



# Epimuscular myofascial force transmission between antagonistic and synergistic muscles can explain movement limitation in spastic paresis

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## Abstract

Details and concepts of intramuscular, extramuscular and intermuscular myofascial force transmission are reviewed. Some new experimental data are added regarding myofascial force transmission between antagonistic muscles across the interosseal membrane of the lower hind limb of the rat. Combined with other result presented in this issue, it can be concluded that myofascial force transmission occurs between all muscles within a limb segment. This means that force generated within sarcomeres of an antagonistic muscle may be exerted at the tendon of target muscle or its synergists.

Some, in vivo, but initial indications for intersegmental myofascial force transmission are discussed. The concept of myofascial force transmission as an additional load on the muscle proved to be fruitful in the analysis of its muscular effects. In spastic paresis and for healthy muscles distal myofascial loads are often encountered, but cannot fully explain the movement limitations in spastic paresis. Therefore, the concept of simultaneous and opposing myofascial loads is analyzed and used to formulate a hypothesis for explaining the movement limitation: Myofascially transmitted antagonistic force is borne by the spastic muscle, but subsequently transmitted again to distal tendons of synergistic muscles.

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## 1. Introduction

Researchers as well as clinicians have grappled to explain the limitations in movement capability and the particular resulting joint positions (e.g. equinus of the ankle and a flexed position of the wrist) of patients suffering from spastic paresis.

This state of affairs may be irritating for scientists, but clinicians are affected more seriously: They are presented with patients suffering from the affliction and have to make decisions regarding treatment, despite the lack of detailed understanding of the mechanisms of the movement impairments.

To be able to do that adequately they have developed a system of practical knowledge and concepts in which they can consider interventions to improve the patients' condition. This knowledge has its definite value and contains elements that are not easily obtained by researchers, but usually lacks validation by detailed confrontation with experiential results. For a more detailed discussion see [Smeulders and Kreulen \(2007, Fig. 4, p. 650\)](#).

The other symposium articles presented in the present issue of this Journal, as well as the present work represent an attempt to try to limit the gap between the two types of disciplines as much as possible, and bridge it if possible.

The prime defect of spastic paresis is one of impeded neural control, thought to lead to exaggerated reflex activity in the affected muscle leading to enhanced resistance to

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lengthening, particularly if the lengthening of the muscle is executed rapidly. As a consequence the joint is held in a flexed position. This means that the target muscle and its synergistic muscles or muscle groups are kept at relatively low lengths for extended periods of time.

The question is raised time and again if we should consider a muscle that has been under spastic neural control for a long time a muscle with physiological muscle properties not principally different from healthy muscles, but fully adapted to the altered conditions imposed by the spastic neural control, or alternatively should be considered as having pathological characteristics itself.

Almost without exception in the clinical, as well as the scientific approach, of spastic paresis, muscles are considered as independent mechanical actuators, and as a consequence the movement limitation and characteristic joint posture is ascribed either to the acute effects of enhanced activity during stretching from low lengths or to limitations imposed by enhanced extramuscular connective tissue structures on the joint in the case of extended exposure to a particular joint position.

If one considers the acute effects after imposing spastic motor control of muscle (i.e. in a state of no muscle pathology), it is hard to understand why one active and shortened muscle should be able to maintain the specific and deviant joint position. A short muscle is likely to be active at the ascending limb of its length force curve and is likely to be able to exert only relatively small forces. As a naïve consideration, one could think that simply voluntarily increasing excitation of the antagonistic muscles, which are at higher lengths, should yield a net moment for ample movement away from the characteristic joint posture. Only severe limitations on excitation of the antagonistic muscle could prevent such action (It should, however, be noted that recent work reports indications of at least some limitation in antagonistic excitation in spastic paresis (Rose and McGill, 2005)). In addition, if the situation in spastic paresis would be very simple, the effect of botulin toxin, which inhibits neuromuscular signal transmission, should have acute effects on the movement limitations, as soon as it binds to its receptors at the postsynaptic sarcolemma. Clinically, it is clear that this is not the case.

Considering the recent work on epimuscular myofascial force transmission (Huijing, 1999a,b, 2002a, 2003; Huijing and Baan, 2003; Huijing and Jaspers, 2005; Yucesoy et al., 2003a,b), it is clear that muscles should not be considered as being mechanically independent. A prime purpose of this review is to analyze the principles of movement limitations of muscle under spastic control as a function of myofascial force transmission.

## 2. Intramuscular myofascial force transmission

In myotendinous transmission, force is transmitted to bone without leaving the muscle–tendon complex volume bounded by the epimysium (muscle fascia) and epitenon (tendon fascia). Myofascial force transmission is so named because there is ample evidence that force is not exclusively

transmitted to the origin or insertion of the muscle fibers (myotendinous force transmission), but in addition, force is transmitted onto the endomysium (Street, 1983; Street and Ramsey, 1965) and from there further onto the whole intramuscular connective tissue stroma, consisting of the intricate network of endomysial perimysial and epimysial tubes or tunnels (Jarvinen et al., 2002; Nishimura et al., 1994, 1996a,b, 1999, 1998, 1997, 1996c; Purslow and Trotter, 1994; Trotter and Purslow, 1992), within which the muscle fibers and fascicles and also the whole muscle are active (see also Fig. 1). A potentially substantial fraction of the muscle fiber force (but in physiological conditions usually less than 50%) is transmitted myofascially.

The connections between the trans-sarcolemmal molecules of the muscle fiber via the laminin molecules of the basal lamina to the collagen IV network of that same structure and from there on to endomysium are important potential pathways for intramuscular myofascial force transmission, as well as for mechano-transduction (i.e. mechanically activating activation processes within the muscle fiber). In physiological conditions these connections are loaded by shearing. The walls of endomysial tunnels themselves may form a pathway to the perimysial tunnels (Fig. 1, inset). In turn the shared walls of the perimysial tunnels of adjacent fascicles form connections to the epimysium. As a consequence the whole intramuscular stroma may act as one deformable element that is lengthened and shortened, with accompanying diameter decreases and increases because of the constancy of muscle volume, and that may be sheared.

### 2.1. New connections discovered recently

In addition to the connections between muscle fibers and the predominantly collagen type I structures of endomysial and perimysial tunnels, in beautiful recent morphological and histochemical work within the group of Jean-Paul Delage at Bordeaux (Passerieux, 2006; Passerieux et al., 2006) specialized collagen type III connections (see Figs. 2 and 3) have been identified connecting the muscle fibers, its basal lamina, as well as the endomysium to the perimysium (i.e. the fascicle fascia). Passerieux and colleagues applied elegant techniques of progressive digestion of bovine and rat muscular connective tissues by sodium hydroxide applied at different temperatures and concentrations and for different durations, followed by freezing and freeze fracture or sectioning to expose the newly discovered connections. The connections consist of relatively thick collagen fiber bundles (each fiber of which is approximately 1  $\mu\text{m}$  in diameter) that branch and form multiple connections on the muscle fiber, basal lamina and endomysial surfaces, somewhat resembling the morphology of a neuromuscular junction. The junctional plates at the end of each cable cover an area of approximately 200  $\mu\text{m}$  in length along the muscle fiber, and are constituted by a collection of multiple attachment points (junctions, Fig. 3).

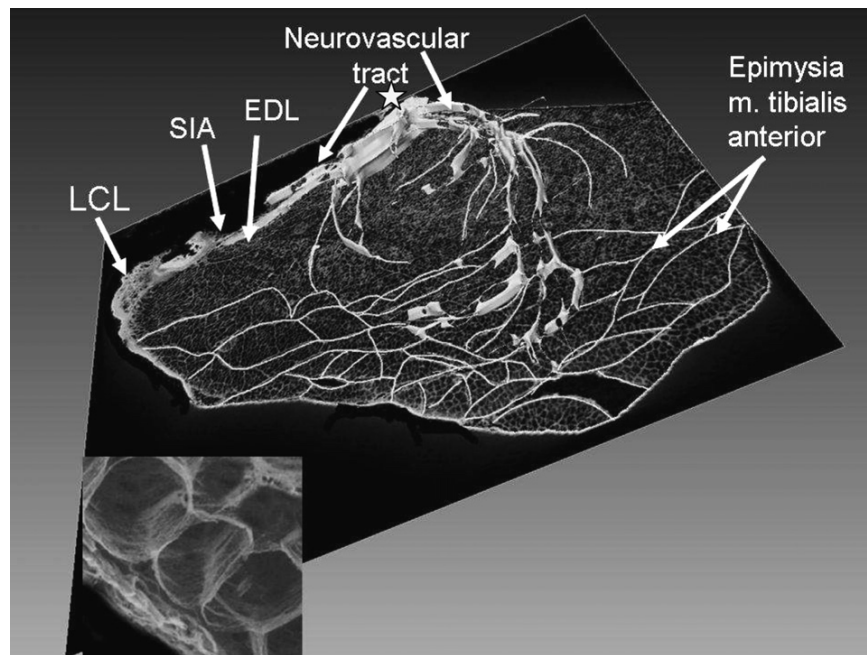


Fig. 1. A 3D-reconstruction of a small proximal part of the neuro-vascular tract of the rat anterior crural compartment. The anterior intermuscular septum (SIA) is suspended from the lateral collateral ligament (LCL) of the knee. The extramuscular neuro-vascular tract is shown at and just below the level of the fenestrated SIA (located at ☆). There the neurovascular tract passes between the peroneal compartment (towards the back left, not shown), the anterior crural compartment and the deep flexor compartment (at the back right, not shown). This tract, reinforcing blood vessels and nerves, branches into intramuscular extensions within the m. tibialis anterior (which encompasses most of the material shown) and m. extensor digitorum longus (EDL). Initially this is seen as thick intramuscular branches, but ultimately further branching extends it into the perimysia of both muscles and into the endomysia surrounding the muscle fibers. Enlargements of several endomysial tunnels are shown in the inset. The diameter of these tunnels varies between 50 and 100  $\mu\text{m}$ . Such sets of endomysial tunnels fill each fascicle. Note that at frontal side the perimysial structure is much more accentuated than at the dorsal side. The 3D reconstruction is placed on an image of an actual cross-section of the anterior crural compartment.

Passerieux and Delage consider such connections as extended parts of the perimysium and named them accordingly (perimysial cables and perimysial junctional plates). However, regarding the nomenclature and more particularly regarding the concept, we think it more fruitful to view these structures as collagen III reinforcements, within the whole fascicular stroma including the inner endomysia of the fascicle, possibly reinforcing blood vessels (arterioles and venules) and nerves that run, not in the fiber direction, but rather at sizeable acute angles with that direction. Anyone who has ever tried dissection or teasing of single muscle fibers from a fascicle, either in fresh material or in material digested with acids or bases, knows that periodically enhanced resistance is encountered by structures running in such directions. It is very well conceivable that these new structures are the substance causing that resistance. In the past, (Purslow and Duance, 1990) argued that the connections formed by the endomysial tunnel walls would be too compliant to be able to transmit force to the epimysium and beyond. Our findings (Huijing et al., 1998) that, after tenotomy, all active force exerted by the different heads of the fully dissected EDL could be transmitted to other parts of the muscle that still had normal myotendinous connections to the proximal tendon contradicted Purslow's reasoning. However, the discovery of these new connections may necessitate a new review of Purslow's reasoning, as it is conceivable that particularly

these cables provide the stiffness necessary for transmission of force from the central muscle fibers to the perimysium.

In any case, these collagen III fibers and specialized junctional plates (Fig. 3) shown to exist on the muscle fiber, its basal lamina and the endomysium deserve special further attention regarding two aspects: (1) to identify exactly where the collagen III cables commence within the perimysium and end within the basal lamina. And (2) their effects on muscle function and adaptation, as they seem to colocalize with nuclei and mitochondria and may thus play a special role in mechano-transduction (e.g. Ingber, 1997, 2003a,b; Huijing and Jaspers, 2005; Passerieux, 2006).

### 3. Epimuscular myofascial force transmission

For force transmission to occur between a muscle and its immediate surrounding tissues the force has to be transmitted beyond its epimysium, forming the connective tissue tunnel within which the whole muscle operates. This condition explains the name given to the phenomenon.

There are two potential paths to be distinguished for epimuscular force transmission:

- (1) *Intermuscular myofascial force transmission*: Direct transmission between the linked intramuscular stromata of two adjacent (synergistic) muscles.

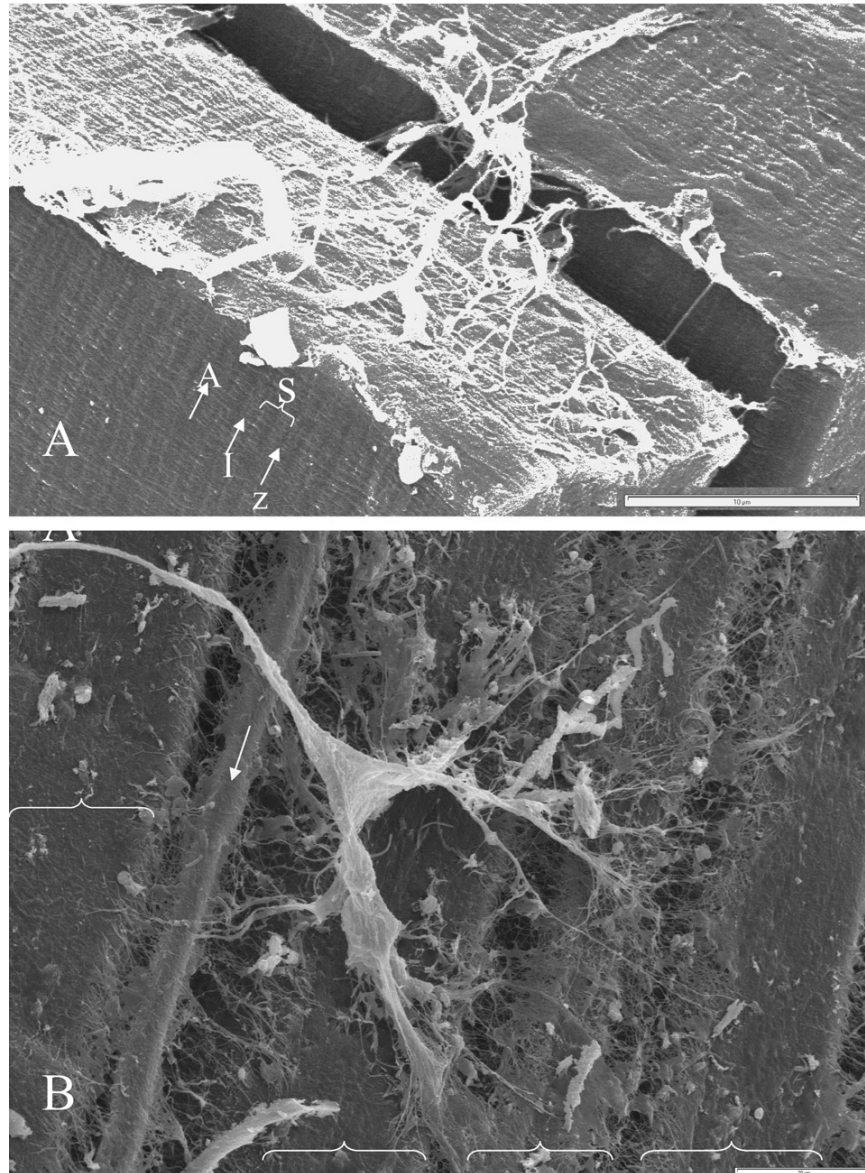


Fig. 2. Examples of newly discovered junctional plates on muscle fibers and their endomysium. A. Junctional plate on the surface of a muscle fiber that has been freeze fractured into two parts. Note that this plate is an assembly of many individual connections. At the side of the muscle fiber, exposing the intracellular content, the banding pattern of striation of the sarcomeres (S) is clearly visible. One can recognize A-bands (A, dark) I-band (I, light) and z-lines (z, thin dark lines). The bar indicates 10  $\mu\text{m}$ . B. Top view of a junctional plate and its strap-like cable. The junctional plate spans four adjacent fractured parts of muscle fibers (indicated by accolades) and is connected to them as well as to the endomysial material that is still present between these parts. It is also connected to as yet not fully unidentified structure (arrow), that could be either a blood vessel that runs in parallel and in close approximation to the left most muscle fiber or a thin extension of another non-spanning muscle fiber. Note the strap-like structure of the whole cable and the fiber-like structure of its final branches. The felt like material seen particularly on the left most muscle fiber part represents the basal lamina covering the muscle fiber. The bar indicates 20  $\mu\text{m}$ . Credit: Both scanning electron micrographs were taken by Emilie Passerieux and Jean-Paul Delage during the Ph.D. work of the former at the University of Bordeaux and were kindly supplied for this article with permission.

(2) *Extramuscular myofascial force transmission*: From a muscle onto extramuscular tissues, such as (a) the neurovascular tract (i.e. the collagen fiber reinforced protection of the blood and lymph vessels and nerves), and (b) the compartment delimiting connective tissues (intermuscular septa, interosseal membranes, periost, general fascia).

It should be noted that the neurovascular tract and compartment tissues as pathways for extramuscular transmission are connected to each other through muscular stromata, which in a way can be considered as the intramuscular continuation of the extramuscular neurovascular tract. The only structures that are not connected directly to this extramuscular system are the aponeuroses or tendon

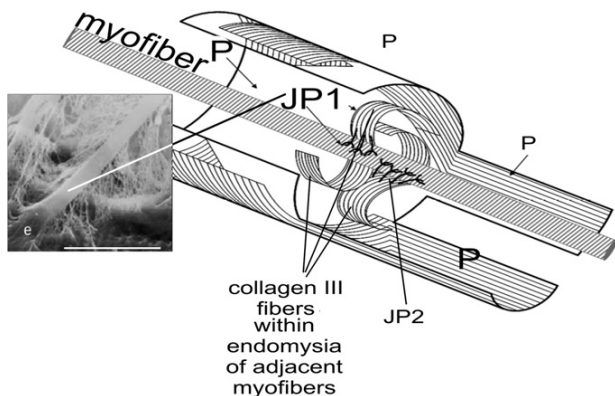


Fig. 3. Schematic representation of a perimysium tunnel with a central muscle fiber and their connections. The perimysium (P) has been opened at the front to show the centrally located muscle fiber and its connections. The other muscle fibers of his fascicle and their endomysial tunnels are not shown to expose the collagen III reinforcements of the fascicular stroma. JP1 and 2 are the junctional plates on the central muscle fiber. The inset shows the end of one of the branches of the cable and its connection (e) on the muscle fiber. The bar within this inset represents 10  $\mu\text{m}$ . In one location additional collagen III reinforcement of endomysial walls belonging to a muscle fiber adjacent to the central one is shown. This composite image was redrawn, with kind permission, and composed based on materials supplied by Jean-Paul Delage and Emilie Passerieux.

plate within the muscle–tendon complex, which is covered by epimysium or epitenon, but not attached to it. Probably, such connection would preclude the large range of motion characteristic for muscle.

The targets of extra-muscularly transmitted force are (I) joint capsules and ligaments, (II) other muscles within the same or other compartments and (III) either directly or ultimately bones.

It is important to note that extramuscular myofascial force transmission from a muscle does not preclude mechanical interaction of two muscles. The distinction (a–b) made above, merely serves to be more specific about the path of transmission. For synergistic muscles, i.e. active within the same compartment, it has been shown (Maas et al., 2006) that the intermuscular path contributes to epimuscular myofascial force transmission, but only to a much more limited extent than that of the extramuscular paths at least for the specific conditions studied (after full lateral fasciotomy). On the other hand the comparison between the effects of increasing lengths of a single muscle (i.e. EDL, with the other anterior crural muscles kept at constant fairly low lengths) and those of increasing length of the whole anterior crural muscle group (i.e. EDL, TA as well as EHL) may indicate a higher importance of intermuscular connections (Meijer et al., 2007). It is clear that settling this issue requires new experiments involving blunt dissection within a intact compartment.

It should be noted that any myofascial force transmission between antagonistic muscles or muscle groups by definition involves extramuscular myofascial connections since such muscles are separated by intermuscular septa or interosseal membranes.

## 4. Features of muscles involved in epimuscular force transmission

### 4.1. Proximo-distal force differences

Simple mechanical reasoning explains the occurrence of proximo-distal force differences: The sums of forces (action and reaction) equals zero. In a fully isolated muscle the proximally and distally directed forces are equal, which explains the fact that for almost two centuries muscular forces have been measured by using only one transducer.

However, it is clear that if epimuscular force transmission is active a net additional load is imposed on the muscle, either in proximal or distal direction. In the simplest of conditions, a proximally directed myofascial load is integrated into the force exerted at the distal tendon, but not at the proximal tendon, and vice versa (see for additional discussions on this point see elsewhere in this issue: (Huijing et al., 2007; Meijer et al., 2007; Yucesoy and Huijing, 2007)), and for examples of graphical illustration of these concepts also (Smeulders and Kreulen, 2007). It should also be noted that direct experimental proof of proximo-distal force differences even in animal experiments is often very difficult because only few muscles, usually poly-articular ones, have tendons accessible at their origin and insertion. Most mono-articular muscles have one tendon with a more direct connection of muscle fibers to bone at the other end. The measurement and modeling of forces exerted by muscle on bone should become an important task for future work. A drawback of such experimental work will be that the summed effects of many muscles are recorded.

### 4.2. Distributions of sarcomere lengths

If they did not exist before, distributions of sarcomere lengths are brought about or altered (if they were already present, e.g. because of differences in fiber length due to different curvatures, or different number of serial sarcomeres for muscle fibers of similar lengths) by inhomogeneous distribution of myofascial loads.

This means that myofascial force transmission precludes one of the, naïve but most common, assumptions made for muscles in the study of human or animal movement, namely the presumed homogeneity of sarcomere lengths!

#### 4.2.1. Serial distribution within muscle fibers

If a myofascial load is not exerted equally on all sarcomeres in series within a muscle fibers, differences in sarcomere lengths will ensue. Any sarcomere will shorten until it equilibrates with the load imposed on it. As a consequence, the sarcomeres exposed to the myofascial plus myotendinous loads will be longer than the sarcomeres exposed to only the similar myotendinous load. As the neurovascular tract enters the muscle belly at specific locations (hilus) relatively stiff connections are provided to the sarcomeres corresponding to the level of the hilus. Usually, such neurovascular connections will be loading the muscle myo-

fascially in proximal direction, unless very high changes in length are imposed at the proximal tendon of muscle. Distal lengthening of muscles may particularly enhance such proximal loading of muscle (see also elsewhere within this issue: (Huijing et al., 2007; Meijer et al., 2007; Rijkeljkhuizen et al., 2007)). The opposite can only be true if the muscle is lengthened substantially at its proximal end, or the whole muscle moved without lengthening in proximal direction, in which case the direction of the neurovascular tracts entering the muscle may reverse. It should be noted that in addition, distally directed myofascial loads by structures other than the neurovascular tract are often present also. This often the case in short muscles or muscle groups (see also elsewhere within this issue: (Huijing et al., 2007 and particularly; Meijer et al., 2007)).

The presence of serial distributions of sarcomere length will have profound functional effects. Even though a muscle fiber (as part of a motor unit) is a unit of excitation and activation, it can no longer be considered as functioning as a unit of force exertion. This serial distribution of sarcomere lengths (and of segments of its collagen reinforced extracellular matrix) is the most important reason for the occurrence of active and passive proximo-distal force differences of muscle fibers and muscle. A proximo-distal force difference is expected to lead to locally different signals for adaptation within muscle fibers (Huijing and Jaspers, 2005). For example, the addition of new sarcomeres preferably at one end of healthy muscle fibers within a muscle immobilized at high lengths may be a result of local conditions created by the imposed serial sarcomere length distribution within muscle fibers of the immobilized muscle. One important function of autocrine products secreted by muscle fibers (e.g. muscle Insulin-like Growth factor IGFm, Goldspink et al., 1995, 1996) to affect muscular sarcolemmal receptors may be perceived as putting a limit to too narrow a localization of adaptive signals and their consequences.

#### 4.2.2. Parallel distribution across muscle fibers

In many cases a muscle has not just one hilus but several. The more distally located ones along the muscle are often thinner and less stiff than the proximal ones. As a consequence muscle fibers located with in distal parts of the muscle are loaded less by net (proximally directed) myofascial loads than fibers belonging to the proximal fiber population of a muscle. This variation in serial distribution of sarcomere length between muscle fibers will give rise to distributions of fiber or fiber-segment mean sarcomere lengths. For more detailed analysis of such features see (Yucesoy and Huijing, 2007).

In addition, under special circumstances, myofascial loads may be exerted locally by compartment fascial elements on segments of the population of fibers of a muscle. A clear example is the distally directed load seen to be exerted on short muscle (TA + EHL) in the experiments with single EDL lengthening (Meijer et al., 2007).

#### 4.3. Relative position dependence of force exerted at origin and insertion

If a muscle, while kept at constant length, is changed in position with respect to its surrounding muscular and non-muscular tissues forces exerted at origin and insertion will vary (Huijing and Baan, 2003; Maas et al., 2004, 2003; Yucesoy et al., 2006). This occurs because the epimuscular myofascial connections, both intermuscular and extramuscular, will vary in length, stiffness and possibly direction. This means that relative position is an important factor co-determining force exerted at origin and insertion.

If muscle length is changed, two variables are affected simultaneously: the lengths (of muscle, fibers and sarcomeres) and the relative position of segments of the muscle. The condition of relative position is quite complex, since the part of the muscle closest to where length changes take place are exposed to big changes in relative position with respect to their surroundings, whereas toward the opposite end of the muscle changes in relative position decrease gradually and progressively to possibly even reach no change at all at the opposite end of the muscle, unless elastic effects of tendinous tissues are present.

There are many functional effects of changes in relative position. For example, changing the length of a muscle at the proximal end, will not yield the same conditions as changing the length of the muscle at the other end, even if the target length is identical (Huijing and Baan, 2003). This means that the factor muscle or muscle tendon complex length, which is of course simply defined as the distance between proximal and distal marker point identifying the muscle is no longer to be considered as a variable describing the conditions of force exertion adequately, without indicating the location of the length change. Therefore for functional reasons, it is necessary to introduce the concepts of proximal and distal lengthening of a muscle. Note that again that the phrase “nothing new under the sun” is applicable, as this concept is very close to the concepts of “punctum fixum” and “punctum mobile” that were prominent in earlier phases of history of myology.

This also means that the practice of adding proximal and distal muscle length changes to calculate total length change of a muscle tendon complex (Grieve et al., 1978; Visser et al., 1990) may not be advisable. It would most likely be more fruitful to keep analysis of proximal and distal length changes of muscle tendon complex separate to allow consideration of position effects at proximal and distal ends.

#### 4.4. The concept of several local myofascial loads on a muscle that may be similarly or oppositely directed

It should be stressed that a presence of a proximo-distal force difference is only an indication for a general net myofascial load. It is very well conceivable, and even highly

likely that conditions are imposed commonly on muscle that involve loading of muscle by more than one myofascial load. If such multiple loading occurs in the same direction it will complicate the analysis of the details of epimuscular myofascial force transmission since there likely to be multiple sources of reaction forces. If multiple loads are imposed on the muscle from opposite directions a much more complicated analysis is necessary, if at all feasible. . .

For example if a proximo-distal difference is small, there are two distinct possibilities: (a) there is very limited epimuscular myofascial force transmission, or (b) similarly big, but opposing effects are present. In a major paragraph below we will consider such complex conditions for spastic paresis. First we need to address a number of other issues.

Recent work presented in this issue (Meijer et al., 2007) indicates again that it is quite important to consider muscular relative position with respect to adjacent muscles and with respect to extramuscular structures, because myofascial effects may be quite different for different conditions of relative position. A comparison was made between effects of increasing length of EDL exclusively and after combined and equal lengthening of the whole anterior crural group. Note that the latter condition removes any effects of differential moment arms of synergistic muscles that exist in vivo movement (e.g. Lieber, 1997). For single EDL lengthening, major EDL proximo-distal force differences indicate major epimuscular myofascial force transmission between EDL and its surrounding tissues. For lengthening of the whole anterior crural muscle group the net EDL proximo-distal active force difference became very small, but extramuscular transmission to antagonistic muscles was enhanced. In addition, as yet unpublished observations at low lengths (Meijer and Huijing) features could be recognized that could not be explained by any other mechanism than effects of parallel distributions of sarcomere lengths due to epimuscular myofascial force transmission. For example, active force exerted lower stimulation frequencies proved to be higher than force exerted at much higher stimulation frequencies. Such results illustrate clearly that the myofascial load on a muscle is a complex net effect, and that its direction may potentially change on activation of muscle.

The potential multiple myofascial loading of muscle should be considered carefully in explaining myofascial effects both qualitatively and quantitatively. This means that one should not assume the most simple myofascial conditions to be present, unless unequivocal evidence can be obtained to that effect.

### 5. Myofascial force transmission and interaction also between antagonistic muscles

Muscles are not independent actuators. This is not only true for synergistic muscles, but also for antagonistic muscle. So far only preliminary data was reported indicat-

ing that myofascial force transmission also occurs between antagonistic muscle (Huijing, 2002a,b; Huijing and Jaspers, 2005). In fact the work presented elsewhere in this issue (Huijing et al., 2007; Meijer et al., 2007; Rijkelijhuizen et al., 2007) indicates that we have to deal with potential force transmission and thus myofascial mechanical interaction between not only adjacent antagonistic muscles within a limb segment, but also antagonistic muscles at opposite (anterior–posterior) sides of the lower limb.

#### 5.1. Some new experimental results on inter-antagonistic myofascial force transmission

The experiments, performed by Yucesoy, Baan and Huijing, were aimed at testing force transmission between the rat peroneal and deep flexor muscle groups. The former was lengthened to collect length-force data and latter was kept at a constant muscle–tendon complex length (yielding approximately 4 N of active force in the initial conditions, i.e. at low PER lengths). The knee joint was kept at approximately 100° and the ankle joint in extreme plantar flexion, in order to allow tendon connections to pass to the distal force transducers. All muscles within the peroneal and anterior crural compartments were either passive or excited fully. The proximal tendon of EDL was kept at a position 2 mm below its reference position (corresponding to a knee angle of 100°), EDL distal tendon was kept at a length 2 mm below distal EDL optimum length and the deep flexor muscles and TA + EHL were set a length that yielded 5 N and 3 N, respectively, initially.

The peroneus muscle group was lengthened by moving its distal force transducer. Results (presented in Fig. 4) indicate that myofascial transmission occurs also between passive muscles, as well as maximally active muscles, located within the anterior crural and deep flexor compartments: As the peroneus muscle group was brought to higher lengths the active and passive force exerted at their distal tendons by the deep flexor muscle group approximately halves. Almost all of decrease in active force can be ascribed to acute effects of myofascial force transmission, since the results for control contractions at a low reference length indicate only very minor effects of length history on the deep flexor muscles. For passive force such length history effects are bigger, but in any case a major part of the decrease is attributable to myofascial force transmission.

These results do not only indicate that extramuscular force transmission between antagonistic muscle groups across the interosseal membrane is feasible, but, in combination with the other results on force transmission presented within this issue, also that myofascial force transmission is active for all muscles and muscle groups within the lower limb, regardless if they are antagonistic or synergistic muscles.

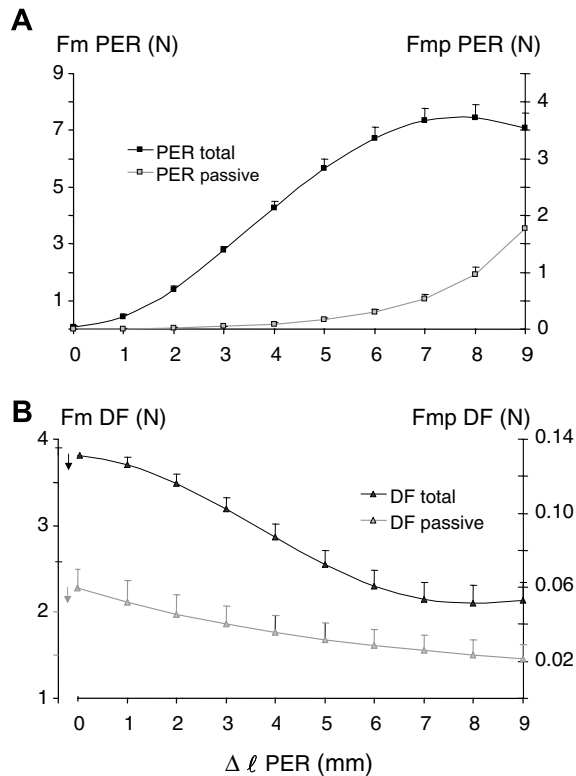


Fig. 4. Effects of increasing length of peroneal muscle group on force exerted by antagonistic deep flexor muscles of the lower hind limb of the rat. (A) Total and passive length distal force characteristics of the Peroneal muscle group (PER). Note that for the experimental conditions imposed, PER passive force is exerted even at low lengths. (B) Total and passive force exerted by the deep flexor muscle group (DF), while being kept at constant muscle-tendon complex length. Therefore, most of the decrease in force seen can be attributed to the effects of increasing PER length. Only a fraction of the force decrease from its initial value (i.e. 0.1 N and 0.2 N, the arrows shown at the left indicating these decreases in force of the control measurements ( $n = 6$ ), corresponding to 3.7% and 40.4% for DF total and passive force respectively) is explained maximally by history effects of activity of PER at high length. For a detailed description of such effects see (Huijing and Baan, 2001). All muscles and muscle groups studied were active maximally. For total forces use the left ordinate and for passive forces the right ordinate. Plots show means  $\pm$  SE ( $n = 7$ ). Note that qualitatively similar effects were found for anterior crural muscles kept at constant length (not shown). These results are a preliminary presentation of data of a, as yet unpublished, detailed study of the force transmission between antagonistic DF and Peroneal and anterior crural muscles by Yucesoy and Baan and Huijing.

### 5.2. Is intersegmental myofascial force transmission also feasible?

Considering such wide spread nature of myofascial force transmission between muscle groups, we should not even exclude the possibility that epimuscular myofascial transmission is not limited to one segment, but that intersegmental myofascial force transmission may exist. Particularly in quadrupeds, having a biceps femoris muscle belly in close approximation to the muscles of the lower limb, Fig. 5, this seems likely.

In addition, based on work in human cadavers, intersegmental mechanical interactions have also been suggested (Vleeming et al., 1995). It is clear that considerable experimental work needs to be performed on living muscles. This can involve animal experiments, but also in vivo imaging of human limbs and limbs of animals with quadruped locomotion.

In a special way the intimate relationship with muscles and the neurovascular tract can be demonstrated also in humans. Recently, my colleague at the Vrije Universiteit Henk Schutte, who is an expert in human anatomy, also in vivo anatomy, showed me the following after a discussion on the paths of the blood vessels and nerves at the knee: With extended knee, the tibial nerve innervating triceps surae muscles and more distal structures lies relatively deep within the popliteal cavity behind the knee joint (Fig. 6). If the knee is flexed and the hip is strongly anteflexed, dorsal flexion of the ankle will bring this nerve and its surrounding tissues to the surface, so that it clearly can be seen and touched. One can feel that the neurovascular tract containing this nerve is under substantial tension. It feels almost like a tendon and is often confused as such by the uninitiated. The potential explanation in terms of myofascial force transmission is as follows; distal lengthening of triceps surae muscle will change the position particularly of both heads of m. gastrocnemius with respect to the neurovascular tract will shear this structure within the compartment and pull it strongly in distal direction. Because of the flexed knee, this will cause a dorsally directed force on the tibial neurovascular tract that brings out the superficial part of it, containing the tibial nerve, beyond the unloaded position of surface of the skin. These changes make it extremely likely that more proximally in the leg (i.e. within the femoral segment) the tibial nerve and the sciatic nerve, from which the former is a branch, will increase their myofascial load on thigh muscles, which in effects constitutes intersegmental extramuscular myofascial transmission of force between gastrocnemius muscle and femoral compartment muscles.

It is conceivable that also some other features that are known (in a non-scientific manner) to both scientists and practitioners within the field of human movement can be explained by intersegmental myofascial force transmission. One example may be the unexpected effects of changing the sequence of joint movement: If one keeps the knees extended, many people have considerable difficulty or find it actually impossible to touch the floor by anteflexing their hip joint and reaching out with the arms. In contrast, if one flexes the knee joints first and touches the floor with the hands, extending the knee joints and simultaneously anteflexing the hip joint will allow most people to reach fully extending knees while still touching the floor with hands, with less difficulty than for the alternative order of joint movement.

A working hypothesis for such phenomena could be that the altered relative positions of muscle will redistribute

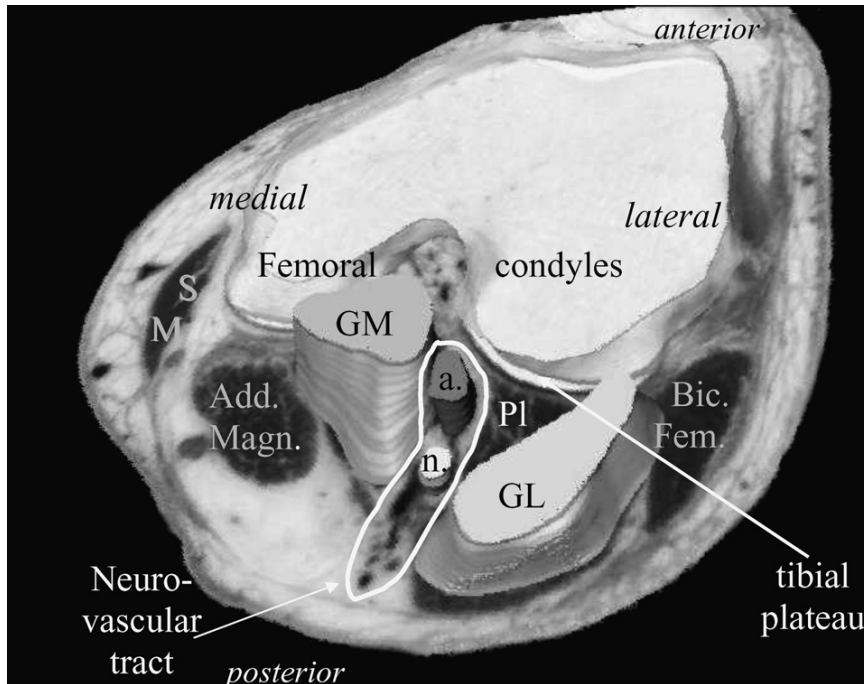


Fig. 5. A magnetic resonance image of the rat lower hind leg. The image shows a 3D reconstruction of elements of the medial view of the leg of selected density range. A number of structures that are likely to play a role in epimuscular myofascial force transmission is represented. In a part of the upper leg the knee (K), femur (F) is seen, as well as sciatic nerve (S) branching into the common peroneal nerve is seen on course to the peroneal compartment. Also the tibial artery on course to the triceps surae is seen. In the lower leg a longitudinal section of the lateral head of m. gastrocnemius (GL) as well as the tibia (T) and fibula (F), and the anterior intermuscular septum (SIA) separating the anterior crural (☆) and peroneal compartments (☆). A longitudinal section of the m. biceps femoris (BF) shows the location of the most distal part of the belly of this hamstring muscle. Note its intimate approximation to GL that is characteristic for quadrupeds. At this interface, intersegmental myofascial force transmission seems likely.

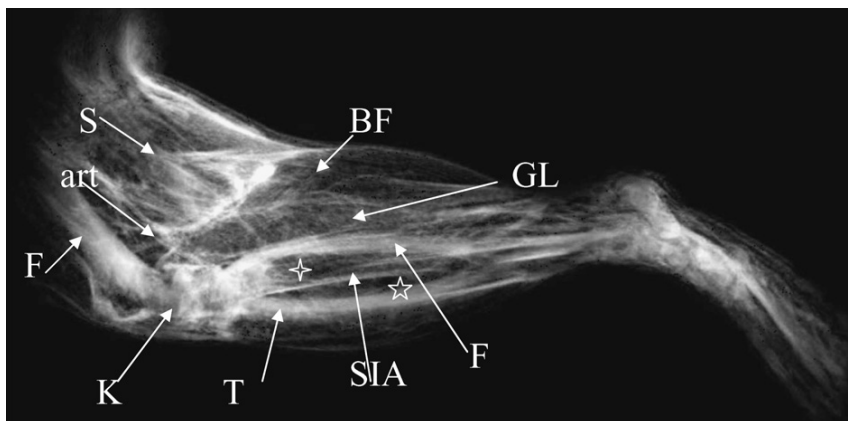


Fig. 6. A partial 3D reconstruction of the popliteal cavity at the back of the knee. The following structures are 3D reconstructed on the basis of data obtained from the cross-sections of the Visible Human Project: The femoral condyles, medial gastrocnemius muscle (GM), lateral gastrocnemius muscle (GL), the tibial nerve (n) and artery (a.). Neighboring structures can be seen within the bottom section shown: Biceps femoris muscle (Bic. Fem.) adductor magnus muscle (Add. Mag.) and semimembranosus muscle (SM). The neurovascular tract reinforcing nerves and blood vessels is indicated by a solid white line. By manipulating the hip, knee and ankle joint angles (see text) the neurovascular tract can be very much tensed and brought beyond the normal level of the skin.

myofascial loads differently allowing a greater range of motion in the hip joint. It is obvious that this hypothesis needs experimental confirmation, by which the difficulty will be to create comparable conditions of excitation in the two conditions for the muscles involved.

5.3. Muscle length, and other history effects and myofascial force transmission?

If one performs experiments, one is bound to make errors of measurement. Fortunately, most of such errors

are random in nature and the characteristic of central tendency of a mean or average will usually remove most of their effects if a sufficiently high number of results is averaged. However, it often also happens that some aspect of the activity during the experiment changes the properties of the muscle during the experiment in a systematic way. In order to quantify such changes it can be useful to perform contractions under (seemingly) constant conditions such as at constant length. If these changes are small it usually does not interfere seriously with the purpose of the experiment. For example the stepwise increasing changes (Fig. 7) during and after subsequent collections of length–force data will enhance the force of the muscle that is not lengthened and only lead to a small underestimation of the myofascially related force decrease of synergistic or antagonistic muscle kept at constant muscle–tendon complex length as the experimental muscle that moment is lengthened. It is wise to choose a low reference length for such control contractions, since many effects are enhanced at low lengths. For example, potentiation of submaximal force (Roszek and Huijing, 1994) by phosphorylation of the myosin light chain (Houston et al., 1985; Manning and Stull, 1982; Palmer and Moore, 1989; Sweeney and Stull, 1990) will have major effects on force at lower lengths.

The same has been shown for length history effects: Activity at high lengths, leads at least initially, to major decreases of active force and proximo-distal force differ-

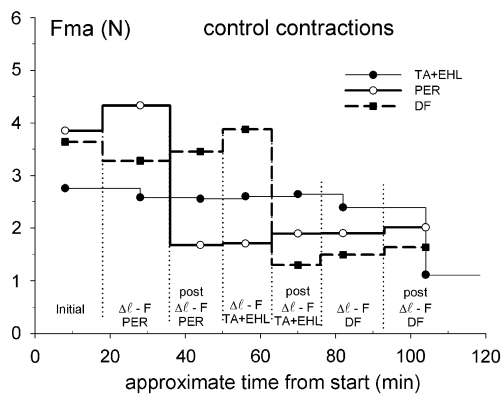


Fig. 7. Examples of history effects on active force exerted at constant length during an experiment. An experiment was performed collecting length–force data (not shown) of three groups of muscles for maximal excitation. Simultaneously, forces exerted by other muscle groups were measured. The sequence of length–force measurements was: (1) peroneal group ( $\Delta\ell - \text{FPER}$ ), (2) tibial muscles ( $\Delta\ell - \text{FTA} + \text{EHL}$ ) and (3) deep flexors ( $\Delta\ell - \text{FDF}$ ). Before and after each length force data set all muscles were brought to a reference position at relatively low lengths (see text). Some relatively small history effects were considered comparing forces during determination of and between two or three subsequent length–force data sets. Very major changes were always recorded for active force of a muscle after that same muscle itself had been active at high lengths. Therefore the major effect is a specific effect of previous activity at high lengths (i.e. over optimum length). This conclusion is in agreement with a previous systematic study for synergistic muscles (Huijing and Baan, 2001).

ences at low lengths, whereas forces and those differences at optimum length are hardly changed (Huijing and Baan, 2001). However, the persistence of proximo-distal force differences indicates that under the new conditions of force exertion myofascial force transmission is still operational, but at different levels of forces. The present work supports the concept of very substantial effects of previous activity at high length. Fig. 7 shows systematic and major decreases in force for muscle at low reference length minutes after it has been active at high lengths. The mechanism(s) cresting such effects are not fully understood and require new and specifically directed research. Intuitively one would think that factors related to creep and stress relaxation within myofascial connections could be quite important. The effects specifically at lower lengths could be related to the steeper slope of the length–force curve at the ascending limb compared to the slopes near optimum length.

It is our experimental experience that such effects can be minimized by alternating contractions at high and low lengths until no further decreases of active force at low lengths are seen and more or less steady state conditions are expected. Presumably this is also what happens during warming up activities or so called stretching exercise where muscle is made active isometrically at high lengths.

In some of our experiments aimed showing force decreases in antagonistic or synergistic muscles kept at constant length such conditions were imposed (e.g. Meijer et al., 2007), but in other (usually older) experiments this was not done (e.g. Huijing et al., 2007), as well as the new data presented in this paragraph. In any case since most of these experiments are fairly straightforward involving only one length–force data set the effect is expected to occur after the data set is collected. This means that the corrections discussed in (Rijkelijkhuisen et al., 2007) are probably not necessary. In any case, the conclusions drawn about such force decreases are not affected much by these phenomena.

If one would study effects of major interventions such as effects of fasciotomy or blunt dissection of inter- or extramuscular connections one needs to be careful not to consider any such history effects as effects of the intervention!

## 6. Myofascial force transmission experiments and in vivo conditions

Of course several experimental conditions described in the articles of the present issue and previous work of our group deviate strongly from healthy in vivo conditions of muscle lengths, degree of activation, degree of recruitment of motor units, etc. Other factors related to intermuscular relative positions are to be considered as well. Examples are: imposing length changes on a single muscle (e.g. Huijing et al., 2007; Meijer et al., 2007) or the lengthening of the peroneus group in combination with a fixed and rather distal relative position of the EDL tendon (Huijing et al., 2007). Such combinations of relative positions of antagonistic muscle do not occur in vivo. As a consequence, some

people consider such experiments as having no clear use. Despite the limitations of generalization that such experiments most definitely involve, we can still draw important conclusions from them that help our understanding of the mechanisms, pathways and materials of myofascial force transmission, particularly if the confrontation of issues is chosen in an appropriate way. Such enhanced understanding may then also be applied to the analysis of in vivo conditions. Examples in the specific cases discussed are (a) the confrontation between effects of single muscle lengthening and muscle group lengthening, leading increased understanding and identification of the structures that are imposing a distal load on shortened muscle and (b) for studying the effects of peroneal length under unusual conditions the fact is noteworthy, that qualitatively and also to a large extent quantitatively the results for peroneal group and tibial muscle complex lengthening were similar (Huijing et al., 2007). We interpret this similarity of results for length effects of peroneal and anterior tibial muscle as an indication that the myofascial connections between antagonistic muscles are set up in a standard way. Distal lengthening of a muscle group decreases the active and passive force exerted at distal tendons of other muscle groups and in roughly similar amounts (disregarding some differences in details) At high tibial muscle complex lengths relative positions that occur in vivo are approximated more. This is the case despite the fact that real life joint movement would have led to a progressively even more shortened peroneal group coinciding with high anterior tibial muscle lengths and corresponding relative positions.

### 7. Adaptive effects of keeping healthy muscles short

The effects of immobilization on muscle are usually studied by doing in vivo animal experiments. One exception is the longitudinal study of adaptive effects on single muscle fibers (Jaspers et al., 2002, 2006, 2004a,b). In contrast to the in vivo character of the usual imposition of immobilization conditions, evaluation of functional effects (e.g. length–force and force–velocity characteristics) in animal experimentation has almost exclusively been performed in muscle fully dissected from its surroundings with the exception of its most proximal aspect of the neurovascular tract to allow circulation and innervation. In view of the above a clear need exists to evaluate also myofascial aspects of immobilization.

For purely morphological aspects such as muscle volume and physiological cross-section the degree of dissection is not important. From immobilization experiments in vivo, it is clear that if muscles (e.g. rat or cat m. soleus and m. gastrocnemius) are kept at low lengths, major atrophy (defined in terms of physiological cross-section) will ensue (Appell, 1986; Booth, 1977; Heslinga and Huijing, 1992b; Kawakami et al., 2001; Mayer et al., 1976; Stillwell et al., 1967; Tardieu et al., 1971). Immobilization per se does not cause the effect since, at worst, only minor atrophy occurred in muscle immobilized a high length condi-

tions and sometimes even some hypertrophy is seen (Tardieu et al., 1974; Williams and Goldspink, 1978). This is related to the muscle length effects known for protein synthesis.

Differential secondary effects of atrophy are reported depending on the degree of pennation (Heslinga and Huijing, 1992a, 1993): In muscle of a low degree of pennation (i.e. rat m. soleus), the atrophy affects predominantly the thickness, but not the length of the muscle belly. In contrast, for highly pennate muscle (i.e. medial gastrocnemius) major atrophy will lead to substantial muscle shortening because of muscular geometry: Muscle fiber cross-section contributes (at an acute angle) significantly to muscle length. In immobilized very pennate muscle, this atrophy related shortening of muscle will cause a progressive lengthening of the muscle fibers (for a schematic of this concept see Fig. 1 in Huijing and Jaspers, 2005) since the muscle tendon complex length needs to be maintained and a major lengthening of the tendon is not likely. Such atrophy related lengthening of muscle fibers is also apparent from a shift of pennate muscle optimum muscle length to the immobilized length, thereby removing the stimulus for adaptation of serial sarcomere number. Accordingly no adaptation of serial sarcomere number was found in very pennate muscle (Heslinga et al., 1995). In contrast, for soleus muscle from the same animals, the number of serial sarcomeres did adapt and was substantially decreased (Heslinga et al., 1995).

For the context of myofascial force transmission, it is important to realize that the in vivo immobilization conditions at low length for the target muscle represent conditions of muscle group shortening of agonistic muscles, but muscle group lengthening of antagonistic muscles. In contrast to experimental conditions of the animal experiment of Meijer et al. (2007), some changes in relative position between synergistic muscles will be present due to differences in moment arms. It should also be noticed that the evaluation of length–force characteristics of muscles immobilized in vivo, has only been done in muscle fully dissected except for its innervation and blood supply. This means that new experiments should be performed evaluating immobilized muscle characteristics within their full myofascial context. Since it is clear that muscular connective tissues also adapt to immobilization (e.g. Williams and Goldspink, 1984).

### 8. How do these myofascial and adaptive features affect spastic muscle and its synergistic and antagonistic muscles in spastic paresis?

#### 8.1. Atrophy and its effects of on spastic pennate muscle and its fiber lengths

Spasticity involves reflex induced acute muscle shortening of the affected muscle (e.g. Botte et al., 1988; O'Dwyer et al., 1994). The reflexes are thought to be exaggerated

because of pathologically diminished inhibition from higher central neural system levels on motor units within the anterior horn of the spinal cord (Gracies, 2005a,b; Young and Wiegner, 1987).

The shortened condition is subsequently sustained over long periods of time, unless stretched very slowly. The muscle may either be passive at quite low lengths, or if stretched rapidly it will resist lengthening actively and, therefore, remain at low length. Due to these low length conditions, atrophy may be unavoidable. It is important to realize that the strains imposed by atrophy related shortening of the muscle belly and the corresponding enhanced myofascial loading may aggravate the conditions of spastic control of the muscle. Usually muscle spindles are considered as length and stretch receptors (e.g. Schafer, 1995). However, within the concept of myofascial force transmission they could potentially be, not only length and velocity sensors, but also as “misalignment detectors”. As the spindles are located within perimysia, they are very likely to be quite sensitive to shearing of the perimysium caused by unequal lengths of adjacent fascicles (disturbance of fascicle alignment). As additional shortening of the muscle belly is expected to increase myofascial loading and this phenomenon is not likely to be distributed homogeneously over different populations of fascicles within the muscle enhanced firing of muscle spindles and monosynaptic reflex activity is expected.

We need to consider the effects of the progression of such atrophy in spastic muscle as it will differ from immobilized muscle. In a muscle spanning a joint that is not fully immobilized (as in spastic paresis), in very pennate muscle the atrophy-related shortening of the muscle belly will occur without necessarily involving changes in muscle fiber length: If the atrophy related muscle belly shortening is not compensated by structural length changes of the tendon a change in joint angle will be present. This means that the joint range of motion and of active force exertion will be changed drastically.

Even though clinicians tend to treat spastic muscles as overly short muscles (e.g. Tardieu et al., 1982), scientific evidence on actual shortness of muscle in spastic paresis is rare (see also discussion in this issue Smeulders and Krenulen, 2007). Modern imaging techniques allow visualization of fascicles for in vivo conditions.

Important work, involving application of ultrasound imaging to medial gastrocnemius muscle in patients suffering from spastic paresis, has been performed by Shortland and co-workers (Malaiya et al., 2007). Their experimental results are of high quality, because they focus specifically on the strong points of imaging analysis, namely morphometrics. In contrast, in many ultrasound imaging publications on in vivo activity, it is common to make unrealistic assumptions about ascribing net forces and net moments exerted at joints, exclusively to particular muscular structures seen and analyzed (e.g. Kubo et al., 2000, 1999; Maganaris, 2001; e.g. Maganaris et al., 1998; Maganaris and Paul, 1999).

Elsewhere within Malaiya et al. (2007), report major atrophy in hemiplegic patients when compared to healthy cohorts. Similar results were reported earlier for diplegic patients (Shortland et al., 2002). Because of a lack of information regarding comparable length reference conditions for the muscle studied in patients and regularly developing children, the authors have been wise to express the atrophy in terms of muscle volume, rather than physiological cross-section. However, for considering functional effects on muscle this is unfortunate, since it is clear that serial effects of atrophy (i.e. decreases in serial sarcomere number) or parallel effects (physiological cross-section related effects) will have quite different functional consequences. What is really needed badly, but very difficult to perform, is a quantitative analysis regarding the presumed change in physiological cross-section of the spastic muscle and changes in muscle belly length. Nevertheless, the relation shown for individual data between muscle volume decrease and the seriousness of the affliction (see this issue Malaiya et al., 2007) is an important supportive point of the idea that atrophy related effects are a major factor in the creation of movement limitations.

For a single spastic muscle, the atrophy related shortening emphasizes the condition of shortening of a single muscle. Compared to the neighboring muscles, the spastic muscle becomes even shorter with presumably serious consequences for myofascial loading (see below).

Despite our above use of the word atrophy (to make an easy link possible to the discussed atrophy effects of immobilized muscle) it may be better to speak of a lack of trophic (growth) in these child patients (but the effects are similar), even though the word atrophy literally means that.

A major other finding of Malaiya et al. (2007) that, with patients and subjects in similar external conditions (lying prone, with the foot only affected, by gravity), the ankle joint attains a more plantar flexed position in patients. The authors assume that in both patients and healthy children this indicates that the whole triceps surae is at its passive slack (i.e. approaching zero force). If this should be true, this would constitute an important length reference for comparison of the two groups. However, there is not any evidence supporting this assumption and in fact the considerations of myofascial force transmission described above make that rather unlikely.

Note, however, that this difference in joint position is attained at similar normalized fascicle lengths for the two groups is a very important observation. From this fact we can deduce that at equal ankle joint angles in more dorsal flexed positions the patients will show longer fascicles (normalized length) than the healthy subjects. Similar effects can actually be detected in data reported earlier for diplegic patients for which group data is available at several joint angles (Shortland et al., 2002). The authors are somewhat reluctant to draw attention to this fact explicitly, but in our opinion such a confrontation is necessary to enhance our understanding. It leads to posing the

question regarding the explanation and mechanisms of this phenomenon (see below).

### 8.2. *The muscle under spastic control is often a single short muscle*

It is important to realize that the shortened conditions, at least initially, apply exclusively to the affected muscle, unless spasticity is affecting a whole muscle group. In this sense the situation is comparable to that of local cramp, with the exception that spastic muscle is not active constantly, but only when its control system is provoked by at least moderately rapid lengthening. This also means that the negative adaptive effects of keeping a muscle short should be expected (i.e. atrophy, decrease in serial sarcomere number), unless additional specific mechanical or biochemical signals of adaptation are present to counteract such effects.

### 8.3. *An enhanced distal myofascial load on spastic muscle*

From the experimental results for healthy animals (Huijing et al., 2007; Meijer et al., 2007; Rijkelijhuizen et al., 2007) it is clear that a distal myofascial load is high on a shortened muscle (i.e. TA + EHL muscle). Also the results for human patients indicate such loads: The tenotomized FCU is lengthened as the wrist is moved from flexion to extension, despite the fact that FCU no longer crosses the wrist (Kreulen et al., 2003; Smeulders and Kreulen, 2007). It is clear that extending the wrist causes a distally directed force to be exerted onto FCU. A most likely pathway is the neurovascular tract that can be seen prior to clinical dissection, (see Fig. 3A of Smeulders and Kreulen, 2007). Alternative pathways could be intermuscular myofascial connections with synergistic wrist muscles. Regardless of the exact pathway, if one considers the FCU in a free body diagram a distally directed load is exerted on FCU muscle fibers. Even with the FCU tendon still attached to its insertion, such distal loading of the muscle will be present. Presumably such distal load will be exerted on rather distally located, sarcomeres within FCU muscle fibers which will tend to transmit a, potentially large, fraction of the force from the muscle before it can reach the FCU tendon.

It should be realized also that the clinical dissection of FCU (performed to allow later transfer of its distal tendon to an extensor insertion site) will by itself move most, but apparently not all of this distal loading. However, in analyzing the patient FCU length–force data obtained after clinical dissection it is difficult to imagine why the movement limitation did previously occur (Smeulders et al., 2004). Of course we have no access to FCU length force data of healthy controls, but the post-dissection patient FCU length–force curves did not appear abnormal (i.e. no extreme high lengths were attained within the joint range available to the patients and no unusually high passive forces were encountered at lower lengths, see also

Smeulders and Kreulen (2007)). This reinforces the concept that the mechanism of movement limitation may have been removed or decreased by clinical dissection and therefore must be related to features of myofascial force transmission. The fact that, even after clinical dissection, there still remains plenty of mechanical interaction between muscles of the forearm is shown by the very different FCU length–force curves measured at different wrist joint angles Fig. 4. of Smeulders and Kreulen (2007). The very complex different results for two groups of patients must be an indication of differences in properties of pathways of myofascial force transmission, but remain to be explained.

### 8.4. *Multiple myofascial loads on spastic muscle, with complex effects on fascicle and fiber length*

Enhanced distal loads on spastic muscle cannot explain the movement limitation encountered, as the distal myofascial load will decrease the, already low force exerted by an atrophied muscle exerted at its distal tendon. From the fact that the joint is forcefully kept in a particular position (e.g. palmar flexion for FCU and plantar flexion for the triceps surae muscle), we can derive that sufficient moments are exerted by muscles in some way. Epimuscular myofascial force transmission will allow for example FCU force to be exerted at the distal tendons of synergistic muscle, but this is not likely to be sufficient. In addition, those synergistic muscles are also at low lengths due to the imposed joint angle, albeit less than their spastic neighbor, so forces exerted by them are expected to be low. So what could be the origin of high forces within the spastic paretic limb? From the experimental results for healthy animals presented elsewhere in this issue (Huijing et al., 2007; Meijer et al., 2007; Rijkelijhuizen et al., 2007) and also in the present article, it is clear that forces generated within sarcomeres of antagonistic muscles by extramuscular myofascial force transmission can be exerted at the distal tendons of a target muscle. This occurs if the antagonistic muscle at high lengths itself is loaded in distal direction by extramuscular pathways which in that way exert a proximally directed extramuscular myofascial load on the target muscle.

In spastic paresis, we propose that such mechanisms may play an important role in forcing the joints into the characteristic flexed positions. In fact, we see for the spastic muscles similar but enhanced ways of interaction of multiple oppositely directed myofascial loads also seen in some of our experiments on healthy animal muscles.

The basic elements of the processes are illustrated in Fig. 8. High proximally directed myofascial loads are imposed on groups of sarcomeres that are located more proximally within the muscle fibers of the spastic muscle. As a consequence these sarcomeres will not shorten as much as when exposed exclusively to low proximally directed myotendinous loads. This means that they will exert higher forces. Somewhere more distally on the same muscle

fibers, the high distally directed epimuscular myofascial load is exerted transmitting force from the muscle onto either the synergistic muscles or passive tissues that cross the joint, thereby exerting major moment at the joint. The opposing moments being low due to the fact that the antagonistic muscles (extensors) have been loaded myofascially in distal direction more proximally. As discussed above the spastic muscle itself will not exert high forces at the joint via its distal tendon. The above described events will lead to very complex serial distributions of sarcomere length within fibers of the spastic muscle: At the most proximal end the sarcomeres will be short, more distally a group

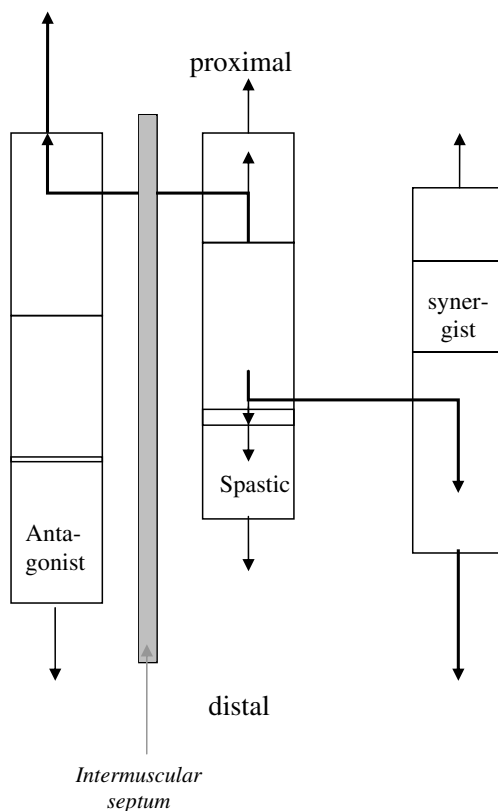


Fig. 8. Schematic of the hypothesis of force transmission between spastic muscle and its antagonistic and synergistic muscles, explaining the movement limitation. The muscle fibers of each of these muscles are represented as rectangles and divided into a proximal, intermediate and distal part. The size of these parts is representative of the length of the sarcomeres within them. The arrows indicate the forces exerted on the muscle fibers or their parts. The thick arrow (at the left) indicates the extramuscular myofascial transmission of force between the antagonistic muscle and the spastic agonist muscle, and the thick arrow at the right the epimuscular myofascial force transmission between the spastic and the synergistic muscles. Note the complex distributions of lengths of sarcomeres arranged in series within muscle fibers (no parallel distributions for sarcomere lengths are shown for the muscles). Also note that according to the hypothesis most of the force causing the movement limitation originates from proximal sarcomeres within the antagonistic muscle is transmitted by sarcomeres and/or their associated collagen fiber reinforced extracellular matrix of intermediate location within the spastic and synergistic muscles, and ultimately exerted at the distal tendons of the synergistic muscles.

of them will be much longer and at the distal end of the same muscle fibers the sarcomeres will be short again. The net effect of this serial distributions of sarcomere length can very well be that the fiber lengths at given joint positions will be increased in the spastic muscle of patients compared to the same muscles of healthy subjects which are expected to show less serial distribution of sarcomeres within its muscle fibers. This would fit well with the observations on data by Malaiya et al. (2007), discussed above.

## 9. Some implications for surgery

In addition to implications of myofascial force transmission discussed elsewhere in this issue (Yucesoy and Huijing, 2007) the following can be added.

If the hypothesis presented above can be confirmed experimentally, a number of consequences for regarding the performance of the surgical operation may be considered.

Since myofascial force transmission has not been a consideration in surgery for long, we need to consider the possibility that, at least in some patients, not target muscle of the surgery itself, but a synergistic muscle could be the cause of the movement limitation problem. In the case of wrist surgery, performing clinical dissection of FCU will still help. As one of the attendants of the symposium expressed it, “Despite FCU not being a part of the problem; it could well be part of the solution”. Any interference with epimuscular myofascial force transmission between synergistic flexor muscles would lower flexion moments exerted at the wrist.

The question even needs to be addressed if tendon transfer to an extensor insertion site is necessary? It is conceivable that just clinical dissection could be sufficient, if the restoration of epimuscular myofascial connections between synergistic flexor muscles can be prevented. New adhesion barrier materials seem to be available that could aid such prevention of restoring myofascial connections.

In any case the conclusion can be drawn that effects of myofascial force transmission should be considered in any step of performing surgery, but also during and after the process of recovery and adaptation to the newly created conditions.

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