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Substantial effects of epimuscular myofascial force transmission on muscular mechanics have major implications on spastic muscle and remedial surgery

Can A. Yucesoy [a,*], Peter A. Huijing [b,c]

[a] *Biomedical Engineering Institute, Boğaziçi University, 34342 Bebek, Istanbul, Turkey*
[b] *Instituut voor Fundamentele en Klinische Bewegingswetenschappen, Faculteit Bewegingswetenschappen, Vrije Universiteit, Amsterdam, The Netherlands*
[c] *Integrated Biomedical Engineering for Restoration of Human Function, Faculteit Constructieve Technische Wetenschappen, Universiteit Twente, Enschede, The Netherlands*

Abstract

The specific aim of this paper is to review the effects of epimuscular myofascial force transmission on muscular mechanics and present some new results on finite element modeling of non-isolated aponeurotomized muscle in order to discuss the dependency of mechanics of spastic muscle, as well as surgery for restoration of function on such force transmission.

The etiology of the effects of spasticity on muscular mechanics is not fully understood. Clinically, such effects feature typically a limited joint range of motion, which at the muscle level must originate from altered muscle length–force characteristics, in particular a limited muscle length range of force exertion. In studies performed to understand what is different in spastic muscle and what the effects of remedial surgery are, muscle is considered as being independent of its surroundings. Conceivably, this is because the classical approach in muscle mechanics is built on experimenting with dissected muscles. Certainly, such approach allowed improving our understanding of fundamental muscle physiology yet it yielded implicitly a narrow point of view of considering muscle length–force characteristics as a fixed property of the muscle itself.

However, within its context of its intact connective tissue surroundings (the in vivo condition) muscle is not an isolated and independent entity. Instead, collagenous linkages between epimysia of adjacent muscles provide direct intermuscular connections, and structures such as the neurovascular tracts provide indirect intermuscular connections. Moreover, compartmental boundaries (e.g., intermuscular septa, interosseal membranes, periost and compartmental fascia) are continuous with neurovascular tracts and connect muscular and non-muscular tissues at several locations additional to the tendon origins and insertions. Epimuscular myofascial force transmission occurring via this integral system of connections has major effects on muscular mechanics including substantial proximo-distal force differences, sizable changes in the determinants of muscle length–force characteristics (e.g. a condition dependent shift in muscle optimum length to a different length or variable muscle optimal force) explained by major serial and parallel distributions of sarcomere lengths. Therefore, due to epimuscular myofascial force transmission, muscle length–force characteristics are variable and muscle length range of force exertion cannot be considered as a fixed property of the muscle.

The findings reviewed presently show that acutely, the mechanical mechanisms manipulated in remedial surgery are dominated by epimuscular myofascial force transmission. Conceivably, this is also true for the mechanism of adaptation during and after recovery from surgery. Moreover, stiffened epimuscular connections and therefore a stiffened integral system of intra- and epimuscular myofascial force transmission are indicated to affect the properties of spastic muscle. We suggest that important advancements in our present understanding of such properties, variability in the outcome of surgery and considerable recurrence of the impeded function after recovery cannot be made without taking into account the effects of epimuscular myofascial force transmission.

* Corresponding author. Tel.: +90 212 359 64 58; fax: +90 212 257 50 30.
E-mail address: can.yucesoy@boun.edu.tr (C.A. Yucesoy).

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1. Major effects of epimuscular myofascial force transmission on muscular mechanics

1.1. Muscle length–force characteristics are not unique properties of individual muscles

In classical muscle mechanics experiments performed to determine muscle isometric length–force characteristics and dynamic characteristics, muscle force is measured in the following conditions: (1) the targeted muscle is fully dissected except for its innervation and blood supply (2) muscle force is measured at one tendon exclusively. In such approach, the muscle studied in situ is considered as ''fully isolated'' from its surroundings (e.g. Frueh et al., 2001). As a consequence, two idealizations have been made, which became in time the established principles of skeletal muscle mechanics: (1) with the implicit assumption: the muscle force exerted at the tendon from which measurements are taken was considered to be equal to the force exerted at the other tendon. (2) Length–force characteristics determined were considered as unique properties of the specific target muscle studied. As a consequence, muscles have been considered commonly as fully independent functional units. Also distinguishing the muscles morphologically contributed to them as being regarded as distinct functional units.

However, recent studies have shown that, due to myofascial force transmission, such functional independence and unique muscle length–force characteristics are not representative, if the muscle is considered within the context of its intact surroundings (the condition in vivo). The effects of this type of force transmission on muscular mechanics will be considered below.

1.1.1. Unequal forces exerted at origin and insertion of a muscle

In order to address the effects of myofascial force transmission, recent muscle mechanics experiments have been designed differently than the classical approach: (i) the experimental muscle belly was not dissected (i.e., its epimuscular connections were left intact) and (ii) instead of measuring the force exerted at only one tendon, the forces exerted at both proximal and distal tendons were measured simultaneously. This approach showed the characteristic effect of epimuscular myofascial force transmission on muscle length–force characteristics: proximo-distal force differences (e.g. Huijing and Baan, 2001a, 2003; Maas et al., 2001, 2003a; Yucesoy et al., 2003b,a; Meijer et al., 2006). Such force differences showed to be substantial in the experimental conditions studied provide a clear evidence for the existence of a potentially important pathway for force transmission additional to the myotendinous pathway.

Such proximo-distal force differences are indicative of differential mechanical effects at muscle origin and insertion. For bi- or polyarticular muscle this has special functional consequences in both healthy and pathological conditions, since such differential effects are exerted at the joints spanned.

A potentially even more important effect of epimuscular myofascial force transmission is that muscle has additional origins and\or insertions since a part of the muscular force is transmitted from the muscle and is exerted at other muscles or non-muscular structures see also elsewhere in the present issue of this journal (Huijing, 2007; Huijing et al., 2007; Meijer et al., 2007; Rijkelijkhuizen et al., 2007).

On the other hand, even in experiments performed on fully dissected muscle (like in most of the classical tests), the minimal condition is to keep blood supply and innervation of a muscle intact as much as possible, since otherwise, the physiological state of the muscle cannot be sustained. For that reason, specific parts of the neurovascular tract (i.e., extramuscular connective tissues in which blood vessels and nerves are embedded) was always left intact (usually proximally located with respect to the muscle). Therefore, the fully dissected experimental muscle in situ cannot actually be considered to be truly isolated mechanically from its surroundings, as the remaining extramuscular connections are still capable of transmitting muscle force, leading to notable proximo-distal force differences (Yucesoy et al., 2003b). Despite that, in the majority of earlier experimental work, it was assumed that a muscle in situ is not different from a truly isolated muscle for which proximal and distal forces has to be identical because of the serial arrangement of muscle fibers and tendons. However, work on fully dissected rat medial gastrocnemius muscle (GM) indicates that physiologist, almost intuitively, may have selected conditions (i.e. knee joint angles) at which extramuscular myofascial effects were minimal (Rijkelijkhuizen et al., 2005). At the knee angle commonly selected in earlier experiments (Woittiez et al., 1985; Jaspers et al., 1999; Haan et al., 2003) the myofascial effect was negligible. Nevertheless, deviation of GM relative position with respect to the neurovascular tract enhanced the extramuscular myofascial effects (Rijkelijkhuizen et al., 2005).

1.1.2. Length range of force exertion is a condition dependent variable rather than a fixed muscle property

Major parameters of muscle length–force characteristics are *muscle optimal force* (the maximum force exerted by an active muscle), *muscle optimum length* (the length at which the muscle exerts its optimal active force) and *muscle active slack length* (the shortest length at which the muscle can still exert non-zero force) as well as the maximal length

of active force exertion. Frequently, the length range between active slack and optimum length is taken as an experimental indicator of the potential joint range of motion and therefore the movement capability within a certain joint.

Movement capability is impeded in patients with spastic paresis and correction of the most common joint position, as well as joint range of motion are the primary goals of several techniques of remedial surgery. Therefore, it is important to understand determinants of muscle length range of active force exertion and the way it is affected by conditions of movement among which those of epimuscular myofascial force transmission. Fig. 1 shows an example of such effects for the extensor digitorum longus (EDL) muscle of the rat. The isometric length–force characteristics of this muscle were measured in two different conditions (Yucesoy et al., 2003a):

(i) With synergistic muscles within the anterior crural compartment, tibialis anterior (TA) and extensor hallucis longus (EHL) muscles present and the connective tissues at the bellies of these muscles left intact. Note that in this condition, both inter- and extramuscular myofascial force transmission mechanisms were active for EDL muscle (referred to as the ''epimuscular connections'').
(ii) With TA and EHL muscles removed subsequent to fasciotomy. Therefore, the pathway of intermuscular myofascial force transmission was removed. However, extramuscular myofascial force transmission was still possible for EDL.

The length–force characteristics of EDL muscle in these conditions showed major differences. For EDL with epimuscular connections: (1) the magnitude of the proximo-distal force differences were much higher. (2) Due to shifting of muscle optimum length to a higher length, the length range of active force exertion was increased substantially. (3) Distally determined optimal force of EDL muscle was significantly higher than that of EDL with extramuscular connections exclusively.

These results suggest that effects of myofascial force transmission on muscle length–force characteristics are dominated by intermuscular myofascial force transmission. However, it is important to note the possible differences in the relative contributions of components of intermuscular myofascial force transmission to such effects: intermuscular myofascial force transmission occurs both via direct linkages between adjacent muscles and via indirect connections provided by extramuscular connections (e.g., common neurovascular tracts). Maas et al. (2005) tested the relative contribution of the direct intermuscular connections exclusively by blunt dissection of intermuscular connective tissue linkages between EDL muscle and its synergists TA and EHL (instead of removing them and therefore blocking both direct and indirect intermuscular myofascial force transmission). Such approach yielded much smaller effects on muscle length–force characteristics. Nevertheless, there were differences in experimental conditions compared to Yucesoy et al. (2003a): (1) an initial full compartmental fasciotomy was done. As the integrity of the compartment is disrupted this may conceivably reduce the stiffness of intermuscular connections. (2) EDL muscle was kept at con-

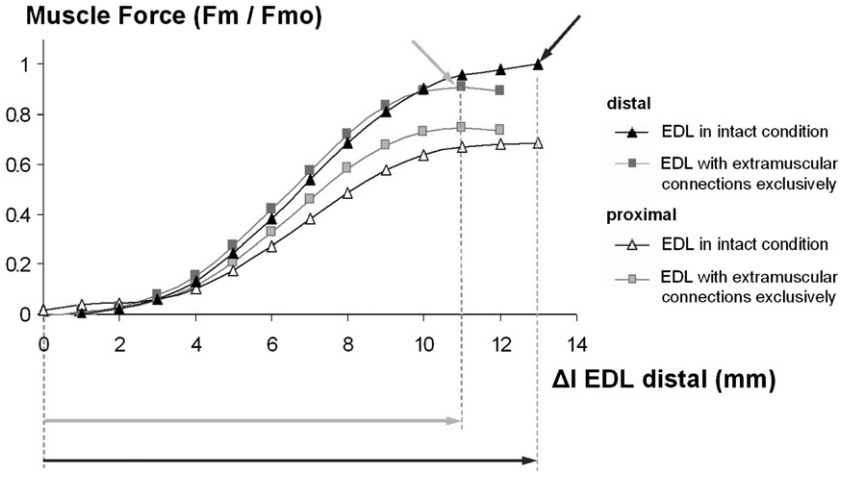

Fig. 1. Effects of different myofascial components of epimuscular force transmission on EDL isometric muscle length–force characteristics. The length–total force characteristics of EDL muscle with intact inter- and extramuscular connections (intact condition) are compared to those of EDL muscle with extramuscular connections exclusively. The length of EDL muscle was changed by moving its distal tendons exclusively. Note, however, that before imposing such distal length changes (1) TA + EHL complex was allowed to shorten distally to such a length that the distal TA + EHL force was equal to approximately 2 N, subsequently the length of this muscle complex was kept constant. (2) The proximal EDL tendon was displaced by 2 mm in the distal direction with respect to a reference position (EDL shortening), and subsequently it was kept at this position throughout the experiment. Length is expressed as a function of deviation (Δl EDL) from active slack length. The length range of force exertion of EDL muscle is significantly higher in intact condition compared to that measured with extramuscular connections exclusively (marked with black and gray horizontal arrows respectively). Moreover, the distal optimal force of EDL muscle is higher in intact condition compared to that measured with extramuscular connections exclusively (black and gray oblique arrows respectively).

stant length and TA + EHL muscle complex was lengthened distally. Such procedure in addition the differently located EDL proximal and distal tendons compared to Yucesoy et al. (2003a) imposes sizable differences in muscle relative position changes (major effects of which are addressed in a section below) to occur. Therefore, it is likely that the relative importance of intermuscular connections is also affected by the conditions in which muscles function. New studies are indicated to address such condition dependencies.

On the other hand, the shift of muscle optimum length shown in Fig. 1 to a higher length indicates an increased heterogeneity in the lengths of sarcomeres (Willems and Huijing, 1994; Huijing, 1998). To study explicitly the effects of myofascial force transmission on distribution of sarcomere lengths, a finite element model was developed. Instead of the common approach of using elements in which both active and passive properties of muscle tissue are lumped (e.g. Johansson et al., 2000; Oomens et al., 2003), a two-domain approach was employed: the intracellular and the extracellular matrix domains of skeletal muscle were represented by two separate but elastically linked meshes (Yucesoy et al., 2002).

This model extended to include inter- and extramuscular connections did show a major heterogeneity (Yucesoy et al., 2003a) for the lengths of sarcomeres arranged in series within the muscle fibers (serial distribution) as well as for the mean fiber sarcomere lengths (parallel distribution). However, earlier it was shown for fully dissected muscle that a higher length range of force exertion due to increased heterogeneity in mean fiber sarcomere lengths causes a decrease in muscle optimal force (Huijing, 1996). Therefore, the higher distal optimal force of EDL muscle in intact condition despite an increased length range of force exertion shows that the force of this muscle exerted at its distal tendon is not determined solely by the force generated within its own sarcomeres. Such additional force is explained as the force transmitted from the synergistic muscles: the force generated within the sarcomeres of TA + EHL muscle complex is exerted onto the distal tendon of EDL muscle after being transmitted via intermuscular myofascial pathways.

An earlier finite element modeling study to investigate the mechanical interaction between two adjacent muscles (Yucesoy et al., 2001) showed results in support of such force transmission: the distal force of the intermuscularly connected muscle was much higher than that of a truly isolated muscle after identical distal lengthening. However, the mean fiber stress found in the isolated muscle was well above that of the intermuscularly connected muscle at high lengths.

Due to the different components of epimuscular myofascial force transmission, also the shape of the muscle length–force characteristics was shown to change as a function of different conditions in which the muscle functions (Huijing and Baan, 2001b; Maas et al., 2005; Yucesoy et al., 2005). This is exemplified in Fig. 2. The extensor hallucis longus

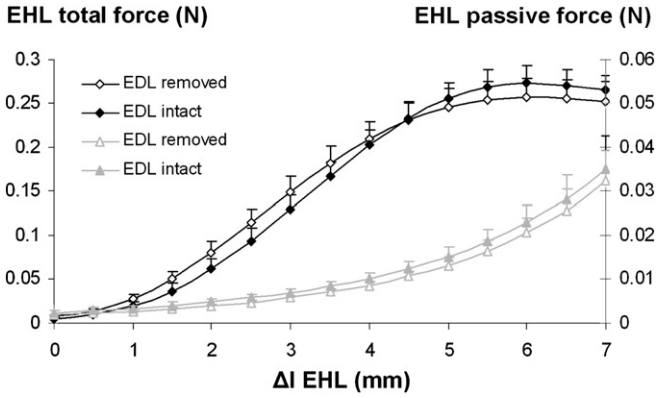

Fig. 2. Effects of different myofascial components of epimuscular force transmission on EHL isometric muscle length–force characteristics. The isometric muscle total as well as passive EHL length–force curves with intact inter- and extramuscular connections (i.e. with EDL is intact) are compared to those of EHL muscle with extramuscular connections exclusively (i.e. after removing EDL). With EDL intact, its proximal tendon was kept at a reference position (corresponding to knee angle approximating 100°) whereas, the distal EDL tendon was displaced by 2 mm in the proximal direction with respect to a distal reference position (corresponding to ankle angle approximating 90°). During the measurements, the length of EDL muscle was not changed. Isometric EHL muscle forces were measured after distal lengthening. Length is expressed as deviation (Δ*l* EHL) from active slack length. In addition to shape changes shown for EHL muscle length–total force characteristics as a result of altered conditions for myofascial force transmission, the non-zero passive forces at all muscle lengths and in both conditions are remarkable. The latter shows that the epimuscular connections are prestrained.

muscle (EHL) was lengthened distally (i) with its synergist, EDL present and (ii) after EDL was removed. As a result of such altered conditions, the magnitude of EHL muscle total forces showed variable differences at different isometric muscle lengths yielding an altered shape of the length–force characteristics.

It is concluded that the force exerted at the origin and insertion of muscle is subject to change not only as a result of altered muscle length but also due to the mechanical conditions in which the muscle is functioning. Such major effect of epimuscular myofascial force transmission makes muscle length–force characteristics variable and therefore, muscle length range of force exertion cannot be considered as a fixed muscular property.

1.1.3. Prestrain in the epimuscular connections indicate sufficient stiffness for force transmission also in vivo

According to results for fully dissected muscle, passive muscle forces are expected to become non-zero at higher muscle lengths, typically near muscle optimum length. However, Fig. 2 shows for both experimental conditions that non-zero passive EHL forces were measured for all muscle lengths, including active slack length. This shows that the epimuscular myofascial force transmission pathways are prestrained. Substantial prestrain was shown for the epimuscular connections of EDL muscle as well (Yucesoy et al., 2005). Such mechanical properties are expected to originate from prestrained connective tissue

structures such as anterior intermuscular septum and interosseal membrane (Fig. 2a), to which the neurovascular tract to EHL and EDL muscles is connected (Fig. 2b). Prestrained fibrous structure is a common feature found in fiber reinforced composites. Prestrain of epimuscular myofascial force transmission pathways is important because it suggests that these pathways are usually sufficiently stiff to transmit force and that the range of neutral positions (i.e., positions with net zero epimuscular force) of a muscle relative to the surrounding muscular and non-muscular structures is likely to be quite limited. Therefore, we concluded that the effectiveness of epimuscular myofascial force transmission on in vivo muscle length–force characteristics is a likely event.

1.2. Lengths of sarcomeres within muscle fibers show major heterogeneity

If the force exerted by an activated sarcomere is not counteracted by an external force it will shorten to its active slack length. It is well known that the sarcomeres arranged in series within a muscle fiber are in mechanical interaction. However, only few researchers have dealt with the dynamic and disordering effects of intersarcomere dynamics on sarcomere lengths (Goldspink et al., 1974; Wohlfart et al., 1977; Hayat et al., 1978; Williams and Goldspink, 1978; Julian and Morgan, 1979a,b; Tabary et al., 1981; Edman and Reggiani, 1984; Morgan et al., 2000). Some researchers consider sarcomere force–velocity characteristics as a factor limiting major serial sarcomere length distributions (van Soest and Bobbert, 1993) and in isolated muscle fiber, their existence were often acknowledged but not considered seriously (e.g., Denoth and Danuser, 2001) as they were considered artifacts (e.g., Edman and Reggiani, 1984) or their effect removed (using length feed back mechanisms) because it interfered with the specific goal of measuring single sarcomere length force curves (e.g., Gordon et al., 1966; Huxley, 1974).

In any case, within the classical point of view in which individual muscle fibers are considered as independent functional units, intersarcomere dynamics would remove any distribution for such interaction at the ascending limb of the length–force curve and for muscle at lengths over optimum length, all sarcomere lengths would be limited to the descending limb of the length–force curve (Julian and Morgan, 1979a,b).

The concept that muscle fibers function independent of each other is mechanically incomplete because it relies on the idea that muscle fibers and intramuscular connective tissue stroma are connected at the myotendinous junctions exclusively.

Therefore, taking into account the integral system of myofascial force transmission, much more pronounced sarcomere length heterogeneities are conceivable for two reasons:

(1) The two domains comprising the skeletal muscle tissue (intracellular and extracellular matrix) are connected mechanically along the full periphery of muscle fibers with complex structures: trans-sarcolemmal molecules connect the cytoskeleton to laminin which is connected to the basal lamina (for a review see Berthier and Blaineau, 1997), which in turn is connected to the endomysium, that forms a 3D structure of tunnels (Trotter and Purslow, 1992) within which the muscle fibers are operating. Therefore, in the first place the length of a sarcomere cannot be determined exclusively by its interaction with the sarcomeres arranged in series with it in the same muscle fiber. Instead, also the forces exerted on it by the fiber reinforced extracellular matrix, as well as the forces of the sarcomeres located in the neighboring muscle fibers should be considered to play a major role.
(2) Due to the continuity of the intramuscular connective tissue stroma and the epimuscular connective tissue of a muscle, the epimuscular loads on the muscle are expected to take part in the balance of forces and therefore in determining the sarcomere length.

It should be noted that such a net epimuscular force is not distributed uniformly: (i) Even though the neurovascular tract is connected to the periphery of the muscle along most of their belly lengths, branches of it carrying the major supply of blood vessels and nerves to the muscle enter only at specific locations. Particularly the collagen fiber reinforced nerves make contact with the muscle fiber at specific locations. In a more integrative view the intramuscular connective tissues are the intramuscular part of the neurovascular tract since they also embed nerves as well as blood and lymph vessels. (ii) Distinguishable parts of the neurovascular tract have variable mechanical stiffness (Yucesoy et al., 2003b) i.e., proximal parts of the neurovascular tract (Fig. 3) are stiffer than the remainder. (iii) Intermuscular forces are exerted onto the muscle through the direct connections between adjacent muscles. However, also such connections are not expected to be distributed homogeneously over the epimysium. Therefore, intuitively one expects heterogeneous effects of epimuscular myofascial force transmission on sarcomere lengths yielding both serial and parallel distributions.

Results of recent experiments, i.e., major proximo-distal force differences (e.g. Huijing et al., 2003; Yucesoy et al., 2003a) and sizable changes in the shape of muscle length–force characteristics (Huijing and Baan, 2001b; Yucesoy et al., 2003a, 2005; Maas et al., 2005) constitutes indications that the distributions of sarcomeres within the muscle fibers and within the muscle are altered due to epimuscular myofascial force transmission. For example, despite its length being kept constant, significant decreases or increases found in TA + EHL muscle complex force after distal (Huijing and Baan, 2003; Yucesoy et al., 2003a) or proximal lengthening (Huijing and Baan, 2003;

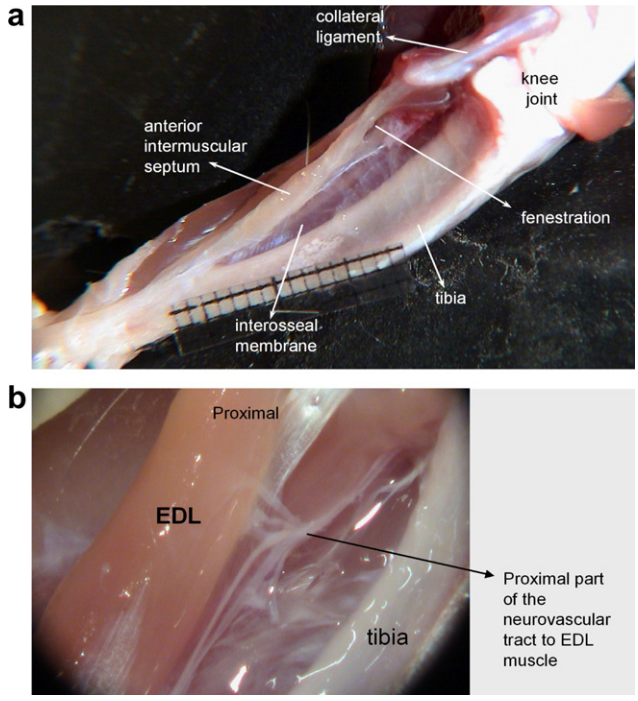

Fig. 3. Components of pathways of extramuscular myofascial force transmission. (a) The dorsal and medial boundaries of the anterior crural compartment of the rat after the muscles contained within the compartment (i.e. EDL, TA and EHL) are removed. In addition to the crural fascia that covers the surface of TA (not shown), the anterior crural compartment is delimited by the anterior intermuscular septum, the anterior part of the interosseal membrane and the periost of the tibia. The fenestration indicated (by labeled arrow), is shown within the anterior intermuscular septum. Through this fenestra the neurovascular tract passes between the peroneal and anterior crural compartments. Note that this neurovascular tract contributes to indirect connections between antagonistic muscles within the two compartments. (b) The more proximal and stiffer part of the neurovascular tract of the anterior crural compartment (for more pictures see Maas et al., 2001; Huijing et al., 2003). Note that the anterior intermuscular septum is continuous with specialized capsule structures of the joint (i.e. the lateral collateral ligament, shown clearly in a). This suggests that, in addition to intermuscular effects epimuscular myofascial force transmission may have additional and direct effects on the joint motion (e.g. increased joint stability). Such additional effects of epimuscular myofascial force transmission may also include bone remodeling.

Huijing et al., 2003) of EDL muscle respectively, is very unlikely without any change in the lengths of sarcomeres within the muscle fibers. In addition, increased length range of force exertion shown experimentally suggests that both the serial distribution and parallel distribution became more pronounced.

Although this points out a substantial need to study sarcomere length distributions experimentally, this involves a considerable difficulty as the targeted muscle is surrounded by compartmental connective tissues and other muscles, which condition obscures the view. On the other hand, if efforts are made to achieve a greater access to the muscle, the myofascial force transmission mechanism may become hindered, in conflict with the main purpose of the experiment. For example, gradual isolation of EDL muscle as a result of systematic manipulation of its inter- and extramuscular connections causes sizable differences in muscle length force characteristics (Huijing and Baan, 2001b).

However, finite element modeling coupled to experiments allowed studying the effects of epimuscular myofascial force transmission on sarcomere length distributions (Maas et al., 2003a; Yucesoy et al., 2003b,a, 2006b).

Fig. 4 exemplifies the detailed results of such modeling showing the fiber direction strain and stress distributions within the EDL muscle of the rat with extramuscular connections exclusively. Fiber direction strain represents a measure of normalized change of length and reflects the lengthening (positive strain) or shortening (negative strain)

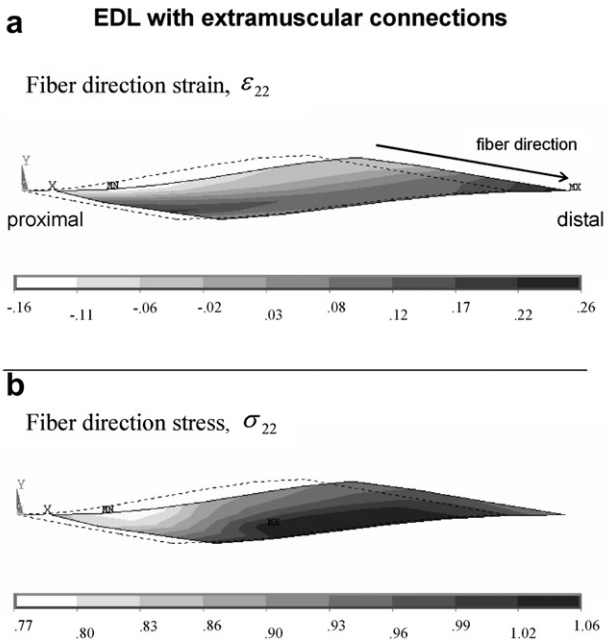

Fig. 4. Finite element model results of EDL muscle of the rat with extramuscular connections exclusively. (a) Distributions of fiber direction strain. Fiber direction strain within the fiber mesh of the modeled EDL muscle was used to assess the non-uniformity of sarcomere lengths arranged in-series within muscle fibers (serial distribution). It was assumed that, at the initial muscle length and in the passive state, the sarcomeres arranged in series within muscle fibers have identical lengths. Positive strain reflects the lengthening and negative strain reflects the shortening of sarcomeres with respect to the initial length. Note that zero strain in the model represents the undeformed state of sarcomeres (i.e., sarcomere length $\cong 2.5$ μm) in the passive condition at initial muscle length (28.7 mm). (b) Distributions of fiber direction stress. Fiber direction stress is studied to investigate the contribution of sarcomeres at different locations to active muscle force. It has two components (i) a component representing the active length–force characteristics and (ii) a component representing the stress due to the passive resistance of the intra-sarcomeric cytoskeleton (assumed to be dominated by titin). The function representing the active length–force characteristics is scaled such that at optimum length, the fiber direction strain is zero and the maximal stress value is unity (for more details on the model see Yucesoy et al., 2002). Note that prior to distal lengthening; the proximal end of the muscle model was moved 2.0 mm in the distal direction, (EDL shortening), and subsequently it was kept at this position, compatible with earlier experiments (Yucesoy et al., 2003a). The dotted line contour indicates muscle geometry at the initial length and position. The fiber direction as well as the proximal and distal ends of the muscle is shown in the lower panel of part (a).

from the initial condition (usually at optimum length). Even though the model does not contain morphological representations of sarcomeres these local strains constitute estimates of local sarcomere lengths. A major distribution of strain was shown within the modeled muscle at high length (Fig. 4a): sarcomeres located at the proximal ends of muscle fibers are shorter than the ones located distally. A remarkable result was that despite the high length of the muscle (over optimal length) the proximal sections of muscle fibers did shorten (by up to 16%) showing that the sarcomeres at these sections are at the ascending limb of their length–force curves. On the other hand, the distal sections of identical muscle fibers were lengthened (by up to 26%) and therefore the sarcomeres at these sections are at the descending limb of their length–force curves. Such results do challenge the concept of sarcomere length distribution being limited to the descending limb of the length–force curve, and show that these distributions range from the domains of the ascending limb to the descending limb of their sarcomere length–force curves. Such results also suggest that within the context of surrounding muscles and connective tissues, distributions of sarcomere length should be expected as physiological phenomena that may affect acute function as well as adaptation of muscle.

Reflecting the effects of sarcomere length distributions, stress in the fiber mesh shows the variable contribution of sarcomeres at different locations to active muscle force. Another remarkable model result is that, in agreement with the proximo-distal force differences in favor of the distal force (Fig. 1) characteristic for distal lengthening of a single muscle, the fiber direction stresses in the distal sections of the muscle fibers are much higher than those in the proximal sections (Fig. 4b). Again challenging a commonly accepted concept, this shows that also the muscle fiber cannot be considered as a unit of force exertion, exerting equal forces at both ends and substantial distributions of force exerted along the length of muscle fibers.

1.3. The effects of epimuscular myofascial force transmission are determined by muscle relative position

In this paper the focus is on the effects of epimuscular myofascial force transmission on muscle length–force characteristics. Therefore, the experimental and modeling work discussed here involve length changes of a target muscle. However, changes in muscle length also cause changes in the position of muscle relative to its neighboring muscles as well as non-muscular structures. Such relative position change is indicated as the major determinant of the effects of epimuscular myofascial force transmission:

(1) As the length of the target muscle increases, the direct collagenous intermuscular connections between the two collagen reinforced extracellular matrices of adjacent muscles are stretched. This will increase the epimuscular load to be exerted on the connective tissue stroma of the muscles. Such an effect requires differential length changes of neighboring muscles to occur due to for example differences in moment arms. However, a major contributor to this change in relative muscular position is the number of joints spanned by different muscles: a poly-articular muscle is expected to show more pronounced length changes relative to its mono-articular neighbor and therefore a substantial change in its relative position.
(2) Upon any length change, the position of a muscle with respect to the fixed bony structures of the musculoskeletal system will change causing loading of the extramuscular connections and transmission of force. It should be noted that these connections feature complex mechanical properties. Therefore, such relative position changes are expected to cause variation in the stiffness of the extramuscular connections and therefore a differential effect on muscle length–force characteristics. A major reason for such variable stiffness is the plausible nonlinear force–deformation characteristics of the extramuscular connective tissues, similar to other connective tissue structures like aponeurosis or tendon (Ettema and Huijing, 1989; Strumpf et al., 1993; Scott and Loeb, 1995): each unit change of relative position of a muscle with respect to non-muscular structures is expected to yield a quantitatively different mechanical effect. In addition, due to inhomogeneous mechanical properties (e.g. Yucesoy et al., 2003b) and conceivable morphological changes in the extramuscular connective tissues (e.g. alterations in the collagen fiber orientations) occurring with the changing muscle length cause changing magnitude as well as direction of extramuscular at different muscle relative positions. Most importantly, the presence of prestrain in the extramuscular connective tissues as discussed above yields a varying stiffness.

Recent experiments showed remarkable results for the differential effects of muscle relative position changes, as imposed by muscle lengthening. Huijing and Baan (2003) studied the effects of equal proximal and distal lengthening of EDL muscle on muscle length–force characteristics. These authors showed for both activated and passive muscle that after distal lengthening, the distal EDL forces were higher than the proximal EDL forces whereas, after proximal lengthening, the proximo-distal force difference favored the proximal force. Moreover, the effects were not symmetric (e.g. distal lengthening yielded a lower distal optimal force than the proximal optimal force measured after proximal lengthening). In another study, after locating the proximal tendon at different positions, lengthening EDL muscle distally yielded substantial differences in muscle length–force characteristics (Maas et al., 2003b). Relative to a reference position, repositioning the proximal tendon in proximal direction caused the active slack length, as well as muscle optimum length to shift to higher muscle lengths. In addition, the magnitude of muscle optimal force

was different for different muscle relative positions. The common remarkable finding in these experiments is those different muscle length–force curves were obtained for the identical muscle as a result of different relative positions. Therefore, a major conclusion is that, in addition to muscle length, muscle relative position is a major determinant of muscle force, due to epimuscular myofascial force transmission.

Clear and convincing further evidence was shown supporting this conclusion with specially designed experiments: the muscle–tendon complex length of the target muscle was fixed and its relative position was changed exclusively. These studies showed experimentally for EDL with extramuscular connections exclusively (Maas et al., 2003a) and for EDL with epimuscular connections (Maas et al., 2004) that (i) for a majority of the positions, the proximal and distal muscle forces are unequal and (ii) the proximo-distal force difference increases at more proximal or more distal muscle relative positions. Moreover, finite element modeling showed that such position changes exclusively cause increased heterogeneity of sarcomere lengths within the muscle fibers (Maas et al., 2003a; Yucesoy et al., 2006b).

2. Effects of aponeurotomy in relation to myofascial force transmission

In spastic paresis, spastic muscles are kept short due to excessive reflex activity, which in time causes the development of a permanent shortness (for a proposed mechanism see also Malaiya et al., 2007). Due to such contractures, the affected muscles' length range of force exertion becomes limited, which may cause severe changes in preferred joint position, restriction in joint range of motion and therefore movement disorders. Several surgical techniques are used for the correction of problems of movement range and favored joint position in spastic paresis. One of these is aponeurotomy (e.g. Baumann and Koch, 1989) i.e. the cutting of the intramuscular aponeurosis in the direction perpendicular to its longitudinal direction (Fig. 5). After aponeurotomy, the joint angle is adjusted such that the target muscle is brought to a high length after which the limb is usually placed in a cast for recovery. The goal of this technique is to alter muscle length–force characteristics: (1) to lengthen the muscle (i.e., to increase its length range of force exertion) if the muscle is overly short and (2) to weaken the muscle (i.e., to reduce muscle force) if there is force imbalance of antagonistic muscles. Such surgical intervention aiming primarily at changes in muscle length–force characteristics is of particular interest to the content of this paper.

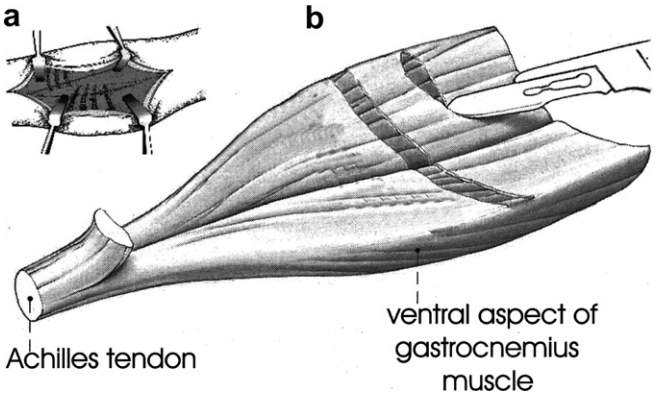

Fig. 5. Intramuscular lengthening of the triceps surae muscles by aponeurotomy. (a) In situ during surgery. (b) The nature of the intervention shown on gastrocnemius muscle. The image is redrawn after Baumann and Koch (1989) and Saraph et al. (2000). Surgical aponeurotomy described by Baumann and Koch (1989) involves at least limited fasciotomy followed by one or multiple incisions in the aponeurosis of the target muscle (in the illustrated case triceps surae muscles). The incisions are made transversely i.e., in the direction perpendicular to the longitudinal direction of aponeurosis.

Clinical success was reported in restoring function after the intervention (e.g. Nather et al., 1984; Reimers, 1990; Nene et al., 1993). However, the recurrence rate is also fairly high (Ejeskar, 1982; Olney et al., 1988), suggesting a lack of clear understanding of the mechanism determining the effects of aponeurotomy. Nevertheless, experiments on rat muscles (fully dissected except for innervation and blood supply) provided information regarding both acute (Jaspers et al., 1999, 2002; Brunner et al., 2000) and long term (Brunner et al., 2000) physiological effects of this intervention. These authors showed changes of muscle length–force characteristics, as well as sizeable changes in muscle fiber lengths, indicating inhomogeneity in the lengths of sarcomeres.

2.1. Dominance of effects on myofascial force transmission over myotendinous force transmission

At the level of the target muscle, even though the intervention involves the aponeurosis and its adjacent epimysium exclusively, lengthening of the muscle during the operation yields progressive rupturing of the connective tissue within the muscle belly below the location of the intervention (Jaspers et al., 1999). Therefore, the intervention causes a discontinuity not only in the aponeurosis (part of myotendinous force transmission pathway), but also in the collagen reinforced extracellular matrix (cECM) i.e., myofascial pathway. In our view, a lack of this concept in considerations of aponeurotomy is a main reason for the lacking understanding of the mechanisms causing acute effects of aponeurotomy and its adaptation during recovery: as the name of the surgery implies such effects have been ascribed exclusively to the interference with the myotendinous force transmission and the altered mechanism of myofascial force transmission has not been accounted for. At this point, finite element modeling offers a big advantage for studying the effects of aponeurotomy (Yucesoy et al., in press). In contrast to experimental studies in which both force transmission mechanisms are affected simultaneously and inseparably, finite element modeling allows distinguishing the effects of (1) a discontinuity within the

sectioned aponeurosis (interfering with the myotendinous force transmission pathway exclusively) and (2) discontinuity within the intramuscular extracellular matrix (partially interfering with intramuscular myofascial force transmission pathways). Therefore, the dominant mechanism of force transmission to determine the acute effects can be identified. To give a specific emphasis, we will present results for a truly isolated muscle below.

Fig. 6 shows the length–force characteristics of a modeled rat muscle after proximal aponeurotomy and in intact condition providing an example of such analysis. If aponeurotomy is modeled with only a discontinuity in the proximal aponeurosis (intact cECM) the effects of the intervention on muscle length–force characteristics are minor: only active slack length was increased and limited reductions in muscle active forces were found (e.g., muscle optimum force decreased by only 1%). In contrast, if additional discontinuities of the muscles' extracellular matrix are accounted for, major effects are shown: a further increase in active slack length (by 1.2 mm, i.e., by 18.5% of length range of force exertion of intact muscle) occurred together with a greater increase in the optimum length (by 2.0 mm, i.e., by 31% of length range of force exertion of intact muscle) causing net increase in length range of force exertion. Moreover, muscle active forces were reduced substantially (e.g., muscle optimum force decreased by 21%).

In general, also regarding muscle geometry and fiber direction strain distributions, the aponeurotomized muscle with intact cECM showed very minor differences compared to the intact muscle (Fig. 7a and b). In contrast, the tearing within the muscle belly divides the muscle into two populations of muscle fibers (proximally and distally located within the muscle: note that this is quite different from distinguishing proximal and distal sarcomeres within fibers of the muscle) and the altered myofascial force transmission caused major sarcomere length distributions describing the mechanism of the effects of the intervention for a truly isolated muscle (Fig. 7c): (i) sarcomeres of the distal fiber population were in general much shorter than those within the proximal population. Note that the most proximal sarcomeres are shortened maximally by 47%. (ii) From proximal ends of muscle fibers to distal ends, the serial distribution of sarcomere lengths within the distal fiber population ranged from the lowest length to high lengths within the distal population and in a reversed manner within the proximal fiber population. Note that within the proximal fiber population, sarcomeres at the proximal ends of muscle fibers are lengthened by up to 69%. Such major distribution of sarcomere lengths explains the shifts in muscle active slack and optimal lengths and the short sarcomeres within the distal population were shown to be responsible with the force reduction after intervention. These results show clearly that rather than interfering with the myotendinous force transmission, alteration of the intramuscular myofascial force transmission mechanism determines the acute effects of aponeurotomy on muscle length–force characteristics.

2.2. Are the effects of aponeurotomy unique?

The continuity of intra- and extramuscular myofascial force transmission pathways and the major effects of extramuscular myofascial force transmission on the mechanics of intact muscle summarized in this paper suggest strongly

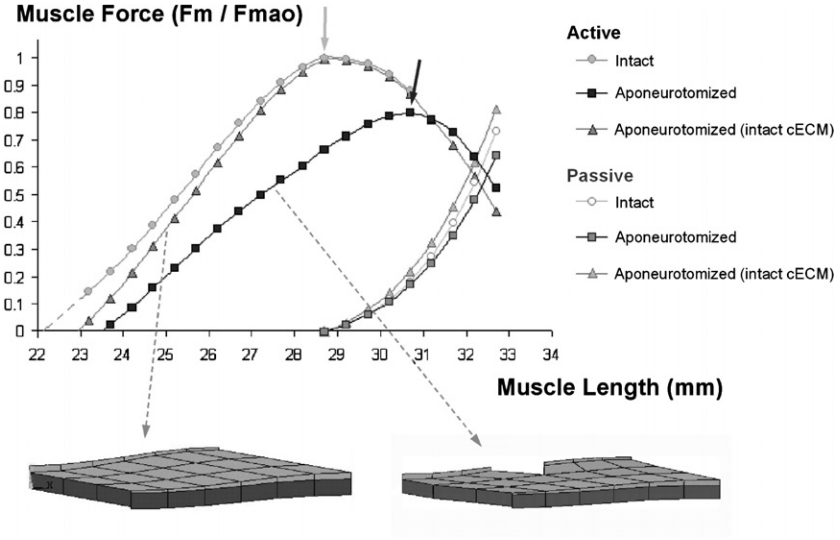

Fig. 6. Isometric muscle length–force characteristics of modeled isolated intact and aponeurotomized EDL muscles of the rat. Compared are: active and passive isometric forces of intact muscle and aponeurotomized muscle, as well as aponeurotomized muscle with an, as yet, intact collagen reinforced extracellular matrix (cECM). All of data are normalized for optimal force of intact muscle. Typical deformed shapes of activated aponeurotomized muscle and aponeurotomized muscle with intact cECM are shown to illustrate the separation of the cut ends of the proximal aponeurosis by a gap, as well as the modeled rupturing of the intramuscular connective tissues. Unlike aponeurotomized muscle with intact cECM that lacks shift of the optimal length to a higher length and reduction in optimal force (indicated by a gray arrow), the aponeurotomized muscle showed a substantial shift of the optimal length and decrease in muscle optimal force (indicated by a black arrow).

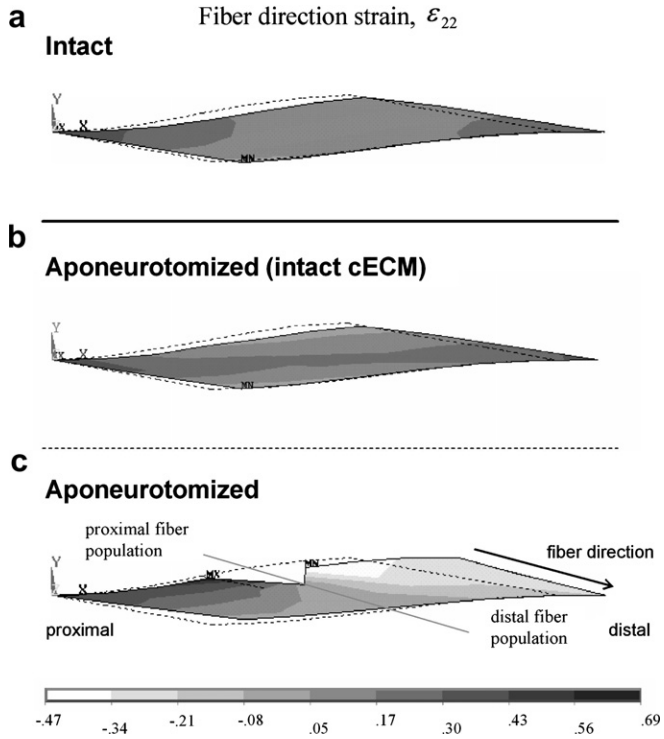

Fig. 7. Fiber direction strain distributions within modeled isolated intact and aponeurotomized rat muscles. The strain distributions within the fiber mesh of active (a) isolated intact muscle (b) isolated aponeurotomized muscle with intact cECM and (c) isolated aponeurotomized muscle are shown at high muscle length (31.2 mm). Note that the location of the intervention was at the middle of the proximal aponeurosis. The dotted line contour indicates passive muscle geometry at the initial length. Within each contour locations of maximal (MX) and minimal (MN) strain are marked. In part (c) a dotted line (near MX) indicates the original position of the disconnected nodes in the passive muscle (i.e., no gap). The proximal and distal populations of muscle fibers in the aponeurotomized muscle are indicated by a line separating them. The local fiber direction as well as the proximal and distal ends of the muscle are indicated. Note the similarity of strain distributions between the intact muscle and aponeurotomized muscle with intact cECM. This indicates that interfering with the myotendinous force transmission exclusively yields very minor effects. In contrast, if the tear develops within the muscle, also myofascial force transmission is interfered with and effects are major.

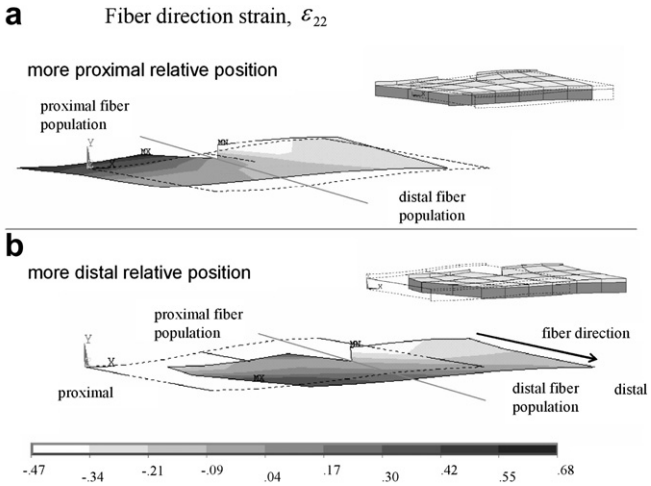

Fig. 8. Effects of muscle relative position on the fiber direction strain distributions within aponeurotomized EDL muscle with extramuscular connections. Strain distributions within the fiber mesh of active aponeurotomized muscle are shown for (a) more proximal (b) more distal muscle relative positions. The muscle modeled was kept at constant high length (30.7 mm), whereas its relative position was changed. The deformed shape of the muscle is shown in the upper right panels for both relative positions. The dotted line contour indicates passive muscle geometry at the initial length. Within each contour locations of maximal (MX) and minimal (MN) strain are marked. The proximal and distal populations of muscle fibers in the aponeurotomized muscle are indicated by a line separating them. The local fiber direction as well as the proximal and distal ends of the muscle are indicated in part (b). Note for both relative positions existence of highly shortened (maximally by 47%) and lengthened (up to 68%) sarcomeres within the muscle. A major model result is that due to extramuscular myofascial force transmission, altered muscle relative position reverses the acute effects of the intervention in the proximal population of muscle fibers.

that the acute effects of aponeurotomy are also not unique. Instead, they may be altered depending on the different mechanical conditions in which the target muscle is functioning.

To illustrate the effects of extramuscular myofascial force transmission and muscle relative position on the acute effects of aponeurotomy we will present some very recent finite element model results. The aponeurotomy EDL muscle model was extended to include extramuscular connections and it was studied at constant high length whereas, its relative position was altered. Fig. 8 shows fiber direction strain distributions within this model at more proximal and more distal relative positions. In general, the ''typical'' effects of aponeurotomy discussed in the earlier section are found for the more proximal position including much shorter sarcomeres in the distal population of muscle fibers and a highly pronounced serial distribution of sarcomere lengths (from highest length to lower, in the proximal to distal direction) in the proximal population. However, at the more distal relative position, a major contrast is shown: although the substantial sarcomere shortening still occurs in the distal population of muscle fibers, the serial distribution of sarcomere lengths in the proximal population is totally reversed in the proximal population (i.e. from lowest length to higher, in the proximal to distal direction). Moreover, the serial distribution in the distal population of muscle fibers becomes more pronounced. Therefore, the effects of muscle relative position alone on sarcomere length distributions as the key determinant of the desired effects of the intervention are substantial.

Another important model result is that the length of the gap between the two cut ends of the proximal aponeurosis shows a decrease of approximately 20% from the most proximal muscle relative position to the most distal muscle relative position studied (Fig. 9). Note that the cut ends of the aponeurosis was shown in animal experiments to be reconnected by newly developed connective tissue such that the recovered aponeurosis was longer compared to its length before the intervention (Jaspers et al., 2005). Therefore, the length of gap attained acutely (which seems com-

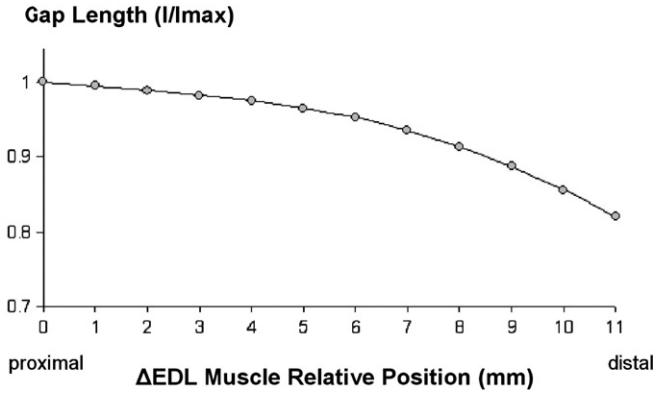

Fig. 9. Effects of muscle relative position on the normalized length of gap within the aponeuerosis. The length of the gap equals the distance between the two cut ends of the proximal aponeurosis. Note the ordinate does not indicate length, but relative position of the modeled muscle, kept at constant length. 0 mm represents the most proximal and 11 mm represents the most distal muscle relative position of the muscle belly. The maximal gap length was calculated for the most proximal muscle relative position. All gap length values were normalized for this value.

promised in certain conditions according to our model results) is indicated to be an important determinant of the potential lengthening effect provided by aponeurotomy. These results suggest strongly that extramuscular myofascial force transmission introduces major mechanical condition dependencies of the outcome of this intervention which are very important to consider in designing and application of the surgical treatment. Similar effects are indicated also for other surgical techniques to correct movement disorders.

3. Reflection on the effects of epimuscular myofascial force transmission on techniques for restoration of function

Surgical aponeurotomy is typically regarded as an intramuscular intervention possibly because the tendinous structures inside the muscle belly are treated and more importantly with reference to the classical point of view of considering muscles as independent functional units. However, our results on extramuscularly connected aponeurotomized muscle presented above suggest that aponeurotomy should not be conceived solely as an intramuscular intervention. In contrast, the effects of epimuscular myofascial force transmission should also be accounted for. Direct effects of such force transmission on the outcome of the intervention or indications parallel to such findings have been reported also for other surgical techniques.

One notable example is tenotomy. Unlike aponeurotomy, tenotomy totally removes one of the distal force transmission paths (the myotendinous one). The acute effects of tenotomy were studied in fully dissected rat EDL (Huijing et al., 1998), a muscle comprised of four heads, each of which having an individual distal tendon. The tendons of selected heads of this muscle were cut in the experiment and the change in isometric muscle force was assessed. Note that in that study, the role of epimuscular connections of EDL muscle was excluded. However, the different heads of EDL present an analogy to synergistic muscles with epimuscular connections. If myotendinous force transmission were the only mechanism of force transmission, the EDL proximal force should decrease proportionally to the physiological cross-sectional area of head being tenotomized. However, muscle optimum force was maintained at 84% of that of the intact muscle despite the fact that 55% of the total muscle mass did not have myotendinous connections. The authors showed a substantial decrease in muscle force only after myotomy (i.e., dissecting the intramuscular myofascial force transmission pathways) performed subsequently.

On the other hand, if epimuscular connections have no mechanical role, tenotomized muscle should shorten to slack length upon activation. During surgery, Kreulen et al. (2003) studied the acute biomechanical effects of tenotomy of flexor carpi ulnaris (FCU) muscle in patients with cerebral palsy (see also Smeulders and Kreulen, 2007). Subsequent to tenotomy, their specific aim was to test if dissecting epimuscular connections of FCU muscle did affect FCU muscle length: (i) in the neutral wrist position and (ii) on passively moving the wrist. In the neutral wrist position, tenotomy alone (no dissection of the epimuscular connections done) caused a minor shortening of the passive muscle and only a limited further shortening was found after the muscle was tetanized. In contrast, after partial dissection of the muscles' epimuscular connections its shortening increased substantially in both passive and active conditions. On passively moving the wrist, the authors showed that FCU excursions measured in intact condition and after tenotomy alone were very similar. However, dissection caused a dramatic decrease in the muscles' excursion. One surgical aim of tenotomy is to prevent the contribution of the target muscle to the joint moment. However, these findings suggest that tenotomy is ineffectual for such purpose, unless accompanied with major dissection of the muscle: intact epimuscular connections allow transmission of muscle force and therefore contribution of muscle to joint moment.

Tendon transfer surgery is performed to redress an "imbalance of antagonistic muscle forces". In this operation, the target muscle is mobilized by partially dissecting its surrounding connective tissues (e.g., Green, 1957) and subsequently its tendon, released from its insertion is transferred onto an antagonistic muscle insertion tendon. It should be noted that such surgical dissection is actually interfering with the epimuscular myofascial force transmission pathways. A recent study designed specifically to test the effects of surgical dissection of rat flexor carpi ulnaris (FCU) muscle showed that muscle length–force characteristics are altered substantially as a consequence of progressive dissection (Smeulders et al., 2002): (i) fasciotomy (partial incision of the antebrachial compartmental fascia) performed also in actual tendon transfer surgery to reach the target muscle caused a decrease in muscle active force

(by 20%) compared to the intact condition. (ii) Dissection of FCU half way up the muscle belly (to allow a sufficient mobilization of the muscle for tendon transfer) caused a further decrease in muscle active force which effect became even more pronounced after the muscle was dissected as much as possible. In addition, the dissections caused shape changes in muscle length–force characteristics (e.g., significant shifts were shown in muscle optimum length to higher muscle lengths). These results yield two important conclusions: (1) clinical dissection of the target muscle interferes with epimuscular myofascial force transmission mechanism and (2) the epimuscular connections that remain intact after clinical dissection are still highly capable of transmitting muscle force.

Studies of Delp's group on tendon transfer surgery performed to correct stiff-knee gait showed results that can be explained with the role of epimuscular myofascial force transmission. In such surgery, the distal tendon of rectus femoris (RF) muscle is detached from the patella and transferred to a knee flexor position in order to improve knee flexion. A graphics-based computer model and anatomical studies showed after recovery that the transferred RF muscle did have a knee flexion moment arm (Delp et al., 1994). However, it still generated knee extension moments when stimulated electrically (Riewald and Delp, 1997). Moreover, RF muscle was shown not to move with the direction of knee flexors (Asakawa et al., 2002) using cine phase-contrast MRI. These authors made a highly interesting suggestion however, without an explanation: RF muscle could continue to function as a knee extensor even after the intervention. Based on our experimental and modeling results we propose the following explanation for this effect: Although the muscles' distal tendon is attached to a knee flexor tendon, a substantial fraction of its force is transmitted via epimuscular myofascial pathways onto its synergistic quadriceps neighbors that still generate a knee extension moment.

Reconstruction of the anterior cruciate ligament (ACL) is commonly performed using the harvested distal tendons of semitendinosus and gracilis muscles. Therefore, the myotendinous force transmission path of these muscles is harmed directly. In such a state, a conceivable postoperative effect could be deficiency in knee flexion. However, after recovery from surgery, several studies reported only a small reduction in peak knee flexion moment, if any (e.g., Lipscomb et al., 1982; Yasuda et al., 1995; Maeda et al., 1996; Ohkoshi et al., 1998). On the other hand, Ohkoshi et al. (1998) showed that even though harvesting the semitendinosus tendon does not affect the value of the peak moment, the knee angle of exerting peak moment during knee flexion is reduced significantly. In the light of our findings, the post operatively unchanged knee flexion peak moment may be ascribable, at least in part, to epimuscular myofascial force transmission via neighboring hamstrings muscles. An explanation for the reduced knee flexion angle may be the altered sarcomere length distributions causing a narrowed length range of force exertion for the synergistic muscles (e.g., semimembranosus) due to altered stiffness of its epimuscular connections postoperatively. In addition using MRI, Hioki et al. (2003) showed that a knee flexion lag up to 10° is found after ACL reconstruction compared to the intact knee. Such lag may be due to the need of stretching epimuscular connections of the muscles involved. On the other hand, in a number of studies tendon-like structures were shown to regenerate during recovery from surgery (e.g., Cross et al., 1992; Eriksson et al., 1999). However, it has not been shown unequivocally that these structures are capable of functioning mechanically as a tendon. After recovery from intramuscular aponeurotomy, Jaspers et al. (2005) showed in rat gastrocnemius medialis muscle that, new connective tissue was formed between the cut ends of the aponeurosis. These authors reported structural differences between the aponeurotic tissue and this newly developed tissue and most importantly a more compliant nature mechanically. Such regenerated structures are conceivable to reconstruct the myotendinous force transmission mechanism. Therefore, both myofascial and myotendinous force transmission pathways are likely to be active after recovery. However, if the higher compliancy of tendon-like structures remains after adaptation to the new conditions imposed by surgery, the relative importance of myofascial force transmission is expected to increase.

We conclude that the integrated effects of mechanisms of intra- and epimuscular myofascial force transmission together with their possible effects on adaptation of the muscle play an important role in determining the outcome of surgery for restoration of function. Therefore, in such surgery the targeted muscle should not be considered independent of its epimuscular connections.

4. Mechanical properties of spastic muscle

The feature central to spastic muscle is hypertonia (e.g., Botte et al., 1988) and conceivably due to that, spastic muscle tissue is considered typically as *stiffened*. Such increased muscle tone originates in part from increased stretch reflex activity. However, since methods (e.g., injection of botulinum-toxin) to suppress the problem are not fully effective this is not likely to be the sole cause of muscle stiffening. A suggestion is that there is a passive component to stiffening of spastic muscle tissue (e.g., Mirbagheri et al., 2000). Despite the general use of classifying intramuscular connective tissues as aureolar or loose connective tissue, their role in force transmission has been shown experimentally (Street and Ramsey, 1965; Street, 1983; Trotter, 1990; Huijing et al., 1998; Huijing, 1999) and by using finite element modeling (Yucesoy et al., 2002, in press, 2006b). Such work shows that rather than being "loose", these structures feature sizable mechanical stiffness. Therefore, an immediate candidate for the passive component of hypertonia is the intramuscular connective tissues: after adapting to the spastic condition, these structures may become stiffer. Several studies suggest that spastic muscle may differ from

healthy muscle. However, the effects reported of such differences on the mechanical properties of muscle tissue are not consistent: (i) At the whole muscle level, there is experimental data presented that is interpreted to indicate that spastic muscle is stiffer than healthy muscle (e.g., Tardieu et al., 1982; Sinkjaer and Magnussen, 1994). (ii) At the muscle fiber level, Friden and Lieber (2003) showed that elastic modulus of isolated single muscle fiber segments (measured passively) taken form people suffering spasticity is twice that of muscle fiber segments taken from healthy people. In contrast, for passive bundles of muscle fibers, these authors showed that despite being hypertropic, cECM of spastic muscle tissue was less stiff (Lieber et al., 2003).

On the other hand, the mechanism of changes in the mechanical properties of collagenous connective tissues of spastic muscle is ascribable to changes in content and arrangement of collagen fibers. For example, Booth et al. (2001) reported accumulation of collagen and a thickened endomysium for spastic muscle tissue. However, others reported a normal collagen content (e.g., Ito et al., 1996; Marbini et al., 2002). It should be noted that even if the relative collagen content of the extracellular matrix is increased due to atrophy of muscle fibers the absolute collagen content of spastic muscle may be constant or even lower. Nevertheless, due to contrasting findings it is not clear if in spastic paresis, such atrophy is the most common occurrence (Ito et al., 1996; Marbini et al., 2002; Shortland et al., 2002; Malaiya et al., 2007).

An additional cause for increased passive resistance to stretch may be spasticity related shortened muscle fibers (i.e., adaptation to prolonged shortened state of muscle yielding a reduction in the number of sarcomeres within muscle fibers). However, using ultrasound imaging, Shortland et al. (2002) and Malaiya et al. (2007) showed no evidence for fascicle length change in GM muscle of spastic children compared to that in healthy children. Also Lieber and Friden (2002) showed using laser diffraction during FCU transfer surgery that lengths of spastic muscle fibers are normal. Nevertheless, these authors reported that the sarcomeres within spastic muscle fibers are highly stretched. In contrast, after clinical dissection Smeulders et al. (2004) presented evidence suggesting that overstretching of sarcomeres is not the general characteristics of spastic muscle: at high FCU lengths (i) the passive force measured during the operation was not exceptionally high, (ii) the active force was still very high indicating abundant overlap of myofillaments within sarcomeres. In our view, Lieber and Friden reached the conclusion of overstretched sarcomeres because they did not account for serial sarcomere length distributions that may occur due to myofascial force transmission. By necessity, they measured the lengths only very locally along the lengths of the fascicles, of albeit big groups, of sarcomeres within different muscle fibers constituting the studied fascicles. Then they generalized their findings to all sarcomeres within the muscle fibers (i.e., at serial locations other than those where measurements were taken, sarcomeres may actually be much shorter).

In light of these arguments, it is hard to say that the correlation between the passive component of hypertonia and the adaptation of intramuscular connective tissues of spastic muscle is evident. However, it is possible that the epimuscular connections of spastic muscle are stiffer (Smeulders, 2004) or the prestraining of these structures (Yucesoy et al., 2005) is increased. If so, this may be a cause for ''muscle stiffening'' by means of an even more emphasized role played by epimuscular myofascial force transmission compared to healthy muscle. For additional description, see also Huijing (2007). Because the effects of such force transmission has not been widely recognized by surgeons and because in remedial surgery there is commonly a physical need to mobilize the targeted muscle, no special care is taken when the epimuscular connections are dissected. However, as discussed in the previous section, such dissection itself seems to be extensive effectively co-determining the acute effects and therefore the final results of the intervention.

On the other hand, if stiffer compared to its healthy counterpart, the non-dissected part of epimuscular connections of the target muscle may still affect the outcome. After tendon transfers for example, when the target muscle moves with the antagonists, the change in its relative position with respect to its former synergists will be much greater, promoting the effects of epimuscular myofascial force transmission played by the non-dissected epimuscular connections. Note that the variability of clinical results may originate from variability of the mechanical properties of epimuscular connections of different patients (Smeulders et al., 2005; Smeulders and Kreulen, 2007) as well as the possible differences in the partial dissection performed by the surgeon. The amount of force transmitted via such non-dissected epimuscular connections should be assessed after tendon transfer. Nevertheless, after recovery, an entire set of epimuscular connections are expected to be reestablished providing a full potential path for epimuscular myofascial force transmission, as well as a potential cause for recurrence of the impeded function.

5. General conclusions

In summary, the effects of the integral system of intra- and epimuscular myofascial force transmission on muscular mechanics are substantial: (1) proximo-distal force differences, (2) shifts in muscle optimum length as well as muscle active slack length to different lengths, (3) alterations in the magnitude of muscle optimal force (4) changes in the shape of muscle length–force characteristics and (5) major serial and parallel distributions of sarcomere lengths are encountered as a function of different conditions in which the muscle functions. Such conditions are determined by muscle relative position with respect to neighboring muscular and non-muscular structures. The effects of epimuscular myofascial force transmission are (1) variable

due to differences in the joint angle dependent moment arms and the number of joints spanned among muscles (i.e., factors that cause relative position changes) as well as the complex mechanical properties of epimuscular connections (e.g., nonlinearity) however, (2) conceivable to substantiate at most muscle relative positions also in vivo due to prestraining increasing the stiffness of epimuscular connections. Therefore, due to epimuscular myofascial force transmission a muscle within the context of its intact connective tissue surroundings (the in vivo condition) cannot be considered as a fully independent functional unit: muscle length–force characteristics are variable depending on conditions of other entities and therefore, muscle length range of force exertion cannot be considered as a fixed property of the muscle.

This particular aspect of epimuscular myofascial force transmission has major implications on spastic muscle mechanically and on surgery for restoration of function. This is because the motivation for such surgery is typically to correct the limited joint range of motion. The results reviewed in this paper suggest strongly that (1) the mechanical mechanism of surgery acutely and the mechanism of adaptation in the long run are affected largely by epimuscular myofascial force transmission, (2) stiffened epimuscular connections and therefore the integral system of intra- and epimuscular myofascial force transmission may be involved in the etiology of the effects of spasticity on muscular mechanics. Therefore, we suggest that a key feature of improving the fundamental understanding on spastic muscle as well as the outcome of remedial surgery is to consider the target muscle as an integral system with its epimuscular connections.

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Can Yücesoy received his B.Sc. and M.Sc. degrees from the Mechanical Engineering Department of Middle East Technical University, in Ankara. Turkey. In 2003, he was granted a Ph.D. degree from the Biomechanical Engineering Department of the Faculty of Mechanical Engineering of Twente University at Enschede, the Netherlands. Subsequently he held a post-docteral fellowship at the Faculteit Bewegingswetenschappen at the Vrije Universiteit in Amsterdam, the Netherlands. Currently, he is working as a faculty member at the Biomedical Engineering Institute of Boğaziçi University in Istanbul, Turkey. His main research interests are biomechanics, experimental and finite element analysis of skeletal muscle mechanics with a specific focus on myofascial force transmission between muscles and between muscles and other tissues, and analysis of human movement.

Peter A. Huijing holds a degree in Physical Education from the Academy of Physical Education in Amsterdam the Netherlands, as well as a PhD. in Physiology from the University of Minnesota, Minneapolis, USA. He has a dual appointment at the Faculteit Bewegings- wetenschappen of the Vrije Universiteit, Amsterdam and the Department of Biomechanical Engineering at Twente University at Enschede, The Netherlands.

He has worked for more than 30 years at the interface of muscle physiology, anatomy and biomechanics, with primary research interests in fundamental aspects of form-function relation of muscles, force transmission from muscle as well as processes of adaptation ranging from the molecular level to whole muscle. Biomechanical modeling has always played an important role in addition to his experimental work. In recent years his work has been applied particularly within the fields of surgery of the locomotor system and rehabilitation. He is the 2007 laureate of the Muybridge Award of the International Society of Biomechanics for career achievements in biomechanics.