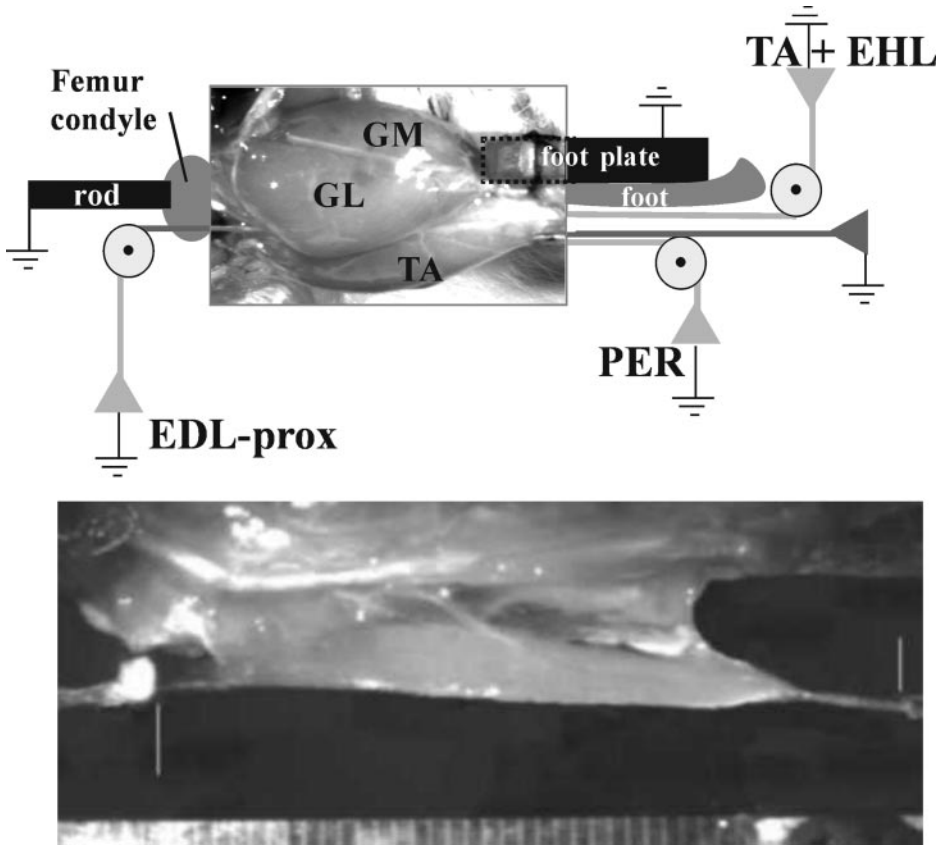

Fig. 1. *Top*: semischematic representation of the experimental setup. Dorsolateral view is shown of the lower leg of the rat as fixed to the experimental setup. Note that only the belly of tibialis anterior (TA) muscle can be seen, in addition to the bellies of the medial (GM) and lateral head of gastrocnemius (GL) muscle. All locations of connection to mechanical ground are indicated by the symbol ⏚. Shaded triangle indicates force transducers for the experimental muscle groups. Low-friction pulleys guiding Kevlar wires connected to the tendons and the force transducers are indicated by shaded circles. Force was measured at the following locations: *1*) proximal tendon of the extensor digitorum longus (EDL-prox), *2*) tied distal tendons of the EDL muscle, and *3*) the tied distal tendons of TA muscle and extensors hallucis longus (EHL) muscle (TA+EHL). Force was also measured at the tied distal tendons of the peroneal (PER) muscles (i.e., peroneus longus, peroneus brevis, peroneus quinti, and peroneus quarti muscles), but those results are not presented in the present work. *Bottom*: to show EDL and its neurovascular tract, as well as its extensions of this tract to the anterior intermuscular septum, TA+EHL had to be removed. Note that this dissection was not performed in the experiments, but was done for purposes of illustration only. After removal of superficial muscles from the anterior crural compartment, EDL still approximately maintained its original position. Therefore, loads needed to be applied in vertical direction to Kevlar wires connected to its tendons (not shown within the image). After such loading, a sheet of connective tissue material becomes visible, showing some nerves and blood vessels.

their attached Kevlar threads (Fig. 1). Subsequently, the plate was firmly attached to the experimental setup with the ankle joint in extreme plantar and ~40° of supination.

The TA+EHL complex was brought to a muscle-tendon complex length corresponding to a summed distal active force of ~3 N. Subsequently, the peroneal muscles were set a muscle-tendon complex length corresponding to a summed distal active force of ~5 N. Such setting of muscle-tendon complex lengths as defined by force exerted has been shown to yield highly reproducible settings.

The EDL distal tendon group was set at a position corresponding approximately to the reference position.

All four Kevlar threads attached to tendons were connected to force transducers (Hottinger Baldwin, maximal output error <0.1%, compliance 0.0048 mm/N). For proximal EDL as well as TA+EHL and the peroneal complex, the Kevlar wires were guided over low-friction pulleys, which were shown not to affect force measurements before the experiments.

The three-dimensional coordinates of the force transducers were manipulated to obtain orthogonal orientations with respect to the Kevlar threads.

The severed end of the sciatic nerve was placed on a bipolar stimulation electrode.

Experimental Procedure and Data Collection

To make sure that any differences in force transducers and their calibration before the experiment introduced no artifact, the two force transducers to be used for measurement of EDL forces were connected to each other with a compliant spring. The output was recorded with the same measurement system [i.e., amplifiers, analog-to-digital (A/D) converters] used in the animal experiment. It is concluded that any major difference in force (>1.36%) at these transducers cannot be ascribed to the measurement system used. In addition to that, the locations of these proximal and distal force transducers for EDL were exchanged in one-half of the experiments.

During the experiments, ambient temperature was kept constant at 22 ± 0.5°C, and air humidity was kept at 80 ± 2% by a computer-controlled air-conditioning system (Holland Heating), creating a downflow of air onto the experimental table. The surface of the anterior crural compartment was rinsed regularly with saline to prevent fluid loss.

TA+EHL and EDL muscles (innervated via the deep peroneal nerve) and also the whole peroneal muscle group (innervated by the superficial peroneal nerve) were excited simultaneously. This was done by stimulating the distal end of the severed, sciatic nerve supramaximally, by using a pair of silver electrodes connected to a constant-current source (square pulse width 100 μs, pulse train 400 ms, 100 Hz). In the preparatory phase of each experiment, current was increased in small steps until no further increase in force was attained. In this condition, currents of ~3 mA were necessary. The constant-current mode of the stimulator delivers the set amplitude of current, even if changes in nerve impedance should occur during the experiment, thereby helping to maintain maximal excitation of the nerve during the course of the experiment. To prevent drying of the nerve, the exposed part was covered with tissue paper and saturated with isotonic saline, which itself was covered by a thin layer of latex.

After lengthening of EDL muscle to any desired target length, two twitches were evoked (200 ms apart). Passive force was determined ~600 ms after the second twitch. Almost immediately after that, the muscles were excited tetanically. During the tetanic plateau (i.e., 275 ms after evoking tetanic stimulation), total isometric muscle force was determined. All force signals were acquired by using an A/D converter (sample frequency 1,000 Hz, resolution of force 0.01 N) and recorded on a microcomputer. A special-purpose microcomputer controlled timing of events related to stimulus generation as well as A/D conversion.

After each isometric contraction, the muscles were allowed to recover for 2 min. For EDL, recovery was allowed to occur near active slack length. The positions of distal tendons of the TA+EHL complex, as well as of the peroneal complex, were kept constant throughout the experiment. As the origins of these muscles were not treated in any way, the muscle-tendon complex length of these muscle groups was constant during experiments.

Experimental Protocol

For the experimental protocol applied, three phases can be distinguished, which were performed in the order shown below.

Initial contractions to eliminate effects of previous activity at high length. Specific care had been taken that experimental muscles did not attain the previous high length of this part of the experiment. Isometric tetanic EDL force was measured at a test length (i.e., a length at which ~0.5 N of active distal force was exerted) with simultaneous measurements of force of the TA+EHL complex, while at constant complex length. Subsequently, EDL was brought to high muscle-tendon complex length (i.e., ~1 mm over optimum length) and activated isometrically. After the recovery period near active slack length, EDL was brought again to the test length and activated. Because of the previous activity at high length, force at the test length was decreased substantially (see also Ref. 28) without changing optimum force.

The procedure described was repeated (~4–5 times) until the previous activity at high length no longer changed the force exerted at the test length.

Distal lengthening for determination of EDL length-force characteristics. EDL muscle-tendon complex length changes were imposed first by moving the distal force transducer (1-mm increments, as determined on a vernier mechanism read to the nearest one-tenth of a millimeter) in between contractions. Proximal tendons were kept at reference position. Length-force data were obtained, starting from active slack length [i.e., the lowest length at which active muscle force (F_{ma}) approaches zero] and ending at ~2 mm over optimum length.

Proximal lengthening for determination of EDL length-force characteristics. Subsequently, EDL muscle-tendon complex length changes were imposed by moving the distal force transducer (1-mm increments, as determined on a vernier mechanism read to the nearest one-tenth of a millimeter) in between contractions. Distal tendons were kept at reference position. Length-force data were obtained starting from active slack length and ending at ~2 mm over optimum length.

Treatment of Data

The individual length-force data sets for passive muscle force (F_{mp}) and muscle length were fitted with an exponential curve (*Eq. 1*), using a least squares criterion

$$y = e^{a_1 + a_2 \cdot x} \quad (1)$$

where y is F_{mp}, x is passive muscle-tendon complex length [i.e., deviation from minimal length (Δl)], and a_1 and a_2 are coefficients determined in the fitting process. F_{ma} was estimated by subtracting passive force calculated according to *Eq. 1* for the appropriate active muscle-tendon complex length from the total force exerted by the muscle at that length.

Data for active EDL force (F_{ma}) in relation to active muscle-tendon complex length (Δl) were fitted by using a polynomial

$$y = b_0 + b_1 \cdot x + b_2 \cdot x^2 + \ldots + b_n \cdot x^n \quad (2)$$

where y is F_{ma}; x is length of the active muscle-tendon complex; n is the order of the polynomial; and $b_0, b_1, b_2 \ldots b_n$ are coefficients determined in the least squares fitting process. The fitting started with a first-order polynomial, and the power was increased up to and including the sixth order. Polynomials that best described the experimental data were selected (see below). These polynomials were used for three purposes: *1*) determining EDL optimal force and *2*) optimum length, and *3*) averaging data and calculating standard errors. For each individual muscle, optimal muscle force (F_{mao}) is defined as the maximum of the fitted polynomial for F_{ma}, and optimum muscle-tendon complex length is defined as the corresponding active length.

Individual data for muscle-tendon complex length are expressed as deviations from minimal EDL length.

Statistics

In the fitting procedure, one-way ANOVA (50) was used to select the lowest order of the polynomials that still added a significant improvement of the description of changes of muscle-tendon complex length and muscle force data for EDL.

Two-way ANOVA for repeated measurements (factors: EDL muscle-tendon complex length and location of EDL force measurement) was performed to test for effects on muscle length-force characteristics measured simultaneously. The same procedure (factors: EDL muscle-tendon complex length and location of EDL lengthening) was applied also on data on

distally exerted EDL force, as well as on proximally exerted force, to test for differential effects of proximal and distal lengthening.

If significant effects were found, post hoc tests were performed by using the Bonferroni procedure for multiple paired comparisons, to further locate significant differences.

To test for any differences in proximal and distal optimum length as well as forces exerted at reference length (l_{ref}) and position or to test further for length effects, one-way ANOVA for repeated measurements was performed.

Any differences at $P \leq 0.05$ were considered significant.

RESULTS

Differences in EDL Length-Force Characteristics Measured Simultaneously at Proximal and Distal Tendons

General features. To facilitate comparison of proximal and distal EDL forces, proximal force is represented as a reaction force (i.e., $-F_{ma}$ or $-F_{mp}$).

Figure 2 shows isometric length-force curves as measured simultaneously at both distal and proximal EDL tendons and obtained after distal lengthening (Fig. 2*A*) and after proximal lengthening (Fig. 2*B*) of EDL muscle, respectively. This classic way of plotting emphasizes a comparison of force for equal muscle-tendon complex lengths. Corresponding values of muscle-tendon complex length normalized for optimum length are shown in Table 1. For both active and passive EDL forces, ANOVA indicates significant effects for factor length and factor proximal or distal location of force measurement, as well as interaction between these factors.

At the tendon at which EDL was lengthened, isometric active force rises much faster with length changes than active force measured at the other (fixed) tendon. This is true for both distal and proximal EDL active forces. Note, however, that optimum lengths for length-force characteristics measured at the tendon at the lengthened end of EDL or at its nonlengthened end are similar (Fig. 2, *A* and *B*, respectively).

Proximodistal differences of force. ACTIVE FORCE. Active forces exerted at proximal and distal tendons of EDL are not equal, except at one particular length

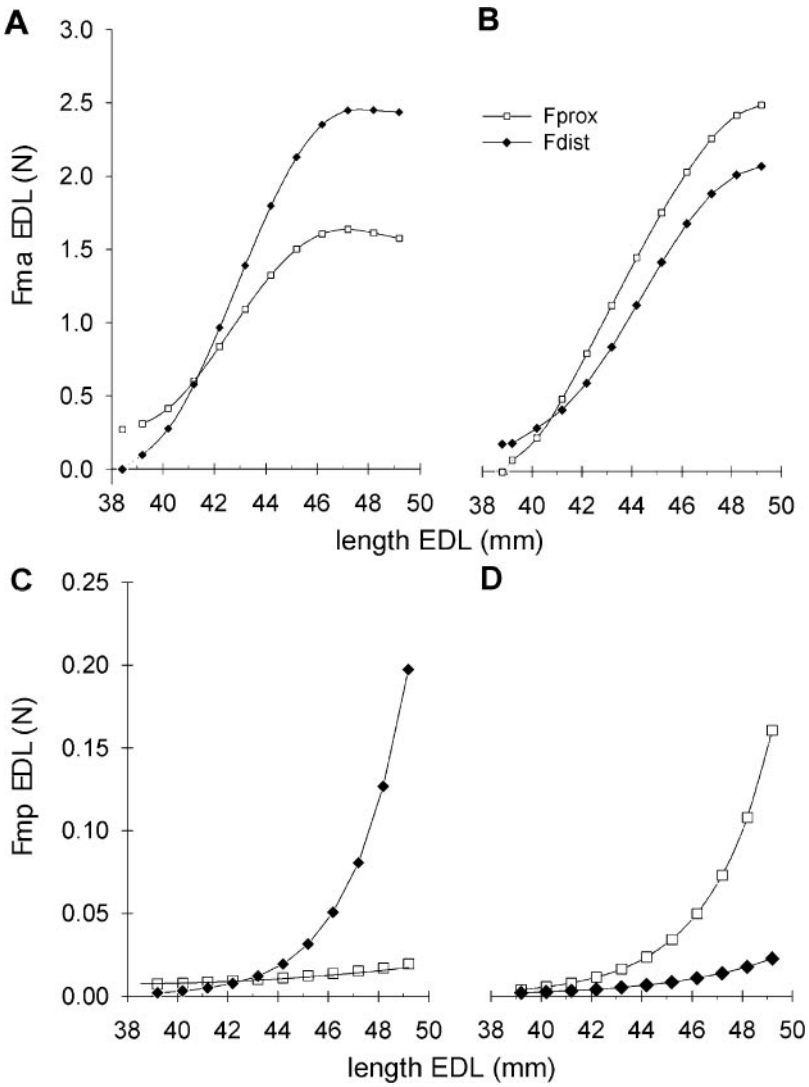

Fig. 2. Length-force characteristics of EDL muscle measured with intact compartmental connective tissues at the muscle belly. Length-force curves were obtained after both proximal (Δl_{prox}) and distal lengthening (Δl_{dist}) of EDL. *A*: isometric active EDL muscle force (F_{ma}) measured at distal (F_{dist}) and proximal tendons (F_{prox}) after Δl_{dist}. *B*: F_{ma} measured at F_{dist} and F_{prox} after Δl_{prox}. *C*: isometric passive EDL muscle force (F_{mp}) measured at F_{dist} and F_{prox} after Δl_{dist}. *D*: F_{mp} measured at F_{dist} and F_{prox} after Δl_{prox}. Absolute length of the EDL muscle tendon complex is expressed in mm.

Table 1. *Corresponding EDL length values normalized for optimum length*

Figure	EDL Muscle-Tendon Complex Length, mm	Fraction of Optimum Length, %
2	38	77.55
	39	79.59
	40	81.63
	41	83.67
	42	85.71
	43	89.80
	44	91.40
	45	91.84
	46	93.88
	47	95.92
	48	97.96
	49	100

	EDL Length Relative to Reference Length, mm			Fraction of EDL Optimum Length for Distal Force After Distal Lengthening, %	Fraction of EDL Optimum Length for Proximal Force After Distal Lengthening, %
	Distal lengthening		Proximal lengthening		
3–6	−2	> equal EDL length <	+2	85.53	86.86
	−1		+1	87.61	88.97
	0		0	89.68	91.08
	+1		−1	91.76	93.19
	+2		−2	93.83	95.30
	+3		−3	95.91	97.41
	+4		−4	97.99	99.52
	+5		−5	100.06	101.62
	+6		−6	102.14	103.72
	+7		−7	104.21	105.84
	+8		−8	106.29	107.95

Mean values are shown. EDL, extensor digitorum longus.

(crossover of curves). Note that crossover length is similar for proximal and distal lengthening (Fig. 2*A*). For the present experimental conditions, no significant differences could be shown for proximal and distal active force at l_{ref}. This means that proximal and distal forces are equal at a length very close to EDL l_{ref} (mean ± SE = 41.27 ± 0.49 mm).

Over crossover length, for distal lengthening, distal EDL active force is higher than proximal force, but for proximal lengthening the reverse is true. This means that, for high lengths, active force exerted at the location of lengthening dominates the active force exerted at the fixed end of EDL over a wide length range.

In contrast, for the smaller length range from active slack lengths up to the crossover length, isometric active force measured at the location of lengthening remained lower initially ($-F_{ma}$ measured at the proximal tendon < F_{ma} measured at the distal tendon on proximal lengthening and vice versa).

It is concluded that proximal and distal lengthening affect the relation of force exerted at the proximal tendon and at the distal tendon in opposite ways.

Any proximodistal difference in force indicates myofascial force transmission (extra- and or intermuscular) from or onto EDL. Such myofascial force transmission can even cause one EDL tendon to be slack (force = 0), whereas, at the other tendon, a substantial force is still exerted (Fig. 2, *A* and *B*; compare values at the respective active slack lengths). As the tendon at the location of lengthening approaches slack, force exerted at the fixed tendon may be substantial (F_{ma} = 0.27 N, Fig. 2*A*; F_{ma} = 0.19 N, Fig. 2*B*). This represents ~16.5 and 9% of the optimal value of proximal force after distal lengthening and of distal force after proximal lengthening, respectively. In these specific conditions, it is clear that all force exerted at the tendon at which the muscle is not lengthened is transmitted from the muscle, without myotendinous force transmission to the other tendon.

Values of the proximodistal active ΔF are quite dependent on the location of lengthening of the muscle. For distal lengthening, this difference ranges from approximately $-0.27\ N < \Delta F_{ma} < +0.90\ N$. For proximal lengthening, the range of ΔF is more limited (i.e., $-0.19\ N < \Delta F_{ma} < +0.41\ N$). Even though these absolute values do not seem high, compared with the active force actually exerted by the muscle, the proximodistal active ΔF values represent very substantial fractions of the actual highest force exerted at a particular length.

It is concluded that the opposite effects of proximal and distal lengthening on active force are not symmetric. More active force is transmitted via myofascial pathways from or onto EDL after distal lengthening than after proximal lengthening.

PASSIVE FORCE. Also for passive force at higher lengths, EDL force exerted at the tendon at the location of lengthening is considerably higher than that at the fixed tendon. At high lengths, passive force may be as much as 9–10 times higher (Fig. 2, *C* and *D*) than

exerted at the fixed tendon. This indicates that, throughout the length range over crossover length, most of the passive force exerted at the tendon of the lengthening location is transmitted to or from extramuscular tissues (extramuscular myofascial force transmission) or other muscles (intermuscular force transmission).

Note that, as for active force, the proximodistal ΔF is higher after distal than after proximal lengthening. Therefore, more passive myofascial force transmission from or onto the muscle did occur as EDL was lengthened distally.

It is concluded that symmetric lengthening of EDL muscle-tendon complex at proximal or distal tendons to equal lengths causes not only opposite but also quite asymmetric effects on active as well as passive length-force characteristics and on the proximodistal differences of force.

Therefore, for muscle length change, not only effects of its magnitude should be considered, but also effects of its direction.

Further directional analysis: comparison of force exerted at a specific EDL tendon in different conditions. The muscle-tendon complex lengths of EDL muscle can also be expressed as deviation from its l_{ref}, i.e., its length with both proximal and distal tendons at their reference positions (corresponding to EDL length encountered with ankle joint at 90° and the knee joints at 100°). For proximal and distal lengthening of the muscle to equal muscle-tendon complex lengths, using that format, Fig. 3 compares EDL with either distal (*A*) or proximal isometric active forces (*B*). It should be noted that, in such a representation, the effect of relative position of the muscle (or parts thereof) is emphasized. Successive points of any one curve are affected by *1*) muscle-tendon complex length and *2*) the location of lengthening. The latter effect involves changes of the relative position of (parts of) the muscle with respect to surrounding muscle and other tissues. In such a case, the *x*-axis also represents the deviation of a major part of the muscle from reference position and the direction of such deviation. For comparisons made for equal absolute values of Δl (i.e., symmetric with respect to l_{ref}), EDL lengths are identical (see also Table 1) but were obtained by lengthening of the muscle-tendon complex by moving tendons at opposite ends and in opposite directions.

For active force (Fig. 3) as well as passive force (Fig. 4), ANOVA showed significant effects of length and location of lengthening and a significant interaction between these factors. Such a result was found for both distal and proximal EDL force.

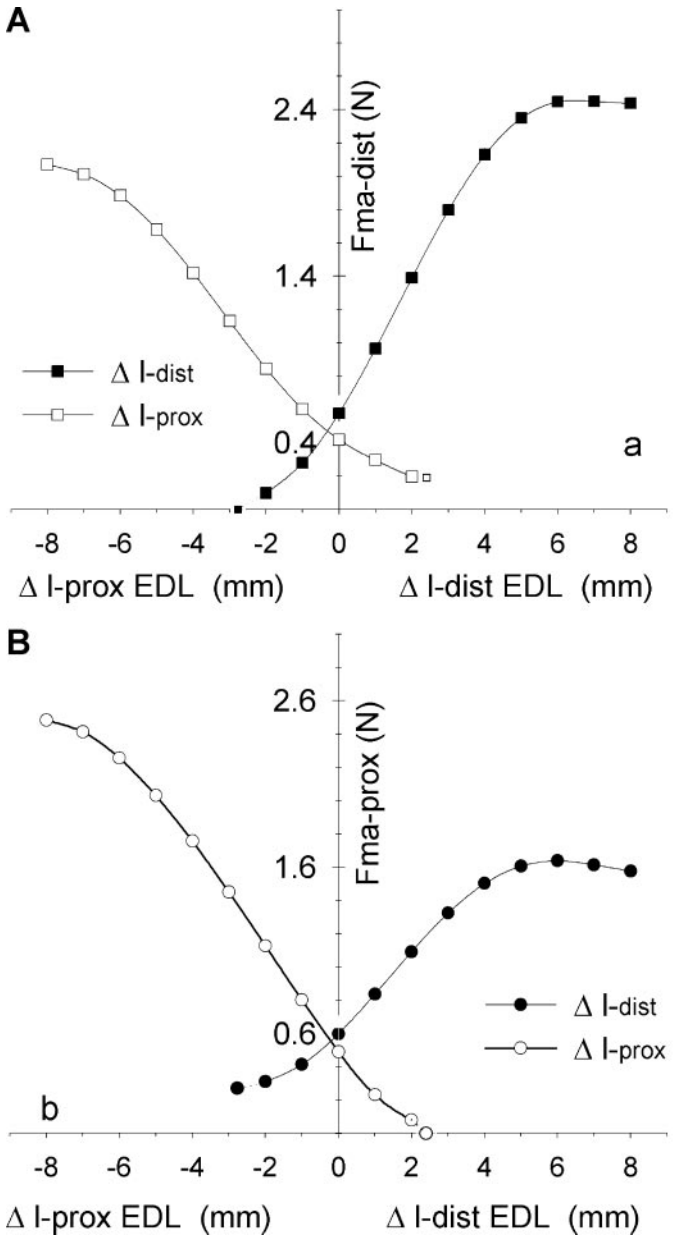

Fig. 3. Length-F_{ma} characteristics of EDL muscle measured with intact compartmental connective tissues at the muscle belly. Length-force curves were obtained after both Δl_{prox} and Δl_{dist} of EDL. *A*: F_{ma} measured at the tied distal tendons ($F_{ma\text{-}dist}$) after Δl_{dist} as well as after Δl_{prox}. *B*: F_{ma} measured at the proximal tendon ($F_{ma\text{-}prox}$) after Δl_{dist} as well as after Δl_{prox}. Length of the EDL muscle-tendon complex is expressed as deviation from reference length, i.e., the length corresponding to a knee joint angle and ankle joint angle of 100 and 90°, respectively.

ACTIVE FORCE. The length range between optimum and active slack lengths is smaller for distal lengthening of EDL than for proximal lengthening. This is true for distal as well as proximal EDL length-active force characteristics (Fig. 3). This higher length range is related predominantly to a higher optimum length after the proximal lengthening than after distal lengthening (Fig. 3, see also Fig. 2*A*: optimum length after proximal lengthening ≈ 49 mm, and Fig. 2*B*: optimum length after distal lengthening ≈ 47 mm). Smaller but opposite differences of active slack lengths cannot fully abolish this effect on length range.

EDL force measurements in the direction of lengthening yield higher forces (e.g., distal lengthening increases distal force more than proximal lengthening). For distal force such an asymmetry, the response is already apparent (Fig. 3*A*). This muscle position-related asymmetry is substantial, particularly at high

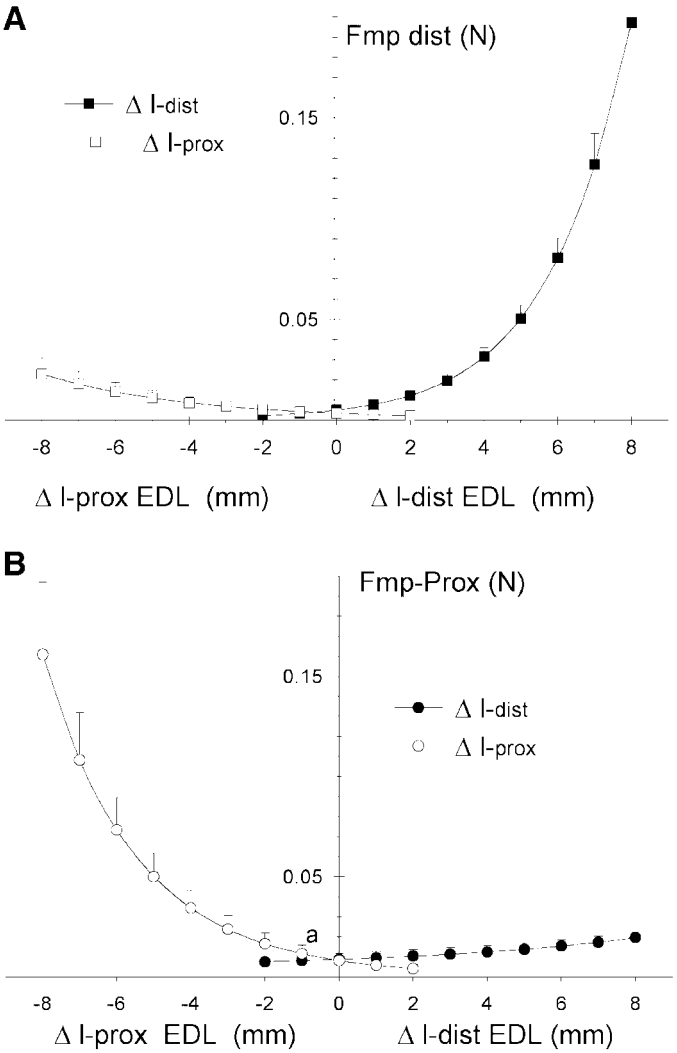

Fig. 4. Length-F_{mp} characteristics of EDL muscle measured with intact compartmental connective tissues at the muscle belly. Length-force curves were obtained after both Δl_{prox} and Δl_{dist} of EDL. *A*: F_{mp} measured at the tied distal tendons ($F_{mp\text{-}dist}$) after Δl_{dist} as well as after Δl_{prox}. *B*: F_{mp} measured after the proximal tendons ($F_{mp\text{-}prox}$) after Δl_{dist} as well as after Δl_{prox}. Length of the EDL muscle-tendon complex is expressed as deviation from reference length, i.e., the length corresponding to a knee joint angle and ankle joint angle of 100 and 90°, respectively.

lengths [$F_{dist}(\Delta l_{dist})/F_{dist}(\Delta l_{prox}) \approx 118\%$, where F_{dist} is F_{ma} measured at distal tendons, Δl_{dist} is distal lengthening, and Δl_{prox} is proximal lengthening]. At high length, the enormous asymmetry of the proximal EDL active length-force curves after distal and proximal lengthening of EDL is striking (Fig. 3*B*). After distal lengthening, much lower isometric forces are exerted at the proximal EDL tendon than after equal distal lengthening [$F_{prox}(\Delta l_{prox})/F_{prox}(\Delta l_{dist}) \approx 157\%$, where F_{prox} is F_{ma} measured at proximal tendons].

Note that the crossover point for the curves of active force exerted at a specific tendon (i.e., equal force after proximal or distal lengthening) of EDL distal as well proximal length-force curves is quite near to EDL l_{ref}. This similarity is due to the fact that, exclusively for this data point, one extra factor is similar (in addition to the identical EDL lengths). This additional factor is the relative position of EDL with respect to surrounding muscles and connective tissue. It should be noted that these conditions are satisfied only for the data at l_{ref}.

It is concluded that, if EDL has a similar length as well as relative position, forces exerted at a specific tendon are similar. However, if only EDL length is similar, but not EDL relative position, then that is clearly not the case.

PASSIVE FORCE. Figure 4 indicates that an extremely high degree of asymmetry, effects of distal and proximal lengthening, is also true for passive EDL force. For distal lengthening, distal force increases more than for proximal lengthening, and proximal force increases more for proximal lengthening than for distal lengthening. The asymmetry increases readily with increasing lengths. Passive forces exerted at a particular tendon increase much more if lengthening was performed at that site rather than at the other end of the muscle (>7–10 times as high for distal and proximal force, respectively).

These results indicate that, at lengths other than l_{ref}, relative position of the muscle with respect to its surroundings is a factor of considerable importance, co-determining (with length) isometric length-force characteristics.

Differential Effects of Proximal or Distal EDL Lengthening on Neighboring Muscles

Synergists: anterior crural group. Because TA+EHL muscle-tendon complex length was constant, TA+EHL active force is plotted as a function of EDL length (i.e., deviation from its l_{ref}: Fig. 5). ANOVA of these data indicates significant main effects (factors: EDL muscle-tendon complex length and location of lengthening), as well as significant interaction between these factors, on TA+EHL active force.

Distal lengthening of EDL affected TA+EHL active force significantly. Post hoc test located significant length effects on TA+EHL force at higher EDL lengths after distal lengthening: active force exerted at the length range $\Delta l_{dist} > 2$ mm differed from that for the length range $\Delta l_{prox} < -2$ mm ($\Delta F_{ma} \approx -0.3$ N, i.e., by approximately −10% of initial force). In contrast, proximal EDL lengthening to equal muscle-tendon complex lengths did not affect TA+EHL force significantly. Such results once more emphasize the asymmetric effects of proximal and distal lengthening of EDL, this time on force exerted by its synergists, that were kept at constant muscle-tendon complex length.

It is concluded that, particularly for distal lengthening, we have evidence of intermuscular myofascial force transmission between the active synergists. This means that active force is transmitted from TA+EHL onto EDL to be exerted at its distal tendon.

ANOVA did not show either significant EDL length effects or interaction on passive TA+EHL force, but did show significant effects of location of EDL lengthening (Fig. 5*B*).

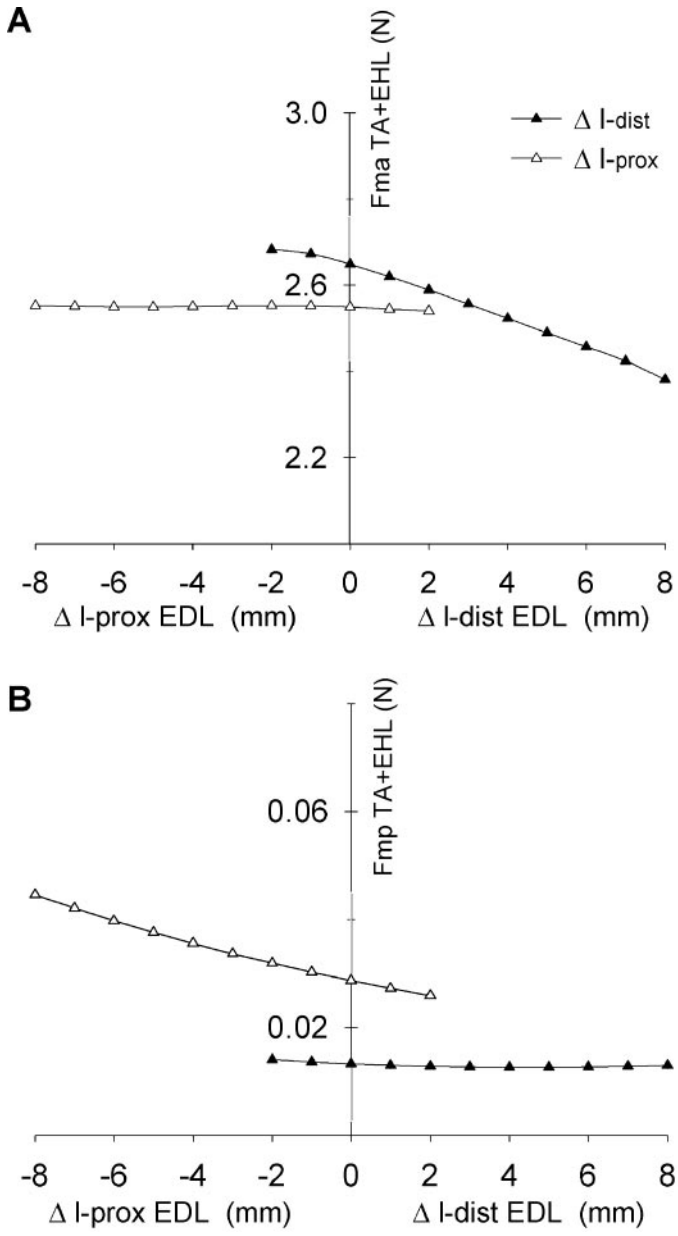

Fig. 5. Summed force exerted at the distal tendons of TA and EHL muscles while kept at constant muscle-tendon complex length. Force was measured with intact compartmental connective tissues at the muscle bellies. Force data were obtained after both Δl_{prox} and Δl_{dist} of EDL, and force is plotted as a function of EDL length. *A*: F_{ma} measured at F_{dist} of TA+EHL after Δl_{dist} as well as after Δl_{prox}. *B*: F_{mp} measured at the F_{dist} of TA+EHL after Δl_{dist} as well as after Δl_{prox}. Length of the EDL muscle-tendon complex is expressed as deviation from reference length, i.e., the length corresponding to a knee joint angle and ankle joint angle of 100 and 90°, respectively.

It is concluded that symmetric lengthening of the adjacent synergist (EDL), at either the proximal or distal tendon to attain equal muscle-tendon complex lengths, has very asymmetric effects on active as well as passive force exerted at the distal tendons of TA and EHL.

It is concluded that symmetric proximal or distal lengthening of the EDL muscle-tendon complex causes differential effects on active force exerted at distal tendons by synergists TA+EHL kept at constant muscle-tendon complex length. In addition, passive force of TA+EHL is affected differentially as well. The asymmetry of these responses to symmetric lengthening at EDL proximal or distal tendons is striking and is related to differences in relative position of EDL with respect to neighboring muscles and extramuscular connective tissue.

DISCUSSION

The almost permanently present difference in proximal and distal EDL forces after proximal lengthening of EDL reported in the present work confirms the phenomenon for somewhat different experimental conditions compared with previous work (26, 28, 39). The reversal of sign of the difference, as well as the differences in magnitude after and symmetric lengthening to equal EDL length, is a novel and surprising finding. These results could not be explained if muscle fibers transmitted their force exclusively in series from sarcomere to sarcomere and then to the tendon.

Myofascial Force Transmission

The general idea of this concept is that, in addition to myotendinous transmission, force is transmitted also between sarcomeres and the collagen fiber-reinforced extracellular matrix of muscle (i.e., the intramuscular connective tissues). Active force generated within the contractile proteins of an individual sarcomere will lead to shortening, unless opposed by an external force. If the opposing force counteracting the sarcomere shortening does not originate from sarcomeres in series, but from the extracellular matrix, we call it active myofascial force transmission. In passive muscle, an external force may stretch sarcomeres. The external force exerted onto a stretched muscle may be transmitted via the myotendinous junction to the sarcomeres or onto the muscle's extracellular matrix. Based on an old observation by Banus and Zetlin (2), before the 1970s the generally accepted view was that passive force was borne by the structures of the extracellular matrix. However, after that time it became clear (e.g., Ref. 41) also that the sarcomeres are stretched, and most of the force exerted on the muscle is borne by stretched passive components [e.g., titin filaments (18)] of the sarcomere. In such a stretched condition, the tendency of the sarcomere to shorten elastically to its initial length is also prevented by the external force. If the external force is transmitted via the extracellular matrix to the sarcomere (rather than via the myotendinous junction and sarcomeres in series), we speak about passive myofascial force transmission.

The feasibility of such force transmission between the muscle fiber and at least its endomysium has been indicated previously (e.g., Refs. 24, 25, 52–54, 63–67).

Intramuscular transmission. Once the force is exerted on an endomysium, there are again two potential paths. The first path is tensile transmission in longitudinal direction. As the endomysium is continuous with a similar network surrounding groups of

tendon collagen fibers, force can be transmitted onto the tendon.

The second path is shear transmission in cross-fiber directions. Force can also be transmitted further on the continuous endomysial stroma, i.e., the endomysium of neighboring muscle fibers of a fascicle. As the endomysial network is also continuous with the perimysium, force can be transmitted again in the two principal directions, either onto the tendon or neighboring fascicles (28, 43) or to the epimysium, as the perimysial network is also continuous with the epimysium.

If the muscle is dissected fully from other structures, intramuscular myofascial force transmission to the tendon is the only path, in addition to direct myotendinous force transmission. In effect, the full stroma of intramuscular connective tissues of a muscle acts as an integrator for active and/or passive force exerted by muscle fibers that have sufficiently stiff connections to their endomysium. This property allows groups of muscle fibers that are no longer connected to their tendon at one end still to contribute to muscle force (29, 31, 32). These contributions are made at lower mean sarcomere lengths than those of fibers with connections to the tendon fully intact, because fiber shortening occurs until sufficient shear strain has accumulated in the basal lamina and endomysium to stiffen these structures to be able to transmit the additional force (43). This introduces or enhances a distribution of fiber mean sarcomere length of the muscle. However, for fully isolated muscle, force exerted at the proximal or distal tendon is equal as the muscle is built of parallel myofascial units exerting identical force at proximal and distal ends.

Extra- and intermuscular transmission. Any proximodistal ΔF for a muscle, as reported also in the present work, indicates that force is transmitted from or onto the muscle-tendon complex anywhere between the locations of force measurement.

In fact, also in such conditions, force originating from sources outside of the muscle is integrated with force exerted by the target muscle. However, the locations of the points of application of the external forces being intermediate between the ends of the muscle fibers causes this integration to create or enhance distribution of lengths of sarcomeres arranged in series within the muscle fibers (73). Also, as a consequence, the distribution of fiber mean sarcomere lengths is expected to be affected.

If active or passive force is transmitted between the intramuscular connective tissues of two muscles, we refer to it as intermuscular myofascial force transmission. In such a case, the paths of transmission are the very short collagen fiber connections between the intermuscular stroma of the two muscles. Sometimes in literature, evidence may be found in general terms that some awareness existed of intermuscular connections of muscles or their conceivable effects (e.g., that muscle may not be fully independent units) (e.g., Refs. 51, 69, 70, 71). A most expressive example may be a short note by Pond (51) in response to a review article by Stein (62). The ideas presented in that note are quite compatible with the concepts of intermuscular myofascial force transmission presented here. Unfortunately, at the time, no follow up was performed by Pond or others to provide any experimental evidence.

If force is transmitted, in either direction, between the intramuscular connective tissue of a muscle and extramuscular connective tissues, we refer to it as extramuscular myofascial force transmission. Examples of extramuscular connective tissue are the fascia constituting the compartmental boundaries or the neurovascular tract (i.e., the string of connective tissue containing bundles of nerves and vessels with its connections to compartment boundaries).

In most physiological experiments, dissection is performed on inter- and extramuscular connective tissues to gain access to the muscle. So far we have been able to find only one reference that stated explicitly that dissection was performed to remove (for the particular experiment) undesired effects of extramuscular and intermuscular connections on the properties of the experimental muscle (11).

Length and Position Effects of EDL

As a novel aspect of our present work, we systematically studied aspects of myofascial force transmission by imposing symmetrical length changes on the EDL muscle tendon complex to equal lengths at either its distal or proximal tendon. For a muscle with the connectivity of its belly to the extracellular matrix of connective tissues and to nearby muscles maintained close to their in vivo condition, as in the present work, three major conclusions can be drawn. *1*) Lengthening of the muscle-tendon complex is always accompanied by changes in configuration of the intra- and extramuscular connective tissues, because of changes of relative positions of most of the muscle. *2*) The combined effects of changing length and muscular relative position are not symmetric for proximal and distal lengthening. *3*) These effects do also change force exerted at distal tendons of synergist and even antagonist muscles, even if the lengths of their muscle-tendon complexes are kept constant.

In combination with muscle-tendon complex length effects, the factor relative muscular position is shown to be a major codeterminant of muscle force: for comparisons at equal length obtained by either proximal or distal lengthening, force actually exerted by EDL may be quite different (e.g., Fig. 3). A most simple representation of this idea is shown in Fig. 6. Note that the length change of the muscle-tendon complex is accompanied by changes in relative position of the muscle with respect to its surroundings. Also note that the conditions as represented in Fig. 6 would have yielded opposite, but symmetrical, effects on the proximodistal ΔF.

The changes in relative position should not be expected to be similar for different segments of the muscle. For distal lengthening, the most distal segments of the muscle fibers are repositioned most with respect to their surroundings, and the most proximal, less.

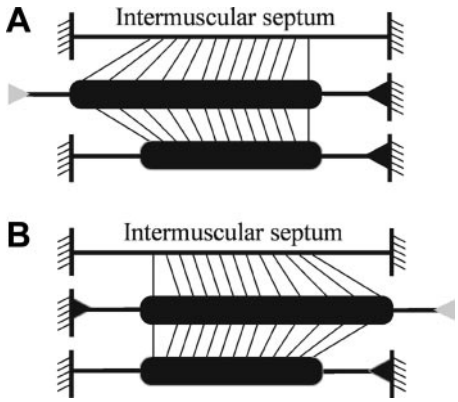

Fig. 6. Schematic representation of intermuscular and extramuscular connections of EDL muscle. *A*: the principle of effects of Δl_{prox} on the relative position of the EDL muscle belly. *B*: effects of Δl_{dist} of that muscle. In both cases, EDL intermuscular connections to TA+EHL muscles, as well as extramuscular connections to the anterior intermuscular septum, are extended nonuniformly with the highest deviations at the lengthened end.

Therefore, the effects should be expected to have a high local variation within muscle fibers and/or their associated endomysial network. It is concluded that imposing a length change on the muscle-tendon complex with intact extra- and intermuscular connections inseparably involves some changes in relative position. It has also proven feasible to assess experimentally effects of relative position without involving length change of the target muscle (for a preliminary report, see Ref. 40). An advantage of the present approach is that effects of different EDL positions were assessed at many EDL lengths by symmetric proximal and distal lengthening of EDL.

Why are effects of EDL relative position so asymmetric? The asymmetry of myofascial force transmission effects may have two major sources.

The first source is extramuscular structures. Because EDL muscle has no insertions of muscle fibers on the walls of the anterior tibial compartment, we argued previously (28) that the neurovascular tract is a major pathway. This may seem counterintuitive, as it is likely that the collagen fibers have been laid down to protect these sensitive structures. The nerves and blood vessels themselves will bear some of the force. However, it should be realized that, in a collagen fiber-reinforced composite (as muscle is), a relatively weak and compliant matrix (including the neurons and blood vessels) will be unloaded by a high concentration of collagen fibers (i.e., the fiber reinforcement). This happens because, at any intersection of paths of force transmission, such collection of fibers "attracts" a high force because of their high stiffness, i.e., most force is transmitted along the stiffest path (24). In this way, the work performed on the structure is minimized.

The proximal-to-distal decreasing size of the neurovascular tract due to the branching pattern of nerves and blood vessels may contribute to the asymmetry. However, this cannot be the only reason as it cannot explain the reversal of the sign of the effect of equal proximal and distal lengthening on proximal as well as distal EDL force.

The second source is intermuscular sources. If the muscles are shifted with respect to each other in such a way that intermuscular connections with EDL are loaded preferentially in one direction, asymmetries due to proximal and distal lengthening will arise. Because we know little about the neutral positions of muscle within a compartment with respect to each other, such contributions are presently fairly hard to judge. However, for the experimental conditions of the present study, it seems likely that, at high lengths, this factor will play a role because of high EDL lengths and the lower TA+EHL length.

Effects on Neighboring Muscles Kept at Constant Lengths

The mechanical interaction between immediately adjacent synergists is most likely intermuscular myofascial force transmission. However, the possibility cannot be excluded that extramuscular pathways also play a role in this phenomenon. There are a number of conceivable paths for extramuscular mechanical interaction in this case.

First, there are connections between both muscles via their neurovascular tracts and the intermuscular septum. EDL as well as the TA+EHL muscles are innervated via nerve branches (from the peroneus profundus nerve). This nerve enters into the anterior crural compartment from the peroneal compartment, through a fenestration within the anterior intermuscular septum (e.g., Fig. 5, *A* and *B*, of Ref. 27). Both muscles receive circulation via branches of blood vessels (e.g., tibialis artery) that enter each compartment from the proximal side. These delicate structures are embedded in a structure of rather stiff connective tissues, with the purpose of protecting them from excessive strain. The connective tissues around nerves and blood vessels of each muscle have connections to the intermuscular septum (Fig. 7). We refer to the complex of all of these elements as the neurovascular tract. For anatomic photographs of this neurovascular tract, see the following references (e.g., see Fig. 4 of Ref. 28, Fig. 5*C* of Ref. 27, and figures of Ref. 30).

Such connections, in principle, do allow mechanical interaction between the muscles by means of extramuscular force transmission. Therefore, this pathway is one of the prime candidates for explaining interaction between adjacent synergists by extramuscular myofascial force transmission.

Second, it should be realized that direct connections of the intramuscular connective tissues of muscle bellies to the septum, presumably with myotendinous junctions, exist in muscle for fibers of EHL (Fig. 7). Because rat EDL muscle fibers have no origin or insertion of muscle fibers onto that structure, it is clear that interactions between these muscles must be mediated by intermuscular connective tissues or the extramuscular neurovascular tract.

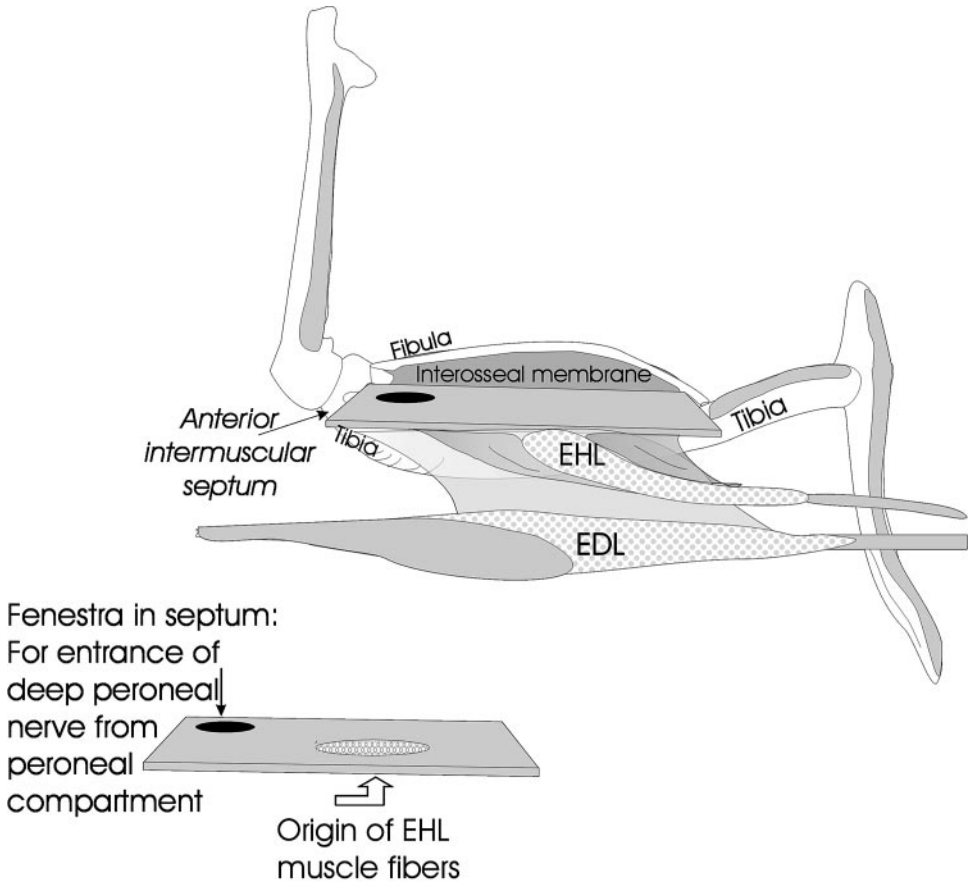

Fig. 7. Schematic exploded view of the extramuscular connections between anterior crural muscles. The TA muscle is removed completely to expose deeper muscles. Synergists (EDL, TA, and EHL) are connected via their neurovascular tract to the anterior intermuscular septum. If EDL and EHL muscles are moved away from their physiological position, the neurovascular tract becomes visible as a sheet containing nerves and blood vessels (not drawn). This causes the muscles to rotate from their original position. In physiological positions, the neurovascular tract is not an extended sheet but a much more compact structure. Changes of length and/or relative position of any of the muscles will cause strain in the neurovascular tracts or parts thereof.

For anatomic photographs of the neurovascular tract, see the following references (e.g., see Fig. 4 of Ref. 28, Fig. 5*C* of Ref. 27, and figures of Ref. 30).

It is concluded that substantial mechanical interaction by myofascial force transmission may be encountered between synergists. Those muscles cannot be considered mechanically as fully independent actuators.

Changes of Muscle Relative Position Are a Common Occurrence in Vivo

Any length change of a muscle will lead to changes in position of the muscle belly with respect to fixed extramuscular elements of the compartment (e.g., bones as well as compartment boundaries), which will strain the connective tissue elements that connect muscle to these structures.

In our present experiments, the length and position of EDL were changed exclusively. In vivo, simultaneous length changes in synergists are determined individually by changes of joint angle and activity of the muscle. It should be realized that any difference in moment arm between synergists would lead to changes in relative position of the muscles. Differences in moment arm for synergists are quite usual and sometimes substantial (e.g., Refs. 12, 13, 15, 19, 38, 43, 68). For example, for the mouse, the moment arm of TA at the ankle is reported to be 17% higher than for EDL (37). Therefore, differences in moment arm may contribute to considerable changes in in vivo muscular relative positions.

In addition to the above, a distinction between monoarticular and bi- or polyarticular muscles should be made. Because length changes of the latter group are determined in two or more joints, these muscles are likely to change their relative position with respect to monoarticular muscles and extramuscular connective tissues.

The size of the mechanical effect of in vivo changes of muscle relative position is codetermined by the actual stiffness of extra- and intermuscular connections. In our present experimental conditions, a group of synergists as well as a group of antagonist muscles were activated maximally (i.e., synchronized maximal activity of all muscles studied). This is not a very likely occurrence in vivo. Nevertheless, coactivation of synergists and antagonists is a common feature of in vivo movement (e.g., Refs. 3, 61). However, as extramuscular myofascial force transmission is expected to be a prominent feature of muscle, higher strains and thus stiffness may have been induced, particularly in extramuscular tissues and possibly also in intermuscular connections, than would be encountered with lower degrees of activation. On the other hand, our results also show sizable effects for EDL relative position in the passive condition also, in which stiffness of different pathways was not very high. Therefore, we expect that in vivo myofascial force transmission and effects of

muscle relative position will have effects of sufficient magnitude to be taken into account, even if levels of recruitment are considerably lower than in our present study.

It is concluded that substantial changes of in vivo muscle position with respect to neighboring muscles and extramuscular structures are likely. Nevertheless, quantification of such effects awaits application of in vivo imaging techniques to this problem.

Some Functional Implications of Effects of Changes of Muscle Relative Position

A major effect of myofascial force transmission is that individual muscle should not be considered anymore as fully independent units of force generation and movement and that muscular relative position is a major codeterminant of force exerted at individual tendons of synergists and antagonists. This altered view is likely to have substantial effects on ideas within a large number of fields.

Joint range of movement. Some of the expected functional effects are mediated by changes in joint angle-moment curves. The joint range of active force exertion of a muscle tendon complex is expected to increase because of increased distributions of sarcomere lengths within the muscle (23). Particularly at lower and higher muscle-tendon complex lengths, even limited enhancement of the active force may, for individuals, have quite important effects, as limitations to movement are removed by enhancing the length range of active force generation. In an isolated muscle, such increases in distribution of sarcomere lengths will always lower active force exerted at optimum length (23). However, in vivo intermuscular force transmission may counteract such effects.

Even within a midrange of joint movement, it is likely that extramuscular forces and possibly also intermuscular forces will cause high strains locally in muscles and connective tissue of a compartment, particularly at higher deviation of muscle relative position from equilibrium positions. Such local deformations may play a major role in the etiology of afflictions of the human locomotion apparatus, such as repetitive strain injury, tennis elbow, musician's arm, etc., and the adaptation of muscle and connective tissues to such conditions.

Motor control. Individual muscles have been distinguished anatomically according to identifiable morphology after dissection, as well as different mechanical effects after being isolated. Our results regarding myofascial force transmission indicate that the mechanical basis underlying motor control is different. Neighboring synergists (EDL, EHL, and TA) form more of a functional unit than foreseen previously.

Of course, even with the view of myofascial force transmission, the basic unit of control remains the motor unit (e.g., Ref. 47). Explaining how the central nervous system controls posture and movement in vivo requires an understanding of how excitation of motor neurons is organized. The rules that have been shown for regulating the control of recruitment of motor units of one muscle (e.g., Refs. 20, 21, 22) have been called the size principle: motor units of a muscle are predominantly recruited in order of their size, which correlates with many other motor unit properties.

Our present results could yield an expectation that the concept of motor unit pool (traditionally limited to the motor units of one muscle) should be extended to include motor units of synergists. In an exclusively muscle-based scheme, the size principle would organize only those motor units within individual muscles, leaving the nervous system with the substantial task of coordinating the relative activities of motor units from different muscles. Cope and Sokoloff (10) reviewed advantages of organization at a higher level than the muscle and speculated about such extension of the concept of the pool. They argued that this would simplify the neural process involved in organizing active motor units: in a muscle ensemble-based scheme, orderly recruitment of all motor units according to the size principle would automatically coordinate motor units both within and across motor nuclei. In subsequent experimental work (60), cat medial and lateral gastrocnemius were distinguished as two separate muscles on the basis of their motor nuclei. In the decerebrate cat, they dissected these heads of the gastrocnemius muscle from surrounding tissues but not from each other. They demonstrated that the size principle applies most of the time to the pool of motor units from these structures, if stretch were applied to the gastrocnemius tendon. However, such recruitment order of motor units may not be applicable to all active synergists within a limb, as they could not consistently show such order of control of motor units for synergist cat medial gastrocnemius and posterior biceps femoris muscles (both knee flexors), if activated by the cutaneous reflex induced by stimulation of the cutaneous crural nerve.

For intramuscular coordination, it has already been pointed out (46) that integration of multiple levels of organization may yield unique mechanical properties of motor units. Our present work indicates that such an idea should be extended to a process of integration across muscles. By varying exclusively relative positions of synergists (40), very different mechanical effects can be obtained. It is expected that varying recruitment would also cause such variable effects because of the mechanics of extra- and intermuscular force transmission.

Perfect coordination of muscular activity is only possible if relative stiffness of different intra-, extra-, and intermuscular pathways, which determine the partition of force transmitted via the major pathways, are optimal for exertion of moments and forces at the joints. However, joint stability is a *conditio sine qua non* for movement. Myofascial force transmission may help to exert forces at the joint. It has been pointed out that joint capsule and capsular ligaments cannot be described as separate entities but should be considered in unity with muscles that are arranged mechanically in series with them (70, 42).

It seems likely that a major role of motor control is the tuning of the stiffness of many connective tissue elements of a limb. This may seem to be an incredibly complex task for the central nervous system because the number and degree of interactions seem almost limitless. Two factors should aid to simplify this problem somewhat.

First, intramuscular and muscle-related receptors of different kinds are reported to be located at specialized positions within muscles to monitor muscle-connective tissue units in a way that does not coincide with the topography of muscles as morphological entities (Ref. 70, chapters 5 and 6). Rather a lot of receptors and nerve endings are located within the walls of the compartment (general fascia, intermuscular septum, and interosseal membrane). The same author (in his chapter 8) also concluded that extramuscular receptors of proprioception aid the perception of movement.

Second, some specific interactions are likely to occur more frequently and may have been learned by humans in the course of many years of development or training.

The actual stiffness of the general fascia and other borders of compartments seems to be very important for the quantity of myofascial force transmission (25, 28, 39). This is also implicitly apparent from the fact that muscle fibers of special muscles (e.g., tensor fascia latea muscles) insert fully on these connective tissue structures. Such a situation may be more common than usually realized, because muscles (such as gluteus maximus muscle, e.g., Ref. 25) may insert the greater majority (and in individual cases even 100%) of their muscle fibers onto these fascia. Of the muscles of our present study, the muscle fibers of rat EHL muscle originate from the anterior intermuscular septum. For humans, there are also indications that the superficial lamina of the thoracolumbar fascia will be tensed by activity of the latissimus dorsi, gluteus maximus, and erector spinae muscle, and even EDL muscle (69). For the cubital region, already in 1984 van Mameren and Drukker (46) viewed compartment walls as stress-conveying structures arranged, not in parallel, but in series with muscles. We propose that stiffening of the connective tissues constituting the walls of different compartments creates a firm origin or insertion for muscle fibers contained within the compartment. A stiff system of muscular compartments may act as a secondary skeleton. Therefore, activity of muscle fibers of this type of muscle is expected much more frequently than only for activities in which they may play a role as prime movers of a joint. Within such a stiff system, changes of length and relative position of muscle may allow very fine control of forces exerted via different pathways.

In summary, for passive and fully active synergists, our present work shows that relative position, with respect to each other and with respect to extramuscular connective tissues of the compartment in which they are located, is an important codeterminant of active and passive force exerted at a specific tendon of muscles. Because this is true also for EDL muscle, which has no connections to its neighboring synergists other than its myofascial connections, these effects are mediated by extra- and intermuscular myofascial force transmission.

REFERENCES

1. **Balice-Gordon RJ and Thompson WJ.** The organization and development of compartmentalised innervation in rat extensor digitorum longus muscle. *J Physiol* 398: 211–231, 1988.
2. **Banus MG and Zetlin AM.** The relation of isometric tension to length in skeletal muscle. *J Cell Comp Physiol* 12: 403–420, 1938.
3. **Baratta R, Solomonow M, Zhou BH, Letson D, Chuinard R, and D'Ambrosia R.** Muscular coactivation. The role of the antagonist musculature in maintaining knee stability. *Am J Sports Med* 16: 113–122, 1988.
4. **Blix M.** Die Länge und Spannung des Muskels: Einleitung. *Skandinavische Archiv für Physiologie* III: 295–318, 1893.
5. **Blix M.** Die Länge und Spannung des Muskels: Zweite Abhandlung. *Skandinavische Archiv für Physiologie* IV: 400–409, 1893.
6. **Blix M.** Die Länge und Spannung des Muskels: Dritte Abhandlung. *Skandinavische Archiv für Physiologie* V: 150–172, 1894.
7. **Blix M.** Die Länge und Spannung des Muskels: Vierte Abhandlung. *Skandinavische Archiv für Physiologie* V: 180–206, 1894.
8. **Bobbert MF, Ettema GC, and Huijing PA.** The force-length relationship of a muscle-tendon complex: experimental results and model calculations. *Eur J Appl Physiol* 61: 323–329, 1990.
9. **Chleboun GS, Patel TJ, and Lieber RL.** Skeletal muscle architecture and fiber-type distribution with the multiple bellies of the mouse extensor digitorum longus muscle. *Acta Anat (Basel)* 159: 147–155, 1997.
10. **Cope TC and Sokoloff AJ.** Orderly recruitment among motoneurons supplying different muscles. *J Physiol (Paris)* 93: 81–85, 1999.
11. **Denny-Brown DE.** The histological features of striped muscle in relation to its functional activity. *Proc R Soc Lond B Biol Sci* B104: 371–411, 1929.
12. **Van Eijden TM, Korfage JA, and Brugman P.** Architecture of the human jaw-closing and jaw-opening muscles. *Anat Rec* 248: 464–474, 1997.
13. **Ettema GJC.** Gastrocnemius muscle length in relation to knee and ankle joint angles: verification of a geometric model and some applications. *Anat Rec* 247: 1–8, 1997.
14. **Filippi GM and Troiani D.** Tetanic tension and muscle length of motor units in cat's peroneus longus. *Arch Ital Biol* 131: 227–234, 1993.
15. **Fukunaga T, Roy RR, Shellock FG, Hodgson JA, and Edgerton VR.** Specific tension of human plantar flexors and dorsiflexors. *J Appl Physiol* 80: 158–165, 1996.
16. **Gareis H, Solomonow M, Baratta R, Best R, and D'Ambrosia R.** The isometric length-force models of nine different skeletal muscles. *J Biomech* 25: 903–916, 1992.
17. **Gordon AM, Huxley AF, and Julian FJ.** The variation in isometric tension with sarcomere length in vertebrate muscle fibres. *J Physiol* 184: 170–192, 1966.
18. **Granzier H, Kellermayer M, Helmes M, and Trombitas K.** Titin elasticity and mechanism of passive force development in rat cardiac myocytes probed by thin-filament extraction. *Biophys J* 73: 2043–2053, 1997.
19. **Van der Helm FC, Veeger HE, Pronk GM, Van der Woude LH, and Rozendal RH.** Geometry parameters for musculoskeletal modelling of the shoulder system. *J Biomech* 25: 129–144, 1992.
20. **Henneman E.** The size-principle: a deterministic output emerges from a set of probabilistic connections. *J Exp Biol* 115: 105–112, 1985.
21. **Henneman E, Clamann HP, Gillies JD, and Skinner RD.** Rank order of motoneurons within a pool: law of combination. *J Neurophysiol* 37: 1338–1349, 1974.
22. **Henneman E and Olson CB.** Relations between structure and function in the design of skeletal muscle. *J Neurophysiol* 28: 581–598, 1965.

23. **Huijing PA.** Important experimental factors for skeletal muscle modelling: non-linear changes of muscle length force characteristics as a function of degree of activity. *Eur J Morphol* 34: 47–54, 1996.
24. **Huijing PA.** Muscle as a collagen fiber reinforced composite material: force transmission in muscle and whole limbs. *J Biomech* 32: 329–345, 1999.
25. **Huijing PA.** Muscular force transmission: a unified, dual or multiple system? A review and some explorative experimental results. *Arch Physiol Biochem* 170: 292–311, 1999.
26. **Huijing PA.** Intra-, extra- and intermuscular myofascial force transmision of synergists and antagonists: effects of muscle length as well as relative position. *J Mech Med Biol* 2: 1–15, 2002.
27. **Huijing PA and Baan GC.** Myofascial force transmission causes interaction between adjacent muscles and connective tissue: effects of blunt dissection and compartmental fasciotomy on length force characteristics of rat extensor digitorum longus muscle. *Arch Physiol Biochem* 109: 97–109, 2001.
28. **Huijing PA and Baan GC.** Extramuscular myofascial force transmission within the rat anterior tibial compartment: proximo-distal differences in muscle force. *Acta Physiol Scand* 173: 1–15, 2001.
29. **Huijing PA, Baan GC, and Rebel G.** Nonmyotendinous force transmission in rat extensor digitorum longus muscle. *J Exp Biol* 201: 682–691, 1998.
30. **Huijing PA, Maas H, and Baan GC.** Compartmental fasciotomy and isolating a muscle from neighbouring muscles interfere with extramuscular myofascial force transmission within the rat anterior tibial compartment. *J Morphology* In press.
31. **Jaspers RT, Brunner R, Baan GC, and Huijing PA.** Acute effects of intramuscular aponeurotomy and tenotomy on multitendoned rat EDL: indications for local adaptation of intramuscular connective tissue. *Anat Rec* 266: 123–135, 2002.
32. **Jaspers RT, Brunner R, Pel JJM, and Huijing PA.** Acute effects of intramuscular aponeurotomy on rat GM: force transmission, muscle force and sarcomere length. *J Biomech* 32: 71–79, 1999.
33. **Julian FJ and Morgan DL.** Tension, stiffness, unloaded shortening speed and potentiation of frog muscle fibres at sarcomere lengths below optimum. *J Physiol* 319: 205–217, 1981.
34. **Julian FJ and Moss RL.** Sarcomere length-tension relations of frog skinned muscle fibres at lengths above the optimum. *J Physiol* 304: 529–539, 1980.
35. **Kaufman KR, An KN, and Chao EY.** Incorporation of muscle architecture into the muscle length-tension relationship. *J Biomech* 22: 943–948, 1989.
36. **Ter Keurs HE, Iwazumi T, and Pollack GH.** The sarcomere length-tension relation in skeletal muscle. *J Gen Physiol* 72: 565–592, 1978.
37. **Lieber RL.** Muscle fiber length and moment arm coordination during dorsi- and plantarflexion in the mouse hindlimb. *Acta Anat (Basel)* 159: 84–89, 1997.
38. **Lieber RL and Boakes JL.** Muscle force and moment arm contributions to torque production in frog hindlimb. *Am J Physiol Cell Physiol* 254: C769–C772, 1988.
39. **Maas H, Baan GC, and Huijing PA.** Intermuscular interaction via myofascial force transmission: effects of tibialis anterior and extensor digitorum longus length on force transmission from rat extensor digitorum longus muscle. *J Biomech* 34: 927–940, 2001.
40. **Maas H, Baan GC, and Huijing PA.** The position of EDL muscle with regard to surrounding tissues is a major determinant of isometric EDL force: effects of inter- and extramuscular myofascial force transmission. In: *Proceedings of the 12th International Conference on Mechanics in Medicine and Biology, Lemnos, Greece*, edited by Karalis TK. Xanthi, Greece: Olga T. Karalis, 2002, p. 63–66.
41. **Magid A and Law DJ.** Myofibrils bear most of the resting tension in frog skeletal muscle. *Science* 230: 1280–1282, 1985.
42. **Van Mameren H and Drukker JA.** Functional anatomical basis of injuries to the ligamentum and other soft tissues around the elbow joint: transmission of tensile and compressive loads. *Int J Sports Med* 5, *Suppl*: 88–92, 1984.
43. **McClearn D.** Anatomy of raccoon (*Procyon lotor*) and coati (*Nasua narica* and *N. nasua*) forearm and leg muscles: relations between fiber length, moment-arm length, and joint-angle excursion. *J Morphol* 183: 87–115, 1985.
44. **Meijer K, Grootenboer HJ, Koopman HFJM, and Huijing PA.** Isometric length-force curves during and after concentric contractions differ from the initial isometric length-force curve in rat muscle. *J Appl Biomech* 16: 164–181, 1996.
45. **Miller JB.** The length-tension relationship of the dorsal longitudinal muscle of a leech. *J Exp Biol* 62: 43–53, 1975.
46. **Monti RJ, Roy RR, and Edgerton VR.** Role of motor unit structure in defining function. *Muscle Nerve* 24: 848–866, 2001.
47. **Monti RJ, Roy RR, Hodgson JA, and Edgerton VR.** Transmission of forces within mammalian skeletal muscles. *J Biomech* 32: 371–380, 1997.
48. **Morgan DL, Claflin DR, and Julian FJ.** Tension as a function of sarcomere length and velocity of shortening in single skeletal muscle fibres of the frog. *J Physiol* 441: 719–732, 1991.
49. **Muhl ZF.** Active length-tension relation and the effect of muscle pinnation on fiber lengthening. *J Morphol* 173: 285–292, 1982.
50. **Neter J, Wasserman W, and Kutner ME.** *Applied Linear Statistical Models: Regression, Analysis of Variance and Experimental Design.* Homewood, IL: Irwin, 1990.
51. **Pond CM.** The importance of connective tissue within and in between muscles. *Behav Brain Sci* 5: 562, 1982.
52. **Purslow PP.** The intramuscular connective tissue matrix and cell-matrix interactions in relation to meat toughness. *45th International Congress of Meat Science and Technology, Yokohama, Japan*. Society of Meat Science and Technology, 1999, p. 210.
53. **Purslow PP and Duance VC.** Structure and function of intramuscular connective tissue. In: *Connective Tissue Matrix*, edited by Hukins DWL. Boca Raton, FL: CRC, 1990, p. 127–166.
54. **Purslow PP and Trotter JA.** The morphology and mechanical properties of endomysium in series-fibered muscles: variations with muscle length. *J Muscle Res Cell Motil* 15: 299–308, 1994.
55. **Rack PMH and Westbury DR.** Effects of length and stimulus rate on tension in the isometric cat soleus muscle. *J Physiol* 204: 443–460, 1969.
56. **Ramsey RW and Street SF.** The isometric length-tension diagram of isolated skeletal muscle fibers of the frog. *J Cell Comp Physiol* 15: 11–34, 1940.
57. **Roszek B, Baan GC, and Huijing PA.** Decreasing stimulation frequency-dependent length-force characteristics of rat muscle. *J Appl Physiol* 77: 2115–2124, 1994.
58. **De Ruiter CJ, de Haan A, and Sargeant AJ.** Physiological characteristics of two extreme muscle compartments in gastrocnemius medialis of the anaesthetized rat. *Acta Physiol Scand* 153: 313–324, 1995.
59. **Smeulders MJC, Kreulen M, Baan GC, and Huijing PA.** Progressive surgical dissection for tendon transposition affects length-force characteristics of rat flexor carpi ulnaris muscle. *J Orthop Res* 20: 863–868, 2002.
60. **Sokoloff AJ, Siegel SG, and Cope TC.** Recruitment order among motoneurons from different motor nuclei. *J Neurophysiol* 81: 2485–2492, 1999.
61. **Solomonow M, Baratta R, Zhou BH, and D'Ambrosia R.** Electromyogram coactivation patterns of the elbow antagonist muscles during slow isokinetic movement. *Exp Neurol* 100: 470–477, 1988.
62. **Stein RB.** What muscle variable(s) does the nervous system control in limb movements? *Behav Brain Sci* 5: 535–577, 1982.
63. **Street SF.** Lateral transmission of tension in frog myofibres: a myofibrillar network and transverse cytoskeletal connections are possible transmitters. *J Cell Physiol* 114: 346–364, 1983.
64. **Street SF and Ramsey RW.** Sarcolemma transmitter of active tension in frog skeletal muscle. *Science* 149: 1379–1380, 1965.
65. **Trotter JA.** Interfiber tension transmission in series-fibered muscles of the cat hindlimb. *J Morphol* 206: 351–361, 1990.
66. **Trotter JA.** Functional morphology of force transmission in skeletal muscle. A brief review. *Acta Anat (Basel)* 146: 205–222, 1993.

67. **Trotter JA, Richmond FJ, and Purslow PP.** Functional morphology and motor control of series-fibered muscles. *Exerc Sport Sci Rev* 23: 167–213, 1995.
68. **Visser JJ, Hoogkamer JE, Bobbert MF, and Huijing PA.** Length and moment arm of human leg muscles as a function of knee and hip-joint angles. *Eur J Appl Physiol* 61: 453–460, 1990.
69. **Vleeming A, Pool-Goudzwaard AL, Stoeckart R, van Wingerden JP, and Snijders CJ.** The posterior layer of the thoracolumbar fascia. Its function in load transfer from spine to legs. *Spine* 20: 753–758, 1995.
70. **Van der Wal JC.** *The Organization of the Substrate of Proprioception in the Elbow Region of the Rat* (PhD thesis). Maastricht, The Netherlands: University of Limburg, 1988.
71. **Wicke RW and Zajac FE.** Isometric torque produced by cat hamstring muscle about the ankle as a function of hindlimb position. *Neuroscience Abstracts* 11: 684, 1981.
72. **Willems ME and Huijing PA.** Hip joint position and architecture of rat semimembranosus muscle: implications for length-force characteristics. *Acta Anat (Basel)* 152: 56–65, 1995.
73. **Yucesoy CA, Koopman, Huijing PA, and Grootenboer HJ.** Finite element modeling of intermuscular interactions and myofascial force transmission. In: *Proceedings of the 23rd Annual International Conference of the IEEE Engineering in Medicine and Biology Society Istanbul* [CD-ROM]. Piscataway, NJ: Institute of Electrical and Electronics Engineers, 2001. (0469.pdf)
74. **Zuurbier CJ, Heslinga JW, Lee de Groot MB, and Van der Laarse WJ.** Mean sarcomere length-force relationship of rat muscle fibre bundles. *J Biomech* 28: 83–87, 1995.
75. **Zuurbier CJ, Lee de Groot MBE, van der Laarse WJ, and Huijing PA.** Effects of in vivo like activation frequency on length dependent force generation of skeletal muscle fibre bundles. *Eur J Appl Physiol* 77: 167–180, 1998.