ACUTE EFFECTS OF APONEUROTOMY PERFORMED AT MULTIPLE LOCATIONS ON MUSCULAR MECHANICS: ASSESSMENT BY FINITE ELEMENT MODELING

by

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ABSTRACT

ACUTE EFFECTS OF APONEUROTOMY PERFORMED AT MULTIPLE LOCATIONS ON MUSCULAR MECHANICS: ASSESSMENT BY FINITE ELEMENT MODELING

The specific goal of the present study is to assess the effects of the number of interventions on the acute effects of aponeurotomy by using finite element modeling. EDL muscle of rat with extramuscular connections was modeled with aponeurotomies at three different locations (Location P,I and D); four conditions including single (Case P), two double (Case P-I and Case P-D) and triple (Case P-I-D) interventions were studied. Muscle length-force characteristics, sarcomere length distributions and muscle geometry of multiple aponeurotomy cases were compared to the ones of single aponeurotomized muscle. It was shown that the intended acute mechanical properties of aponeurotomy were enhanced mostly by triple interventions, but even these enhancements were fairly limited: (1) In triple aponeurotomized muscle, further force reduction was small (e.g. distal optimal forces for Case P and P-I-D are 68% and 64% of that of intact muscle). (2) The distal length range of active force exertion was increased by only 0.025% by two additive interventions, whereas single intervention (Case P) increased this length range by 44% of that of intact muscle. The proximal length range was even narrowed with additional interventions. (3) The sarcomere length distributions were not altered with multiple aponeurotomies. Our results indicate that the multiple interventions in the aponeurotomy should be questioned in terms of their limited enhancements in acute mechanical effects. Nevertheless, the geometrical changes might have clinical importance and this effect should be studied.

Keywords: Aponeurotomy, Muscle mechanics, Finite element modeling, Extramuscular myofascial force transmission, EDL.

ÖZET

CERRAHİ APONÖROTOMİDE KESİ SAYISININ KAS MEKANİĞİNE AKUT ETKİLERİ: SONLU ELEMANLAR ANALİZİ

Çalışmanın temel amacı, kesi sayısının aponörotominin akut mekanik etkilerine katkısının sonlu elemanlar metodu ile incelenmesidir. Aponörotomize edilmiş ekstramüsküler bağlantılı sıçan EDL kası, üç farklı lokasyondaki kesiyle modellenmiş; tek (Koşul P), ikili (Koşul P-I ve P-D) ve üçlü (Koşul P-I-D) kesili olmak üzere dört farklı koşul çalışılmılştır. Çoklu kesi koşullarındaki kas boyu-kuvvet karakteristiği, sarkomer boy dağılımları ve kas geometrisi tek kesi koşulundakiler ile kıyaslanmıştır. Aponörotominin amaçlanan akut mekanik etkilerinin üçlü kesi ile arttığı, fakat bu artışın oldukça sınırlı kaldığı görülmüştür: (1) Üçlü kesi ile sağlanan kuvvet düşüşündeki ilerleme çok azdır (örneğin, Koşul P ve P-I-D'deki distal optimal kuvvetler sırasıyla, intakt kasın %68 ve %64'ü kadardır). (2) Kasın kuvvet üretebildiği boy aralığı, Koşul P ile intakt kasınkine oranla aralığı ilave kesilerle birlikte daralma göstermiştir. (3) Genel sarkomer boy dağılımında ise değişiklik görülmemiştir. Sonuçlarımız, cerrahi aponörotomide çoklu kesi uygulamasının akut mekanik etkiler yönünden katkısının sorgulanması gerektiğine işaret etmektedir. Ancak, kas geometrisinde oluşan değişimler klinik önem taşıyor olabilir ve bu araştırılmalıdır.

Anahtar Sözcükler: Aponörotomi, Kas mekaniği,Sonlu elemanlar analizi, Ekstramüsküler miyobağdokusal kuvvet iletimi, EDL kası.

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$\underline{S}$	Second Piola Kirchhoff Stress
W	Strain Energy Density Function
$\underline{L}^G$	Green-Lagrange Strain Tensor
I_i	Invariant of Strain Tensor
F_{ma}	Muscle Active Force
F_{mao}	Optimum Active Muscle Force
ΔF_{mt}	Proximo-distal Total Force Difference
ε_{ij}	Green-Lagrange Strains
σ	Cauchy Stress Tensor
κ	Bulk Modulus
ν	Poisson's Ratio

LIST OF ABBREVIATIONS

EDL	Extensor Digitorum Longus
GM	Gastrocnemius Medialis
lfmm	Linked fiber-matrix mesh
mm	Millimeter
N/mm^2	Newton per millimeter square
μm	Micrometer

1. INTRODUCTION

1.1 Skeletal Muscle

Muscle is an excitable soft tissue, the function of which is to generate and exert force to produce movement. The force production is maintained via contractions which refer as muscle shortening when it is stimulated by nerve. Highly specialized muscle tissue namely *skeletal muscle*, which generally acts voluntarily, is the prime mover of the body locomotion. They are striated muscles connected to the skeletal system and are responsible for body locomotion by transmitting the force they generate to bones and joints through tendons [1,2].

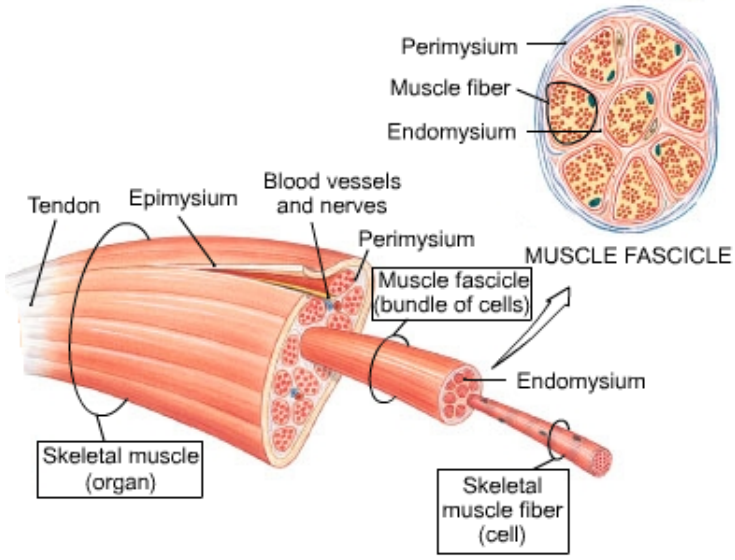

Figure 1.1 Schematic diagram of the organization of skeletal muscle[1]

The structure of muscle can be analyzed by considering muscle to be comprised of structural units with decreasing size (For hierarchical structure of muscle see Figure 1.1). The whole muscle is surrounded by fascia, which is a sheet of connective tissue covering or binding together body structures, and a further connective tissue known as epimysium. The epimysium separates the muscle from surrounding tissues and

organs. The perimysium divides the skeletal muscle into a series of compartments, each containing a fascicle which is a bundle of muscle fibers. In addition to collagen and elastic fibers, the perimysium contains blood vessels and nerves that maintain blood flow and innervate the fascicles. Each fascicle receives branches of these blood vessels and nerves. Within the fascicles, endomysium connective tissue surrounds the individual skeletal muscle fibers and enable the interconnections of adjacent muscle fibers. [1,3]

1.2 Muscle Fiber Structure

Muscle fibers are the individual muscle cells which in many ways, are like any other body cells. However because muscle cell function is highly specialized to produce force and movement, the cellular components are also highly specialized. Muscle fibers are comprised of *myofibrils* which lying parallel to each other. The typical striated pattern of the skeletal muscle is gained by the systematic arrangement of the myofibrils [4]. Myofibrils, a string of sarcomeres arranged in series are the largest functional unit of contractile filament. *Sarcomere* is the basic contractile element and the regularly repeating pattern in the muscle. It can be said that sarcomeres are the smallest functional units which still behave like a muscle [5].

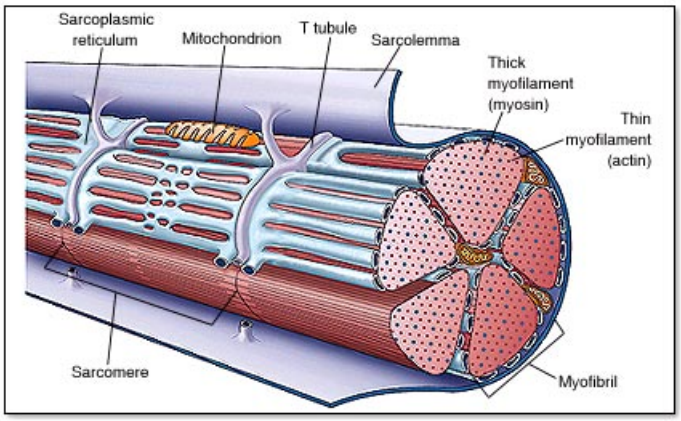

Figure 1.2 Schematic diagram of the organization of muscle fiber [2].

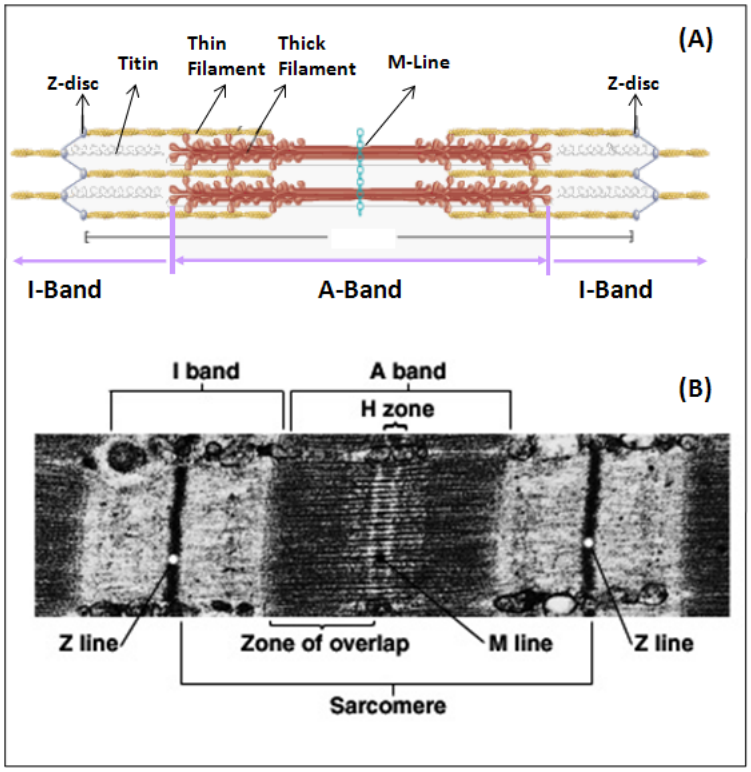

Figure 1.3 (A)Schematic representation and, (B) a corresponding view of a sarcomere in a myofibril from a muscle fiber in the gastrocnemius muscle of the calf under transmission electron microscope [1]

Sarcomeres are composed of contractile filaments termed myofilaments: myosin (thick filaments) and actin (thin filaments). (Figure1.3) The dark bands, namely A-Band comprised of myosin filaments as well as some actin filaments which overlap and build cross bridges with thick filaments. The I-band is the light area of the sarcomere at where only actin filaments present. The thick filament is connected from the M-Line to the Z-Disc by titin which provides binding sites for numerous proteins and is thought to play an important role as sarcomeric ruler and as draft for the assembly of the sarcomere[1,3].

The myosin heads have the ability to establish a link between the thick and thin filaments; they are called as cross-bridges. The force is produced within the sarcomeres as a result of contraction which is established by these cross-bridges.

1.3 Force Transmission Pathways

The movement in the vertebrates is maintained by the transmission of the force that is generated by the activation of contractile elements within the muscle fibers onto the bones of the skeleton [6]. Tendon collagen fibers are attached to the invaginations of sarcolemmal-endomysium complex at one or both ends of muscle fibers. Such *myotendinous junctions* provide the transmission of muscle force onto the tendon and are widely accepted as the exclusive pathway for transmission of muscle force onto the bony skeleton, namely *myotendinous force transmission*. However, many previous studies have shown that also other pathways present by means of muscle force is transmitted indirectly to the bone. This kind of transmission is referred to as *myofascial force transmission* [6,7].

1.3.1 Myotendinous Force Transmission

The myotendinous force transmission, in which the muscle force generated within muscle fibers is transmitted onto the tendon and to the muscle via the tendon, is widely accepted as the exclusive pathway for force transmission to cause movement. The morphological specialization of the muscle fiber ends reveals this mechanism. As myofibers approach tendon at origin or insertion, their diameter decreases in size considerably and the sarcolemma greatly folds in the longitudinal direction. Collagen fibers are present in these folds which are referred as invaginations, and the force is assumed to be transmitted from the intracellular medium to these collagen fibers [6]. At each end of muscle fibers; epymisium, perymisium and endomysium come together and form an attachment to aponeurosis, which is a broad tendinous sheet that may serve as the origin or insertion of a skeletal muscle, via the collagen fibers [1]. The tendon and aponeurosis form indirect attachments and force transmission from muscles to the periosteum of bones or to the connective tissue of other muscles[1,8]. (Figure1.4)

The results of myotendinous junctions being exclusive channel of force transmission would be: (1) Muscle fibers transmitting their forces only in longitudinal direction

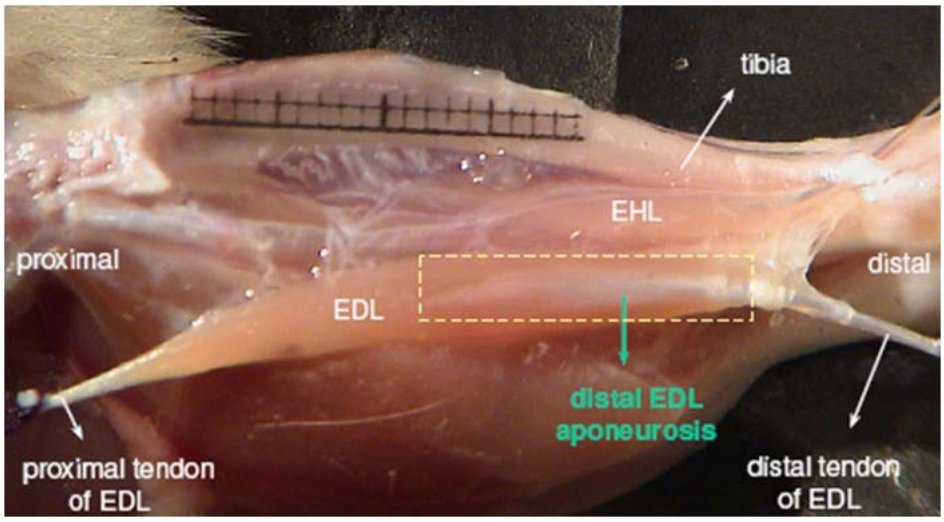

Figure 1.4 EDL muscle with its proximal and distal aponeuroses and extramuscular connections

(in series), and, (2) Measuring equal forces from two ends of the muscles. In the classical approach, these issues are widely to be valid. However, many previous studies have shown that the myotendinous junction is not the only pathway for force transmission; also other structures play substantial roles in muscle force transmission. (For review see [9])

1.3.2 Myofascial Force Transmission

The skeletal muscle may be described as muscle fibers being contractile elements surrounded by tunnel like network of connective tissue. Myofibers are connected to the extracellular space by the trans-srcolemmal proteins. Myofibers that are located parallel to each other are connected to one to another by means of these sarcolemmal cytoskeleton. Moreover; besides transmission at the end of fibre, previous experiments on single fibre [10] showed that myofibrillar force is transmittable also through the sarcolemma-endomysium complex along the fibre.

Furthermore, in natural environment, the skeletal muscles are connected to their muscles and non-muscular structures; thus they are not isolated. These connections between muscles' extramuscular matrix and (i) surrounding muscles called intermuscular

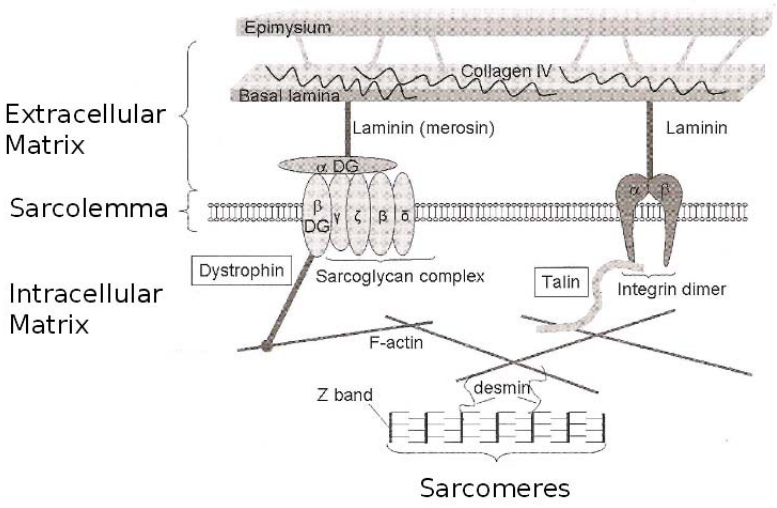

Figure 1.5 Schematic view of organization of transsarcolemmal connections between the cytoskeleton and the endomysium [7]

connections (i.e., collageneous connections at the immediate interface between muscle bellies) and (i) surrounding non-muscular tissues are referred to as extramuscular connections (comprised of neurovascular tracts in addition to compartmental boundaries).

All these mechanical relations have shown to play important roles in muscular force transmission and are proven to be an additional pathway to the myotendinous pathway. This kind of force transmission mechanism is referred as myofascial force transmission [6].

The pathways of myofascial force transmission are classified in three groups according to the type of connections that the transmission takes place at:

i) Intramuscular myofascial force transmission: The force generated by the sarcomeres is transmitted to the transsarcolemmal proteins by the help of cytoskeleton. The transmission of force is maintained by the means of these paths onto the endomysium (Figure1.5).

Previously, the endomysium of muscle fibre is shown to be continuous with the endomysium of neighbouring fibers, as well as with the perimysium and the epimy-

sium. Endomysium, perimysium and epimysium build a network of connective tissue altogether within the muscle. The muscle fibers, fascicles and whole muscle are embedded in this network (Figure1.5) Therefore, the muscle force is transmitted from the muscle fibre onto the endomysium, and it is transmitted throughout the entire muscle from this path. Such transmission within the skeletal muscle is referred to as intramuscular myofascial force transmission[6].

One of the studies that showed the occurrence of such transmission is the tenotomy experiment of rat extensor digitorum longus muscle (EDL). Subsequent tenotomy heads of EDL in proximal-distal order caused only very minor drop in muscle force. Consequently, muscle force generated muscle fibers are transmitted myofascially onto the muscles' tendon by means of intramuscular connective tissue network without passing one or more of the myotendinous junctions [11].

ii) Intermuscular myofascial force transmission: The epimysium of the muscle is shown to be connected to the epimysia of neighboring synergist muscles. Consequently, the intramuscular myofascial stroma from adjacent muscles is continuous. It is shown that these intermuscular myofascial connections are sufficiently stiff to transmit forces. Therefore, intermuscular myofascial connections maintain the force transmission directly between adjacent muscles by means of their epimysia. Such force transmission is referred to as intermuscular myofascial force transmission[12].

iii) Extramuscular myofascial force transmission: The force transmission from muscles' extracellular matrix onto the surrounding non-muscular elements of compartement, such as fascie, intermuscular septa, interrosseal membranes, epitendinous tissues and neurovascular tracts and bones is referred to as extramuscular myofascial force transmission. [7] Particularly, the collagen reinforcement of neurovascular tract is connected to muscles' intramuscular stroma.

By the indication of such continuities, the connective tissues of a compartment can be considered as a unit. Therefore, force can be transmitted between muscle fibers of different muscles by means of inter- as well as extramuscular myofascial

pathways[7]. Such force transmission pathways are shown to have substantial affects on muscle mechanics, including (1) pronounced proximo-distal force differences, (e.g. [13]) (2) sarcomere length distributions throughout the muscle (e.g. [13]) (3) alteration of length-force characteristics of a muscle in relation to its position according to its synergist muscles.

1.4 Aponeurotomy as Remedial Surgery

In patients with spastic movement disorders such as cerebral palsy, motor impairment is due to a number of deficits, such as poor muscle control, weakness, impaired balance, hypertonicity, and spasticity. As a consequence, muscles are contracted over time and later become even structurally short with respect to their antagonist. Due to such contractures and muscle shortening, muscles' length range of force exertion becomes limited. This limitation may cause severe changes in preferred joint position, restriction in joint range of motion and consequently movement disorders([14-16).

In case of extremely short muscles, surgical interventions are performed onto the muscle in order to restore the joint range of motion. These interventions might be performed outside the muscle belly on the tendon or at the muscle belly by incisions on aponeurosis which is referred to as aponeurotomy. In the aponeurotomy technique, the intramuscular aponeurosis is cut at one or usually more incisions in the direction perpendicular to its longitudinal direction (Figure1.6). After aponeurotomy, the joint angle is adjusted in order to bring the muscle to high length after surgery. The limb is usually is placed in a cast for the recovery period.([14,17]) The aponeurotomy technique has two main goals: (i) to lengthen the overly short muscle (i.e. increasing the length range of force exertion) and (ii) weakening of the muscle if there is force imbalance between antagonistic muscles. The clinical effectiveness of aponeurotomy surgery in the short term is reported by many researchers before [18,19]. However in the long term, some patients show recurrence of limited range of joint motion (e.g. [18]). The mechanisms of aponeurotomy leading to success or failure are poorly known. Recent experimental studies on isolated rat muscles provided information regarding both acute

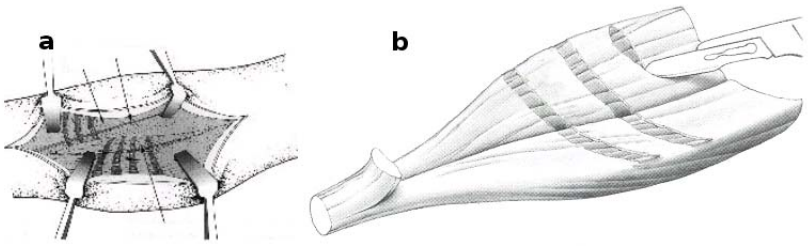

Figure 1.6 Lengthening of triceps surea muscles with intramuscular aponeurotomy (a) in situ during surgery [17], (b) the nature of the intervention shown on gastrocnemius muscle(modified from[15])

[16,20,21] and long term [21,22] effects of aponeurotomy. Moreover, modeling studies on isolated [23] and extramuscularly connected [24,25] rat muscles gave the chance to see the detailed analysis of the effects of aponeurotomy. However all these studies have examined the effects of aponeurotomy with single intervention.

1.5 Goal of the Study

In aponeurotomy technique, applying multiple interventions instead of a single intervention is frequently used. Many researchers referred multiple recessions on aponeurosis by applying two or more interventions(e.g.[15,26,27]). According to the procedures reported in those studies, the numbers and locations of the incisions show alterations among them: There is not any procedural consensus. More importantly, it is not known about the effects of these variations on muscle mechanics.

The specific goal of the present study is to investigate the acute effects of number and locations of aponeurotomies on muscle mechanics. By using finite elements analysis, rat EDL muscle is modeled with extramuscular connections and variations in number of aponeurotomies is considered on the model. For this purpose, previously developed "linked fiber-matrix mesh model" is advanced such that multiple aponeurotomy conditions is studied. It is aimed at how the increase in number of aponeurotomies affects acutely muscle length-force characteristics as well as muscle geometry and sarcomere length distributions.

2. METHODS

2.1 Description of the "Linked Fiber-Matrix Mesh Model"

Using the linked fiber-matrix mesh model (lfmm model [28]) , skeletal muscle is considered explicitly as two separate domains: (1) the intracellular domain and (2) extracellular matrix domain. The transsarcolemmal attachments are considered as elastic links between the two domains.

Two self-programmed elements were developed and were introduced as user-defined elements into the finite element program ANSYS 9.0. One of these elements (*extracellular matrix element*) represents the collagen reinforced extracellular matrix, which includes the basal lamina and connective tissue components such as endomysium, perimysium and epimysium. A second element models the muscle fibers (*myofiber element*). Within the biological context, each combined *muscle element* represents a segment of a bundle of muscle fibers with identical material properties, its connective tissues and the links between them. This is realized as a linked system of extracellular matrix and myofiber elements (for a schematic 2D-representation of an arrangement of these muscle elements see [28]). A whole fascicle is constructed by putting three muscle elements in series.

In the lfmm model, the extracellular matrix domain is represented by a mesh of extracellular matrix elements (*matrix mesh*). In the same space, a separate mesh of myofiber elements is built to represent the intracellular domain (*fiber mesh*). The two meshes are rigidly connected to single layers of elements modeling proximal and distal aponeuroses: a node representing myotendinous connection sites is the common node of all three (extracellular matrix, myofiber and aponeurosis) elements. In contrast, at the intermediate nodes, fiber and matrix meshes are linked elastically to represent the transmembranous attachments of the cytoskeleton and extracellular matrix. For these links (the model includes a total of 28 of them: 14 in each of the upper and lower model

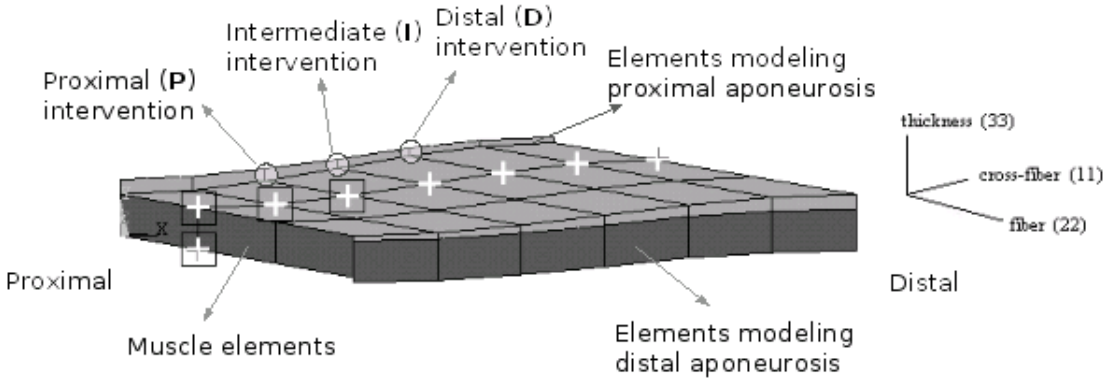

Figure 2.1 The model of EDL muscle, locations of aponeurotomy and of modeled extramuscular connections. A 3D local coordinate system representing the fiber, cross-fiber (normal to the fiber direction in the transverse plane), and thickness directions is used for analysis and presentation of the model results. The nodes of the matrix mesh marked by a white "+" sign have connections to mechanical ground representing the muscles' extramuscular connections. The part of neurovascular tract represented in the model is marked also by a black square show the stiffer proximal segment of extramuscular connections. The nodes at the upper face of the model marked by a circle indicate the different locations of aponeurotomy modeled. Proximal aponeurotomy is modeled by disconnecting the common nodes of two neighboring aponeurosis elements located in the middle of the proximal aponeurosis of the modeled muscle. Note that the nodes at the same location of the lower face of the model are also disconnected.

surfaces) the standard element, COMBIN39 is used from the element library of ANSYS 9.0. This is a 2-node spring element, which is set to be uniaxial and have linear high stiffness characteristics representing non-pathological connections between the muscle fibers and the extracellular matrix (for an analysis of the effects of stiff or compliant links, see [28]). Note that at the initial muscle length and in passive condition, these links have a length equaling zero.

Both extracellular matrix and myofiber elements have eight nodes; linear interpolation functions and a large deformation analysis formulation are applied. A 3D local coordinate system representing the fiber, cross-fiber (normal to the fiber direction), and thickness directions is used. The stress formulation, $\underline{S}$ based on Second Piola-Kirchoff definition constitutes the derivative of the strain energy density function, W with respect to the Green-Lagrange strain tensor,$\underline{L}^G$.

$$\underline{S} = \frac{dW}{d\underline{L}^G} \tag{2.1}$$

2.1.1 Extracellular Matrix Element

The strain energy density function mechanically characterizing the extracellular matrix includes two parts:

$$W = W_1 + W_2 \tag{2.2}$$

The first part represents the non-linear and anisotropic material properties [29]:

$$W_1 = W_{ij}(\varepsilon_{ij}) \tag{2.3}$$

where

$$\begin{aligned} W_{ij} &= k.(e^{a_{ij}.z_{ij}} - a_{ij}.\varepsilon_{ij}) \text{ for } \varepsilon_{ij} > 0 \text{ or,} \\ W_{ij} &= -W_{ij}(|\varepsilon_{ij}|) \text{ for } \varepsilon_{ij} < 0 \text{ and } i \neq j. \end{aligned} \tag{2.4}$$

ε_{ij} are the Green-Lagrange strains in the local coordinates. The indices $i = 1...3$ and $j = 1...3$ represent the local cross-fiber, fiber and thickness directions respectively. a_{ij} and k are constants (Table 1). Note that, initial passive stiffness (k) and passive fiber direction stiffness (a_{22}) values were estimated by fitting the experimental data by Meijer et al[30]. Based on the experimental data on dog diaphragm [31], passive cross-fiber stiffness ($a_{11} = a_{33}$) was taken to be higher than fiber direction stiffness. Identical values are used for fiber and fiber-cross fiber shear stiffness ($a_{12} = a_{23} = a_{31}$). However, in contrast to the non-symmetric stress-strain relationships defined for fiber and cross-fiber directions, a symmetric stress-strain relationship is used for shearing (for the resulting stress-strain curve see Figure 2.2).

The second part includes a penalty function to account for the constancy of muscle volume. The intracellular fluid and solid elastic structures are considered as separate constituents of muscle tissue with different responses to deformation: The intracellular fluid is assumed to have the ability to migrate freely within the cell, whereas, the solid elastic structures housing muscle fibers (e.g. basal lamina and endomysium) are restricted in moving as they are being constrained by neighboring cells. Therefore,

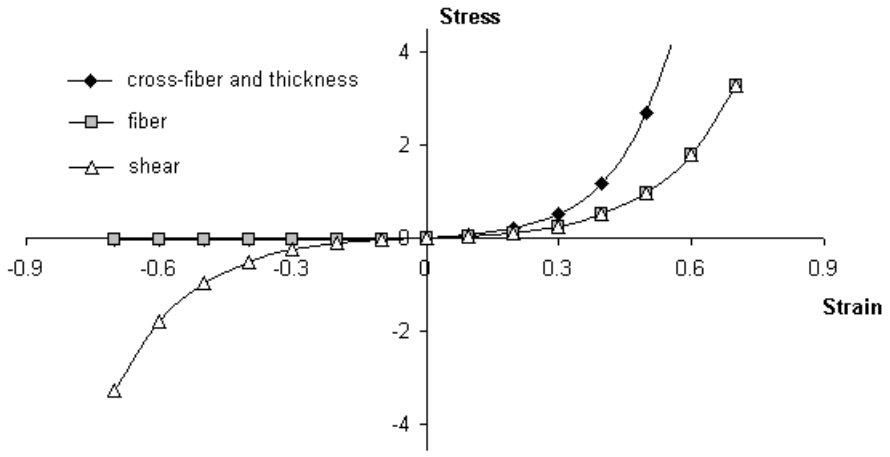

Figure 2.2 EDL muscle with its proximal and distal aponeuroses and extramuscular connections

a penalty function consisting of two parts was used:

$$W_2 = S_s.(I_3 - 1)^2 + S_f.(I_3^{avg} - 1) \tag{2.5}$$

where I_3 is the third invariant (determinant) of the Right Cauchy-Green strain tensor providing a ratio of the deformed local volume over the undeformed local volume for each Gaussian point.

If all I_3's are kept as unity, the element is considered as solid and the local volumes are conserved. If the weighted mean of all I_3's per element, (I_3^{avg}) is kept as unity, the element is considered as a fluid. The penalty parameters S_s(for the solid volume) and S_f(for the fluid volume), allow determining the penalty given for each part. Note that if both I_3's and I_3^{avg}'s are unity the volume is constant. The parameters S_s and S_f (Table 1) chosen after performing specific tests on the extracellular matrix element allow a good representation of constancy of muscle volume with optimal numerical efforts: for even very large deformations (e.g. length changes greater than 40% the maximal deviation from undeformed volume remains below 5%).

2.1.2 Myofiber Element

Maximally activated muscle is studied. Sarcomeres within the muscle fibers, are assumed to have identical material properties. The force-velocity characteristics are not considered due to the isometric nature of the present work. The total stress for the intracellular domain (σ_{22f}) is a Cauchy stress acting in the local fiber direction exclusively and is the sum of the active stress of the contractile elements ($\sigma_{22contr}$) and the stress due to intracellular passive tension (σ_{22icp}).

To define the active length-force characteristics, an exponential function (for the stress-strain curve see Figure2.3) was fit to the experimental data of isolated small rat gastrocnemius medialis (GM) fiber bundles [32]. This function is scaled such that at optimum length, the fiber direction strain (ε_{22}) is zero and the maximal stress value is unity.

$$\sigma_{22contr}(\varepsilon_{22}) = b_3 e^{b_2 e_{22}^3} \text{ for} \varepsilon_{ij} > 0$$

or,

$$\sigma_{22contr}(\varepsilon_{22}) = \quad b_3 e^{b_1 e_{22}^4} \text{ for } \varepsilon_{ij} < 0. \tag{2.6}$$

where b_1, b_2 and b_3 are constants (Table 1).

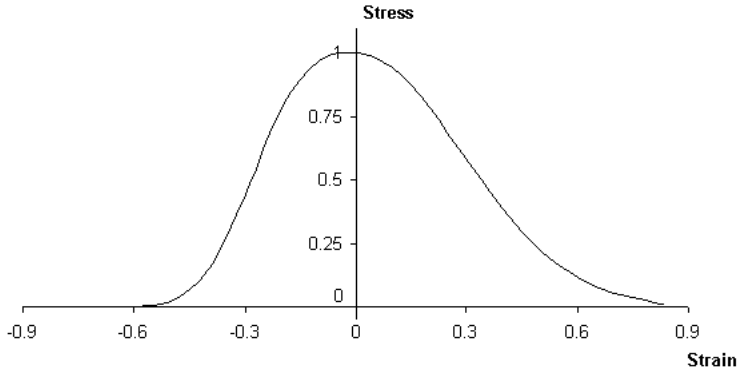

Figure 2.3 Active stress and strain relationship of the myofiber element representing the contractile apparatus. The curve is normalized for the maximum value of active contractile stress. Note that the active mechanical property of the myofiber element is valid only for the local fiber direction.

The source of intracellular passive tension is the intra-sarcomeric cytoskeleton[33], which is composed of several proteins. In this work titin is considered to play the dominant role. Experimental tension-sarcomere length data for a single rabbit skeletal muscle fiber was fitted using a parabolic function and scaled to make it compatible to the stress-strain characteristics of the contractile part (Figure2.4).

$$\sigma_{22icp}(\varepsilon_{22}) = t_1\varepsilon_{22}^2 + t_2\varepsilon_{22} + t_3 \text{ for } \varepsilon_{22} > 0$$

and

$$\sigma_{22icp}(\varepsilon_{22}) = 0 \text{ for } \varepsilon_{22} < 0 \tag{2.7}$$

where t_1, t_2 and t_3 are constants (Table 1).

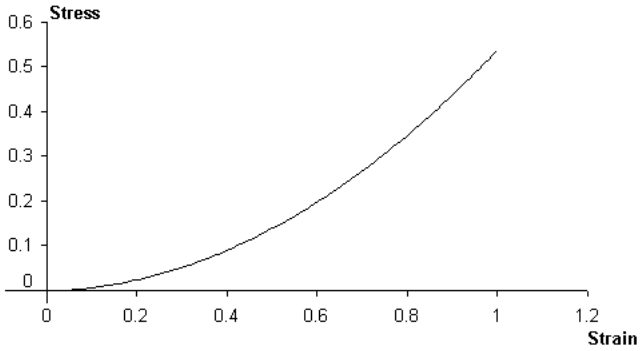

Figure 2.4 Mechanical properties representing the cytoskeleton of which the titin filaments dominates the passive resistance in the myofiber element. The curve is normalized for the maximum value of active contractile stress. Note that passive mechanical property of the myofiber element is valid only for the local fiber direction.

2.1.3 Aponeurosis Element

To represent the aponeuroses, a standard 3D, 8-node element HYPER58, from the element library of ANSYS 9.0 is used. This element has a hyperelastic mechanical formulation for which the strain energy density function is defined using the two parameter Mooney-Rivlin material law:

$$W = a_{10}(\overline{I}_1 - 3) + a_{01}(\overline{I}_2 - 3) + \frac{\kappa}{2}(\overline{I}_3 - 1)^2 \tag{2.8}$$

where,$\overline{I}_i$ are reduced invariants of right-Cauchy strain tensor for $i = 1...3$, a_{10} and a_{01} are Mooney-Rivlin material constants, $\kappa = 2(a_{10}+a_{01})/(1-2\nu)$ is the bulk modulus and ν is the Poisson's ratio. The parameters used (Table 2.1) ensure sufficient stiffness for the aponeuroses for a representative role in force transmission and providing muscular integrity as in real muscle.

It is assumed that, at the initial muscle length in the passive state, the sarcomeres arranged in series within muscle fibers have identical lengths. Local strain, as a measure of change of length, reflects the lengthening (positive strain) or shortening (negative strain) of sarcomeres. 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). Fiber direction strain within the fiber mesh of the lfmm model was used to assess the non-uniformity of lengths of sarcomeres arranged in-series within muscle fibers (referred to as serial distribution). Muscle length equaling 25.2 mm and 31.2 mm will be referred to as *low muscle length* and *high muscle length* respectively.

2.2 Aponeurotomized EDL Muscle Models With Extramuscular Connections

EDL muscle of the rat was modeled. This muscle has a relatively simple geometry: it is a unipennate muscle with rather small pennation angles and minimal variation of the fiber direction within the muscle belly. The geometry of the model (Figure 2.1) is defined as the contour of a longitudinal slice at the middle of the isolated rat EDL muscle belly. Three muscle elements in series and six in parallel fill this slice. All aponeurosis elements have identical mechanical properties but using a variable thickness in the fiber-cross fiber plane, the increasing cross-sectional area of the aponeurosis toward the tendon is accounted for.

An extramuscular connective tissue connects EDL all along the muscle to the

Table 2.1
Values and definitions of the model constants.

Constant	Value	Unit	Deformation
k	0.05	N/mm^2	Initial passive stiffness (Eq. 2.4)
a_{11}	8.0	-	Passive cross-fiber direction stiffness, $a_{11} = a_{33}$ (Eq. 2.4)
a_{22}	6.0	-	Passive fiber direction stiffness (Eq. 2.4)
a_{12}	6.0	-	Passive fiber-cross-fiber shear stiffness, $a_{12} = a_{23} = a_{31}$ (Eq. 2.4)
S_s	10.0	N/mm^2	Weight factor in the penalty function for the solid volume (Eq. 2.5)
S_f	80.0	N/mm^2	Weight factor in the penalty function for the fluid volume (Eq. 2.5)
b_1	30.0	-	Coefficient for the stress-strain relation of the contractile elements (Eq. 2.6)
b_2	-6.0	-	Coefficient for the stress-strain relation of the contractile elements (Eq. 2.6)
b_3	1	-	Coefficient for the stress-strain relation of the contractile elements (Eq. 2.6)
t_1	0.522	-	Coefficient for the stress-strain relation of the intracellular passive elements (Eq. 2.7)
t_2	0.019	-	Coefficient for the stress-strain relation of the intracellular passive elements (Eq. 2.7)
t_3	-0.002	-	Coefficient for the stress-strain relation of the intracellular passive elements (Eq. 2.7)
a_{10}	7.9	N/mm^2	Mooney-Rivlin material constant for aponeurosis elements (Eq. 2.8)
a_{01}	7.9	N/mm^2	Mooney-Rivlin material constant for aponeurosis elements (Eq. 2.8)
ν	0.3	-	Poisson's ratio for aponeurosis elements (Eq. 2.8)

tibia, part of interosseal membrane and anterior intermuscular septum. This structure defines the anatomical path of extramuscular myofascial force transmission and provides connections to bone, in addition to myotendinous connections to muscle origin and insertion. In our previous experimental study , the locations of the extramuscular connections to EDL muscle were determined to be predominantly at one-third of the fascicle length from the most proximal end of each muscle fascicle. In that study it was also shown that the neurovascular tract (i.e. extramuscular connective tissue structure embedding and reinforcing nerves and blood vessels) supplying the EDL muscle proximally are much stiffer than the distal of the connective tissue structure.

In order to model the muscles' extramuscular connections and to account for their continuity with the muscular extracellular matrix, a set of nodes of the matrix mesh were linked using spring elements (COMBIN39, from the element library of ANSYS 9.0) to a set of fixed points (Figure 2.1). Our modeling considerations were: (1) the set of fixed points comprising "mechanical ground" represent bone, which is assumed to be rigid. (2) the spring elements modeling the muscles' extramuscular connections were set to be uniaxial and have linear length-force characteristics. (3) Initially (i.e. muscle length = 28.7 mm, and before changing any of the tendon positions), the fixed points and the corresponding nodes of the model were at identical locations (i.e., the spring elements modeling the muscles' extramuscular connections were at a length of zero). (4) The higher stiffness of the connective tissues constituting the neurovascular tract near the EDL muscle is taken into account by making the three most proximal links to the muscle stiffer than distal ones. Stiffness values determined previously were used (i.e. k = 0.286 unit force/mm for stiffer part and, k = 0.067 unit force/mm for the more distal links).

In surgery, after cutting its aponeurosis transversely (Figure 1.6), the muscle is lengthened passively. Experiments and previous finite element modeling on aponeurotomized rat muscle showed that below the location of the aponeurotomy, intramuscular connective tissue ruptures in the muscle fiber direction on activation leading to a gap separating the two cut ends of the aponeurosis. Such effects increase progressively after isometric activity at higher muscle length. Presumably in human patients, further

rupturing occurs on activation of the immobilized muscle after the actual operation.

The effects of proximal aponeurotomy were modeled by disconnecting the common nodes of two neighboring aponeurosis elements in the targeted location of the proximal aponeurosis (Figure 2.1), as well as the two parallel arranged muscle elements located below it. Three separate aponeurotomy locations were studied: i) a proximal location (*location P*), ii) an intermediate location (*location I*) and iii) a distal location (*location D*).

Note that: (1) In the model, the tear depth is limited by the length of the proximal muscle elements in the fiber direction. (2) Location P (i.e., the most proximal aponeurotomy studied) is located at a third of the proximal aponeurosis length from its proximal end. (3) Intramuscular connective tissue rupture creates two populations of muscle fibers: proximal population (with intact myotendinous connections to muscle origin and insertion) and distal population (without myotendinous connection to the muscles' origin, but intact myotendinous connections to muscle insertion).

Extramuscularly connected aponeurotomized EDL muscle was modeled in several conditions including a single, double and triple aponeurotomy performed on the proximal aponeurosis at: i) location P exclusively (*Case P*), ii) locations P and I (*Case P-I*), iii) locations P and D (*Case P-D*) and iv) locations P, I and D (*Case P-I-D*). Note that to ensure a systematical assessment of the effects of multiple interventions, Case P was considered as the reference of our present study and the additional interventions were located distally with respect to location P.

2.3 Solution Procedure

The analysis type used in ANSYS was static and large strain effects were included. During the entire solution procedure, the models studied were stable and no mesh refinement was performed. A force based convergence criterion was used with a tolerance of 0.5%.

Initially, at the passive state, the activation coefficient b_3(Eq. 2.6) equaled 0. Maximal activation of the muscles modeled was achieved by increasing b_3 incrementally up to 1, using fixed increments. Subsequent to activation, the proximal end of the muscles modeled was displaced distally by 2 mm which position was then kept constant during the entire modeling. Muscle length was altered by changing the position of muscle distal tendon: first in proximal direction (i.e. to shorten the muscle) then in distal direction (i.e. to lengthen the muscle).

3. RESULTS

3.1 Effects of Multiple Interventions on the Geometry of Aponeurotomized Muscle

Figure 3.1 shows distributions of fiber direction strain and the deformed geometry of the aponeurotomized muscles at low and high muscle lengths. Regardless of the number of aponeurotomies performed, each intervention leads to a gap separating the cut ends of aponeurosis, the length of which is a function of muscle length: Gap length, which cause muscle length to increase, are not constant, but increase with increasing muscle length and force.

The additional effects of multiple interventions that cause changes in muscle geometry are sizable:

(1) For the multiple aponeurotomies, the lengths of the gaps are not equal. The gap with the highest length (referred to as the main gap) always occurs at the location of the most proximal intervention (Location P). For example, in Case P-I-D, at high muscle length, the main gap length is 88% higher than the sum of the length of the other two gaps).

(2) For all muscle lengths studied, total gap length increases (Figure 3.2) with an increasing number of interventions (e.g. at high muscle length, total gap length increase compared to the length of the gap for Case P was by 16%, 25% and 27%, respectively for Cases P-I, P-D and P-I-D). However, the length of the main gap decreases as a function of increasing number of interventions (e.g. at high muscle length, compared to the length of the gap in Case P, the length of the main gap for Cases P-I, P-D and P-I-D is lower by 16%, 16% and 17%, respectively).

(3) The angle between the distal muscle fibers and muscle line of pull increases

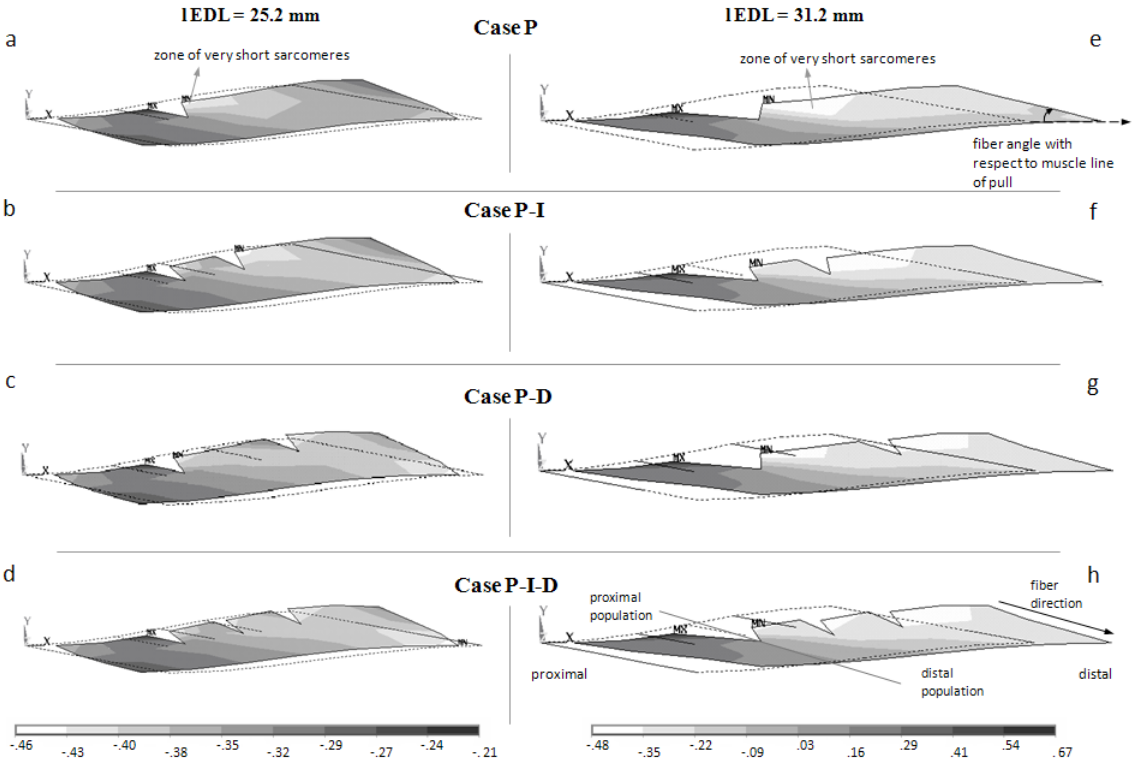

Figure 3.1 Muscle geometry and fiber direction strains within modeled aponeurotomized muscles. The dotted line contour indicates intact passive muscle geometry at the initial length. The local fiber direction, as well as the proximal and distal ends of the muscle are indicated (see contour h). The fiber angle with respect to the muscle line of pull is shown for the most distal muscle fascicle (see contour e). Left panels(a-d): for low muscle length(25.2 mm), Right panels(e-h): for high muscle length(31.2 mm).

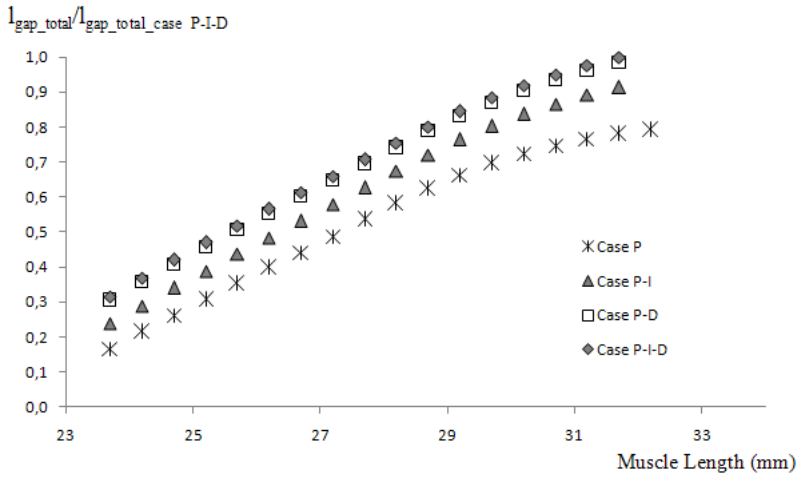

Figure 3.2 Effects of muscle length on the total gap length within the EDL proximal aponeurosis. In the case of one intervention, total gap length equals to the length of the single gap. Data are normalized for maximum gap length of Case P-I-D.

with an increasing number of interventions (e.g. The angle between the most distal fiber (see Figure 3.1e for definition) at high muscle length, is 13.8°, 15.5°, 16.6° and 16.9° for Cases P, P-I, P-D and P-I-D, respectively). This is indicative of some additional fiber shortening allowed by the added gaps. In contrast, such angle between the most proximal muscle fiber and muscle line of pull was not affected by additional interventions. We conclude that the effects of multiple interventions on muscle geometry are substantial predominantly for the distal population of muscle fibers.

3.2 Effects of Multiple Interventions on Sarcomere Length Distributions

For all cases, the most proximal intervention determines the fraction of the muscle comprised of muscle fibers without myotendinous connection to the muscles' origin (i.e., distal population of muscle fibers). Therefore; despite the sizable geometrical differences, as cases have same proximal intervention location, the amount of muscle tissue comprising the distal population of muscle fibers is identical. Nevertheless, the effects of multiple interventions on sarcomere length distributions differ for the proximal and distal populations.

Distal Population of Muscle Fibers :

The dominant effect of any aponeurotomy, characterized by substantially shortened sarcomeres at the proximal ends of muscle fibers of the distal fiber population is found in all cases (Figure 3.1). Even at high muscle length, these sarcomeres are at the ascending limb of their length-force characteristics.

However, a more pronounced shortening of sarcomeres was shown to be a function of increasing number of interventions: (1) the shortest sarcomeres within the aponeurotomized muscle were encountered just below the distal edge of the cut, marking the beginning of a zone of very short sarcomeres. Each additional intervention also

yields such a zone and hence, both for low and high muscle lengths, the total area of very short sarcomeres becomes larger as a function of increasing number of interventions. (2) The mean value (negative) of the fiber direction strains calculated for the distal population of muscle fibers showed a limited increase with added interventions: i) at low muscle length, average sarcomere shortening was 37%, 38%, 38% and 39% respectively, for Cases P, P-I, P-D and P-I-D and ii) at high muscle length, average sarcomere shortening was 26%, 29%, 30% and 31% respectively, for Cases P, P-I, P-D and P-I-D.

Proximal Population of Muscle Fibers:

Effects of number of interventions on the distribution of sarcomere lengths in the proximal population of muscle fibers were present but minor. Despite the increased number of interventions: (1) the general pattern of sarcomere length heterogeneity (i.e., the longest sarcomeres located at proximal ends of muscle fibers and shorter sarcomeres towards the distal ends) was not changed. (2) The mean value (positive) of the fiber direction strains calculated for the proximal population of muscle fibers was similar: i) at low muscle length, the mean value was unchanged for all cases and ii) at high muscle length, it decreased only a little by adding interventions (on average, sarcomere lengthening was 19% for Case P and 18% for all multiple aponeurotomy cases).

We conclude that within the distal population of muscle fibers, especially at higher muscle lengths additional sarcomere shortening occurs as a function of increasing number of interventions whereas, multiple aponeurotomy causes no major effects on sarcomere length distributions within the proximal population.

3.3 Effects of Multiple Aponeurotomy on Muscle Length-Force Characteristics

Figure3.3 shows effects of an increasing number of aponeurotomy and their locations on passive and active muscle length-force characteristics.

Some reduction in active muscle force Both distally (Figure 3.3a) and proximally (Figure 3.3b) exerted active forces are higher for aponeurotomized muscle with a single intervention (Case P) then after multiple aponeurotomy. This shows that some reduction in muscle active force is caused by the additional interventions with Case P-I-D showing the highest force reduction. However, for both higher and lower muscle lengths, the differences in forces between the multiple aponeurotomy cases and Case P remained limited to approximaly 5-11%. At low muscle length (i.e. 25.7 mm); i) distal active forces of Cases P-I, P-D and P-I-D equal 95%, 95% and 94% respectively of that of Case P and ii) for proximal active forces these values were 91%, 91% and 89% respectively of that of Case P. At optimum length, i) distal optimal forces of Cases P-I, P-D and P-I-D represent 95%, 95% and 93% respectively of that of Case P and ii) proximal optimal forces of Cases P-I, P-D and P-I-D represent 93%, 93% and 91% respectively of the one of Case P.

Distributions of fiber direction active stress within the model fiber mesh indicate the contribution of sarcomeres at different locations to active muscle force (Figure 3.4). For the distal population of muscle fibers, aponeurotomy in general reduces fiber direction stress (due to substantial shortening of the sarcomeres), an effect that increases as a function of number of interventions: (1) At low muscle length, this effect is small: the mean value of fiber direction stress equals, 0.16, 0.14, 0.13 and 0.12 for Cases P, P-I, P-D and P-I-D respectively. (2) At high muscle length this effect is more pronounced: the mean value of fiber stress equals for Cases P, P-I, P-D and P-I-D respectively 0.49, 0.39, 0.38 and 0.33. In contrast, for the proximal muscle fiber population, the fiber direction stress values remain high in general, and variation of local stress was minor for multiple interventions: e.g., at high muscle length, the mean

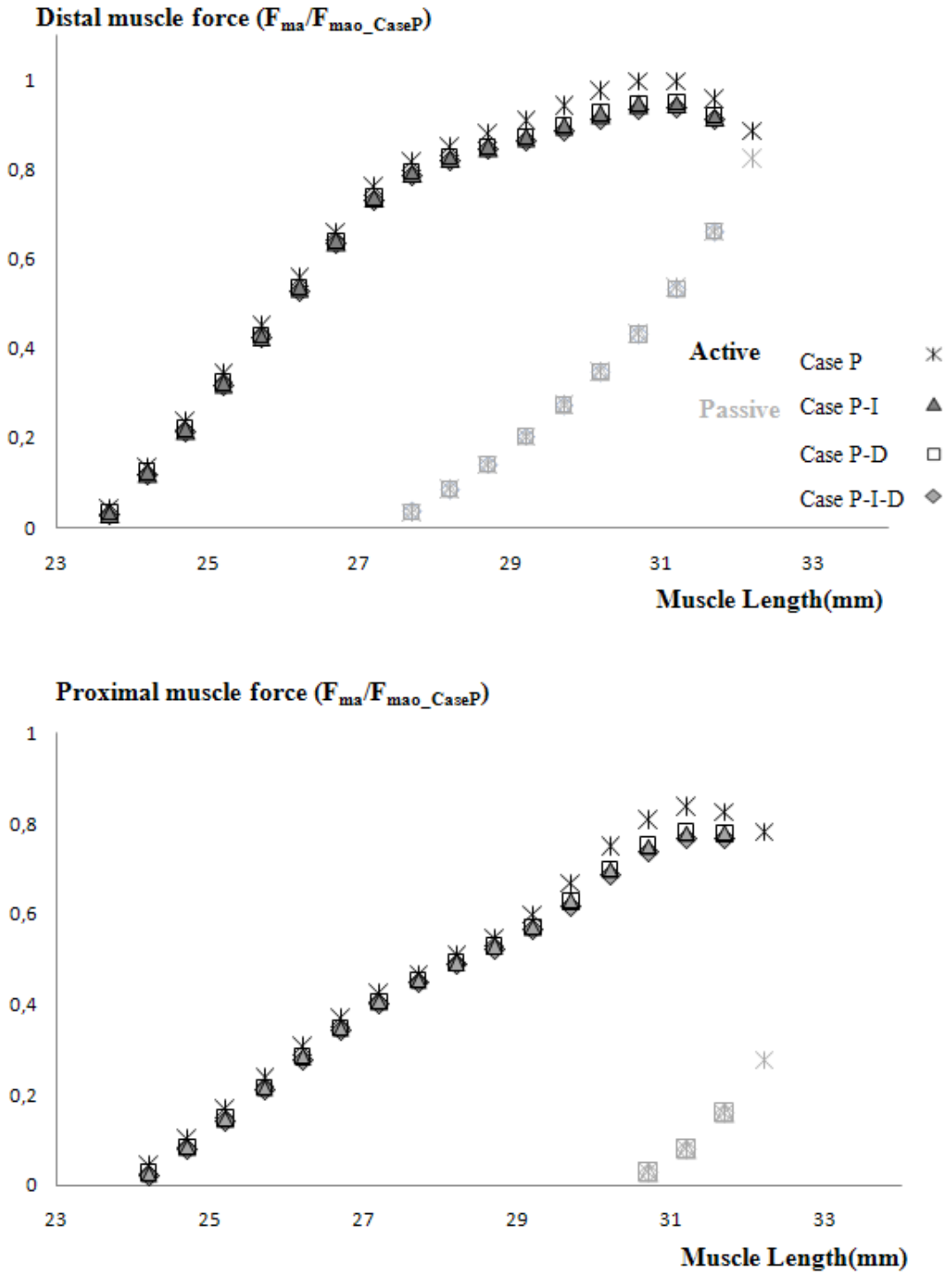

Figure 3.3 Isometric muscle length-force characteristics of modeled aponeurotomized EDL muscles. (a) Distal active and passive isometric length-force curves of aponeurotomized muscles (b) Proximal active and passive isometric length-force curves of aponeurotomized muscles. All force data are normalized for optimal distal force of Case P.

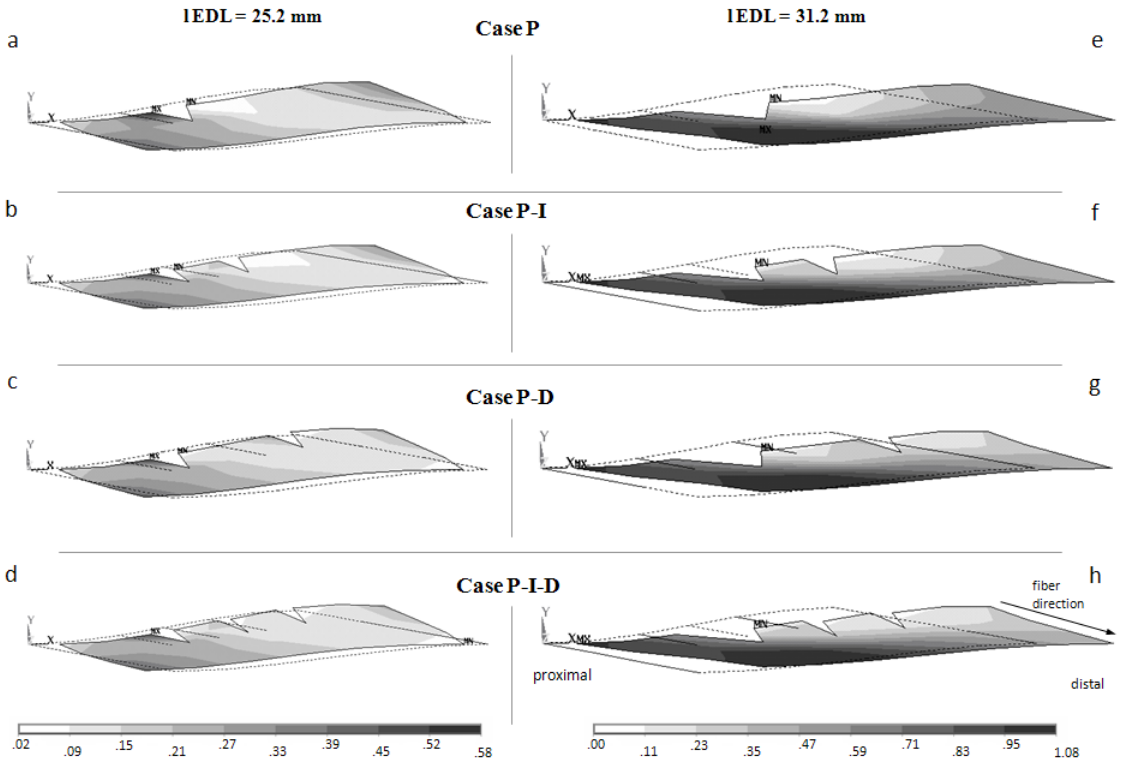

Figure 3.4 Fiber direction stresses within modeled aponeurotomized muscles. Contour plots with stress distributions within the fiber mesh of active aponeurotomized muscles: (left panel: a-d) for low muscle length (i.e. 25.2 mm) and, (right panel: e-h) for high muscle length (i.e. 31.2 mm).

value of fiber stress equaled 0.83, 0.82, 0.82 and 0.82 for Cases P, P-I, P-D and P-I-D respectively.

We conclude that the decrease in fiber direction stress in the distal population does explain the reduction in active muscle force for the multiple aponeurotomy cases, which for the proximal muscle fiber population remains quite limited because an increased number of interventions cause no notable changes in the stress within this population.

3.3.1 Changes in Length Range of Force Exertion

The effects of multiple interventions on determinants of muscle length range of distal active force exertion were also minor. A detailed analysis (not shown in Figure 3.3) shows that compared to Case P, even for Case P-I-D muscle length range

of distal active force exertion was limited to only 0.2% (distal optimum length of aponeurotomized muscle shifted to a higher length by 0.075 mm, i.e., by only 0.25%; and the shift in distal muscle active slack length to a higher length was only 0.06 mm).

For length range of active force exerted proximally; compared to Case P, Case P-I-D caused a somewhat higher but still limited effect: a decrease in muscle length range of force exertion was found (by 1.9%, caused by a shift of proximal optimum length to a higher length by 0.025 mm, i.e. by only 0.08%; and a shift of muscle active slack length to higher length by 0.17 mm).

3.3.2 Muscle Passive Forces

No notable change in passive force was shown with increased numbers of interventions.

We conclude that the effects of multiple aponeurotomy on muscle active and passive length-force characteristics remain quite limited.

3.4 Effects of Extramuscular Myofascial Force Transmission on Muscular Mechanics

A major effect of extramuscular myofascial force transmission on muscle force is a sizable proximo-distal force difference. This was also found in the present study in which we studied distal lengthening of the muscle: distal active as well as passive forces of all aponeurotomized muscles were higher than proximal forces (Figures 3.3 and 3.5).

The proximo-distal force difference always being positive is indicative for a net proximally directed myofascial load on the aponeurotomized muscle. Note that Figure 3.5 shows almost identical values for such loads, regardless of the number and location

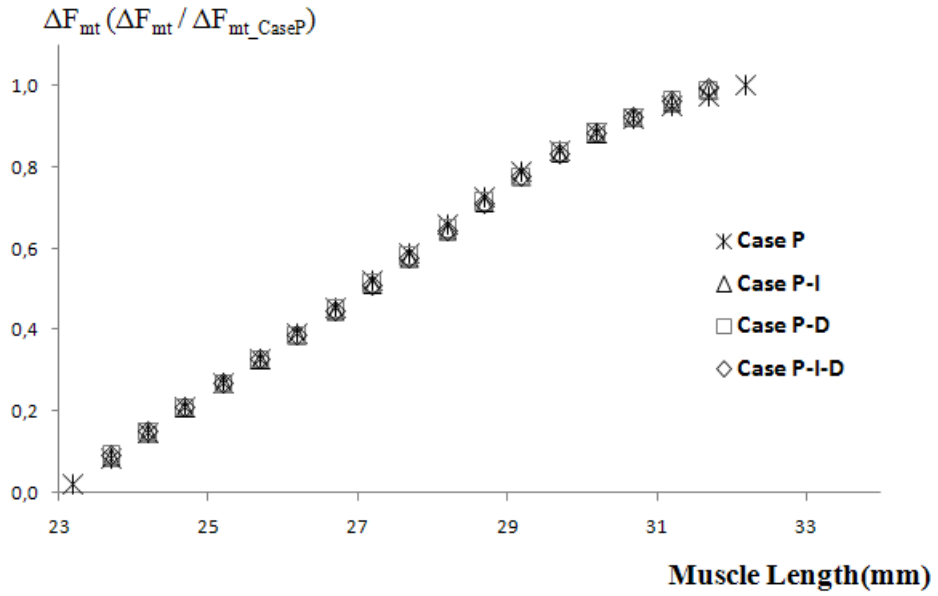

Figure 3.5 The proximo-distal total force differences for modeled aponeurotomized muscles. Total (ΔF_{mt}) proximo-distal force differences (calculated as Fdist-Fprox) of muscles with aponeurotomy modeled at single or multiple locations. All data is normalized for such force difference of Case P-I-D, encountered at muscle length = 31.7 mm.

variations of the additional interventions.

Moreover, the distal passive forces attain nonzero values at considerably lower muscle lengths ($\approx$ 3.0 mm) than proximal forces. Such an effect is also found for active forces, but to much more limited extent ($\approx$ 0.5 mm). This indicates that the proximal ends of the muscle fibers remain slack even at higher muscle lengths, because the net proximally directed load prevents any force to be transmitted to the most proximal ends of muscle fibers and muscular extracellular matrix, until the muscle is lengthened more. In a detailed analysis (not shown in Figure 3.5), the forces in the linking elements representing extramuscular connections are individually examined. This analysis shows that for the different cases of aponeurotomy studied; also the distribution of the extramuscular myofascial loads remains similar.

Therefore, we conclude that the extramuscular myofascial loads were not affected much by the number of aponeurotomies.

4. DISCUSSION

4.1 Additional Interventions Do Not Improve the Intended Effects of Aponeurotomy Acutely

4.1.1 Lengthening of the Target Muscle

Hypertonia is a common symptom (e.g. in cerebral palsy), leading to muscle contractures that restrict joint motion. Therefore, a major goal of surgical aponeurotomy is to increase muscle length range of force exertion. A change in muscle length range of force exertion occurs due to a shift in i) muscle optimum length and ii) muscle active slack length. In intact muscle, it was shown experimentally [16,21] and using finite element modeling [23] that the former is ascribable to heterogeneity in lengths of sarcomeres within muscle; primarily to increased parallel distribution of sarcomere lengths. Such substantial parallel distribution of sarcomere lengths shown to occur also within aponeurotomized muscle is chiefly responsible with the muscle lengthening effect aimed at [23,24]. On the other hand, the latter is determined by the last sarcomere that manages to overcome opposing internal forces within the fiber and muscle and exert force on the outside world. It was shown previously that aponeurotomy causes at lower muscle lengths generally shorter sarcomeres than in intact muscle leading to a shift in muscle active slack length to a higher muscle length [23]. Therefore, for the intended effect of muscle lengthening to take place, a greater shift in muscle optimum length to a higher length than such shift in muscle active slack length needs to occur. For aponeurotomy performed at a single location, this was shown to occur, leading to a major muscle lengthening for both isolated [16,23] and extramuscularly connected muscle [24].

Our present results showed that compared to the lengthening effect of single aponeurotomy case studied presently, the effects of multiple intervention cases on muscle length range of force exertion are differential for the distal and proximal joints.

Distally For the single aponeurotomy case, our recent results [24] showed that compared to intact muscle, the increase in muscle length range of force exertion was substantial (by 46%). In contrast, the additional muscle lengthening effect was shown presently to be very limited for the multiple intervention cases (e.g., for Case P-I-D increase in length range is only 46.01% was found). This is due to similar but minor shifts in muscle active slack and optimum lengths to higher lengths encountered for the multiple aponeurotomy cases studied.

Proximally Multiple aponeurotomy cases caused a narrowing of muscle length range of force exertion: compared to intact muscle [24] single aponeurotomy case yielded an increase of 13% in muscle length range whereas, e.g., for Case P-I-D such increase was less (by 11%). This is due to a further shift in muscle active slack length to a higher length while muscle optimum length was minor.

Our present results showed that multiple aponeurotomy cases do not lead to a more pronounced parallel distribution of sarcomere lengths which effect is ascribable to the identical distal and proximal populations of muscle fibers for all cases studied. This explains both for the distal and proximal ends of the muscle, the highly limited alterations in muscle optimum length. On the other hand, additional modeling (results are not presented) shows that at very low muscle lengths the sarcomeres within aponeurotomized muscle are getting shorter as the number of interventions increase. Therefore, as all the sarcomeres in multiple aponeurotomized muscles are too short to exert any force (i.e. at active slack length), still some sarcomeres in single aponeurotomized muscle are able to produce force. Consequently, the further shift in muscle active slack length to a higher length proximally is explained by increased shortening of sarcomeres with multiple aponeurotomies at very low muscle lengths.

4.1.2 Force Decrease of the Target Muscle

The contractures or unusual positions of a joint may also be caused by the imbalance of antagonistic muscles. Weakening of the agonist muscle (i.e. muscle force

reduction) might increase the relative effect of antagonistic one in such a condition. Therefore, aponeurotomy is indicated as a clinical means of such weakening of muscle (e.g.[18]). Force reduction acutely after aponeurotomy was reported to occur due to: 1) rupture in the intramuscular connective tissues leading discontinuity in intramuscular myofascial force transmission, and 2) substantial shortening of sarcomeres in the distal population of muscle fibers [16,20,23,24]. Previous experimental studies on isolated muscle showed that acutely after aponeurotomy, the intramuscular connective tissue ruptures and the muscle force drops substantially [20]. Moreover, in our previous modeling study of aponeurotomy on isolated muscle [23]; cutting the aponeurosis but leaving the intramuscular tissues intact caused the force reductions remain very limited. In short, mechanical mechanism of aponeurotomy was shown to be dominated by intramuscular myofascial force transmission, rather than the myotendinous force transmission [16,20,23]. As being another major reason; the muscle isometric active force reduction was shown to correlate positively with the muscle fiber shortening in the distal population in previous experiments [16]. Moreover, the substantial shortening of these sarcomeres and pronounced drop in their stress values are shown and quantified in both isolated [23] and extramuscularly connected [24] aponeurotomized muscles by previous modeling studies.

Our present results show that as the number of aponeurotomies increase, both distally and proximally exerted muscle forces become lower at any muscle length. However; even though compared to the intact muscle the muscle force reductions for single aponeurotomy case are substantial, the further force reductions remain very limited after multiple interventions. The greatest reduction among all conditions is obtained with triple aponeurotomy case and at optimal force; but even such reduction is very limited: (1) distal optimal forces of Case P and P-I-D are 68% and 64% respectively of that of intact muscle, (2) proximal optimal forces of Case P and P-I-D are 60% and 55% of that of intact muscle.

In the present study; in order to simulate multiple aponeurotomies accurately, intramuscular connective tissue rupture is modeled for any intervention which results in discontinuities throughout intramuscular myofascial force transmission. Further muscle

force reduction at both proximal and distal joints with additional interventions may be explained primarily by these disruptions in intramuscular myofascial force transmission. Moreover; further shortening in sarcomeres within distal population of muscle fibers, accordingly drop in their stress values shown in the present results is another major reason for the contribution of multiple aponeurotomies to the muscle force reduction.

However, in spite of all those effects reported, the muscle force reductions after multiple aponeurotomies are limited compared to the effect of single intervention condition presently studied. The primary explanation to this limited enhancement in force reduction is the identical distal populations of muscle fibers for all cases studied. Regardless of the number of aponeurotomies, the population of much shortened sarcomeres which accordingly are able to exert very low active forces remained at the same size. It should be noted that active forces are predominantly generated by the proximal population of muscle fibers in all aponeurotomy conditions: average stress values of distal population muscle fibers are 51%,41%,40% and 36% of that of proximal population of muscle fibers for Cases P, P-I, P-D and P-I-D respectively.

Furthermore, our present results show that the effects of additional interventions on sarcomere lengths are differential for distal and proximal population of muscle fibers, even though these populations remain the same among aponeurotomy conditions. While the average shortening of sarcomeres at the distal population is getting pronounced with increasing number of interventions, the distribution and mean value of sarcomere lengths at proximal population are not affected from multiple aponeurotomies.

In conclusion; since the dominant force is generated by the proximal population but the main effects of additional interventions (disruption of intramuscular myofascial force transmission and further shortening of sarcomeres) are observed at the distal population of muscle fibers, the further force decrease by multiple aponeurotomies remained quite limited compared to the force reduction effect of single intervention case studied.

4.2 Clinical Implications and Limitations of the Study

4.2.1 Location of the intervention closest to the tendon is major determinant:

In our previous modeling study [25], the intended effects of aponeurotomy operation was shown to vary substantially with the location of intervention, and an intervention closer to the tendon was shown to enhance these effects considerably. Major determinants of such enhancements were explained as increased parallel distribution of sarcomeres, increase in size of distal population of muscle fibers, increased gap length and decreased active fiber stress. Also the proximo-distal force difference became more pronounced.

In multiple aponeurotomy conditions, the size of population of muscle fibers without myotendinous connection to either origin or insertion is determined by the location of intervention closest to the tendon. In order to overrule the effects of the size of distal population of muscle fibers which is an important determinant of the intended effects, the location of the intervention closest to the tendon (Location P) was kept presently the same in all conditions. So that the comparison of multiple aponeurotomy conditions to the single aponeurotomy case (Case P) becomes reasonable.

The present results showed that in addition to the size of the distal population, most of the determinants of the intended effects of aponeurotomy, such as parallel sarcomere length distributions, active fiber stress drop and effects of extramuscular myofascial force transmission are not altered substantially with multiple aponeurotomies as long as the location of most proximal intervention remains the same. It should also be noted that; even though multiple aponeurotomy conditions cause small changes in length-force characteristics compared to single intervention case, the differences among the effects of these multiple aponeurotomy cases are even negligible. Thus, an important conclusion is that the differences in locations and numbers of the interventions on the disconnected part of aponeurosis do not cause any alterations.

Our present model results further confirm the conclusion that the location of the intervention closest to the tendon is a major determinant of the impact of the intended effects of aponeurotomy.

4.2.2 Greater Gap Length in Multiple Aponeurotomy Cases May Be Favorable After Recovery

Previous experimental studies [16] on rat muscle showed that after aponeurotomy, the intramuscular connective tissue ruptures progressively in the muscle fiber direction and a gap between distal and proximal populations of muscles' fibers appears. In experimental as well as modeling studies [23,24] the gap length was shown to increase as the muscle is lengthened. In a recent study on rat muscle [22], the cut ends of the aponeurosis were shown to be reconnected by newly developed connective tissue consisting of thin collagen fibers which are densely packed and mostly arranged in the aponeurosis direction. This new aponeurotic tisue was shown to be more compliant than the original one and that the recovered aponeurosis was longer than its length before the intervention.

Our present results show that each intervention causes a separate gap between the cut ends of the aponeurosis which is a function of muscle length and the total gap length becomes higher as the number of aponeurotomies increase. Even though these geometrical changes do not have substantial effects on muscle mechanics acutely, the length of gap might be an important determinant of the potential lengthening effect provided by aponeurotomy in the healing period: higher gap length may be an indicator of greater quantities of new aponeurotic tissue after recovery which brings compliance to the aponeurosis and most probably to the muscle as well.

Moreover, the present results show that the length of the main gap which occurs at the intervention closest to the tendon is substantially higher than that of the other gaps. Note that a decrease in the length of the main gap is obtained as the number of interventions increase. Since we assume that all these gaps will be connected by

new aponeurotomic tissues, the distribution of new connective tissues and reducing the dominance of one gap may also be favorable for after healing. On the other hand, it should also be noted that an increase in number of incisions and gap length might cause the surgery becoming more invasive and make the healing more difficult.

As the recurrence of the limited joint range of motion is highly observed after operation (e.g.[18]), the mechanisms active during recovery period should be analyzed and our suggestions above should be tested with additional studies.

4.2.3 Limitations of the Study:

After aponeurotomy, the rupturing of the connective tissue occurs progressively with the muscle lengthening (e.g. [20]) However, in our model the depth of the rupture is kept constant independent the muscle length. Therefore, the progressive nature of rupturing is not represented. Anyhow, the depth of the tear was shown to be stabilized afterwards [16] and the modeling study is assumed to represent this stabilized depth. Moreover, the depths of the tears in all interventions were modeled as the same. There is no experimental study investigating multiple aponeurotomy conditions; thus, in spite of our assumption the depths might differ among the intramuscular ruptures in real muscle.

Another limitation is that the mechanical properties of the modeled EDL muscle and its extramuscular connections represent those of healthy muscle. Some differences were suggested to exist between spastic and healthy muscle mechanics [34]. The spastic muscles are reported to be stiffer than that of healthy muscle both in muscular an [36]. These differences might have important effects; however the basic principles of mechanical mechanism of the intervention would remain the same.

In conclusion, our modeling results show that; regardless of the numbers of aponeurotomies, size of the distal population of muscle fibers and resultantly the dominant effects on muscle mechanics are determined by the location of the interven-

tion closest to the tendon. With additional interventions on the disconnected part of aponeurosis, the substantial shortening of the sarcomeres in the distal population of muscle fibers is gone even further especially at higher muscle lengths and resultantly the active stresses generated by these fibers decreased. The further force decrease obtained at both proximal and distal tendon is in accordance with this result. However, in spite of the geometrical differences created by additional interventions, the results showing the application of multiple interventions not bringing great improvements to the intended acute mechanical effects of aponeurotomy surgery should be taken into account.

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