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# Healing of the aponeurosis during recovery from aponeurotomy: morphological and histological adaptation and related changes in mechanical properties

R.T. Jaspers<sup>a,\*</sup>, R. Brunner<sup>b</sup>, U.N. Riede<sup>c</sup>, P.A. Huijijng<sup>a,d</sup>

<sup>a</sup> *Instituut voor Fundamentele en Klinische Bewegingswetenschappen, Faculteit Bewegingswetenschappen, Vrije Universiteit, van der Boechorststraat 9, 1081 BT Amsterdam, The Netherlands*

<sup>b</sup> *Pediatric Orthopaedic Department, Children's Hospital, University of Basle, Switzerland*

<sup>c</sup> *Pathologisches Institut, Universität Freiburg, Germany*

<sup>d</sup> *Integrated Biomedical Engineering for Restoration of Human Function, Biomedisch Technologisch Instituut, Universiteit Twente, Enschede, The Netherlands*

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## Abstract

Aponeurotomy, which is the transection of an aponeurosis perpendicular to its length, is performed to lengthen spastic and/or short muscles. During recovery, the cut ends of the aponeurosis are reconnected by new connective tissue bridging both ends. The aim of this study is to investigate the histological features of this new connective tissue as well as its mechanical properties after recovery from aponeurotomy. For this purpose, aponeurotomy was performed on the proximal aponeurosis of rat m. gastrocnemius medialis (GM), which was followed by six weeks of recovery. The lengths of aponeurotic tissues were measured as a function of active muscle length. The results are compared to a control group as well as to the acute effects and a sham operated group.

Activation of the muscle at increasing lengths after aponeurotomy caused a gap between the cut ends of the aponeurosis. However, after recovery, new connective tissue is formed bridging the aponeurotic ends, consisting of thin collagen fibres, which are densely packed and generally arranged in the direction of the aponeurosis. The number of fibroblasts was three to five times higher than that of aponeurotic tissue of the intact parts as well as that of the acute and sham operated muscles. The strain of the new connective tissue as a function of active muscle length was shown to be about three times higher than that of the aponeurosis. It is concluded that the inserted new aponeurotic tissue is more compliant and that the aponeurosis becomes 10–15% longer than in untreated muscle. As a consequence, the muscle fibres located distally to the new aponeurotic tissue will become shorter than prior to aponeurotomy. This explains a shift of the length–force curve, which favours the restoration of the range of joint motion.

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**Keywords:** Aponeurotomy; Connective tissue; Adaptation; Aponeurosis; m. gastrocnemius; Rat

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## Introduction

In spastic movement disorders such as cerebral palsy, certain affected muscles are overly active and remain functionally and later even structurally short with re-

spect to their antagonists. As a consequence, the joint range of motion is limited and motor function may be impeded (e.g. [30]). In case of extremely short muscles, the joint range of motion is restored clinically by performing surgical interventions onto the muscles. Surgical interventions are performed either outside the muscle belly on the tendon (tendon lengthening; see for review [22]) or at the muscle belly by cutting the

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\* Corresponding author. Tel.: +31 20 4448463; fax: +31 20 4448529.  
E-mail address: r\_jaspers@fbw.vu.nl (R.T. Jaspers).

aponeurosis (aponeurotomy; [2,17]). On the short term, both interventions have been shown to be effective in the restoration of the joint range of motion (e.g. [1,9,23,29]). However, on the long-term some patients (4–50%) show recurrence of a limited range of joint motion (e.g. [19,21,23]). For experimental lengthening of the cat achilles tendon it was shown that after long-term recovery, optimum muscle force and range of active force exertion of the m. triceps surae were substantially reduced [26]. This adaptation explains the recurrences of a restricted joint range of motion reported. The mechanisms underlying the success or failure of aponeurotomy are poorly understood. Experimental aponeurotomy on rat m. gastrocnemius (GM) and m. extensor digitorum longus (EDL) has shown that activation of the muscle acutely after the proximal intervention results in a spontaneous rupturing of the intramuscular connective tissue along the direction of the muscle fibres. This is accompanied by a reduction in optimal muscle force of about 50% and a 10% increase in the muscle length range of active force exertion [12,13]. These acute effects of aponeurotomy are favourable for the restoration of the joint range of motion. However, investigation of the muscle length–force characteristics after six weeks of recovery showed that both parts of the proximal aponeurosis were reconnected and that muscle optimal force and the length range of active force exertion were restored to at least the pre-aponeurotomy values [4].

Optimisation of aponeurotomy and post-operative treatment requires insight in how the muscle adapts during the phase of recovery. It is hypothesised that both parts of the aponeurosis are reconnected by new aponeurotic tissue bridging their ends, which is more compliant than the original, intact aponeurotic tissue. The aim of this study is to investigate the morphological and histological changes of the muscle and in particular those of the aponeurosis. In addition, these morphological changes are related to the changes in the mechanical properties of the connective tissue of the proximal aponeurosis.

## Methods

### *Animals and experimental protocol*

All surgical and experimental procedures were performed in strict agreement with Swiss law and regulations concerning animal welfare. Experiments were performed on the right m. gastrocnemius (GM) of male Wistar rats.

Four experimental groups of animals are distinguished.

### *Control*

Length–force characteristics were measured of the right, dissected in situ GM of 10 animals (mean age 14.9 weeks, body mass  $357.1 \pm 11.3$  g (mean  $\pm$  S.D.)). This group of rats will be referred to as “control” group.

### *Surgery*

- (1) Aponeurotomy was performed on the proximal aponeurosis of rat GM ( $n = 6$ , mean age 9.1 weeks, body mass  $373.3 \pm 17.7$  g (mean  $\pm$  S.D.)). The proximal aponeurosis of the right leg was cut at its middle perpendicular to its length using a scalpel (blade number 23) as well as the proximal aponeuroses of all remaining parts of the right triceps surae. The location of the transection of the aponeurosis was marked by silk 0000 sutures at each side of the aponeurotic transection. Following surgery, a lower leg-walking cast was applied to the right leg to maintain maximal dorsiflexion for three days. After six weeks of recovery, length–force characteristics were determined of the dissected in situ GM. This group of animals will be referred to as “AT-recovery”.
- (2) A sham operation was performed on the right leg of the rat ( $n = 6$ , mean age 9.1 weeks, body mass  $383.8 \pm 20.5$  g (mean  $\pm$  S.D.)). This implies opening of the skin and performing blunt dissection of the connective tissue surrounding the muscle, similar as done for the “AT-recovery” group. The middle of the aponeurosis was marked with silk 0000 sutures, but the aponeurosis was not cut. Subsequently a lower leg-walking cast was applied to the right leg to maintain maximal dorsiflexion for three days. After six weeks of recovery, length–force characteristics were determined of the dissected in situ GM. This group of animals will be referred to as “sham”.
- (3) Aponeurotomy was performed on the proximal aponeurosis of the right, dissected in situ GM. Subsequently, the muscle was stimulated at increasing lengths (acute trajectory). During this procedure the intramuscular connective tissue below location of the cut ruptured spontaneously along the direction of the muscle fibres. After this, the muscle was a stable entity again and length–force characteristics were determined. This group of animals will be referred to as “AT-acute”. After the initial measurements of length–force characteristics, for this experimental group we used the animals of the control group (see above).

Surgery, application and removal of casts for the AT-recovery and sham group were performed under general anaesthesia (Halothane 0.5–2%, head mask). For a post-operative period of three days the animals were kept under paracetamol (200 mg/kg per day) as analgetic. The animals were housed under standard conditions. For all groups (i.e. control and surgery), the anaesthetics during the determination of the length–force curves was similar (see below).

### *Measurement of length–force curves*

Animals were anaesthetised by intraperitoneal injection of sodium pentobarbitone (initial dose 8 mg/100 g body mass) and ventilated mechanically if needed. The medial head of GM of the right leg was dissected, leaving its origin intact. The blood supply was left undisturbed whereas the sciatic nerve was cut as proximally as possible. The achilles tendon with a piece of calcaneal bone still attached was connected by a metal wire to a force transducer (A&D Company LC-4101). Small copper markers were inserted into the muscle to mark the origin of the muscle, the distal end of the proximal aponeurosis and the distal end of the distal fibre (Fig. 1). It should be noted that this set-up, with a dissected in situ muscle, any effects of myofascial force transmission between neighbouring muscles and extramuscular connective tissue are excluded (cf. [11]).

The animal was positioned in the experimental set-up by rigidly clamping the femur. Ambient temperature was controlled at 27 °C. The proximal end of the nerve was stimulated supramaximally (100 Hz, 0.15 ms square wave pulse) using silver electrodes connected to a constant current source. Tetanic isometric contractions of 600 ms were induced at a series of muscle lengths, beginning near active slack length and ending at the length at which force was about 90% of maximum force (1 mm increments). Each tetanic contraction was preceded by two twitches with a 1 s interval to allow adjustment of the muscle to that length. Three seconds after the last twitch, a tetanic contraction was evoked. The interval between two subsequent tetanic contractions was 2 min, during which the muscle was allowed to recover at short length. The muscle was photographed (Cannon A1 camera, macrolens, exposure time 1/125 s, 400 ASA slide film) in passive state (2 s before activation) and in fully active state (200 ms after evoking

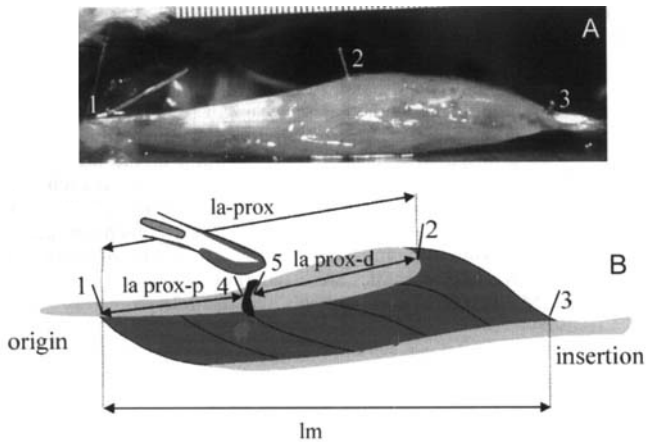


Fig. 1. Dissected in situ GM and aponeurotomy and its muscle geometry in the mid-longitudinal plane. (A) Dorsolateral view of the active muscle, near optimum length before aponeurotomy. Small copper markers inserted into the muscle mark: (1) the origin of the muscle, (2) the distal end of the proximal aponeurosis and (3) the distal end of the distal muscle fibre. (B) Schematic drawing of the aponeurotomy and the definition of muscle and aponeurosis parameters. For the intact muscle before aponeurotomy, as well as for sham and control muscles, the length of the proximal aponeurosis ( $la\text{-prox}$ ) was determined by measurement of the distance between markers 1 and 2. The distance between markers 1 and 3 yields active muscle length ( $lma$ ). While the muscle was positioned at about optimum length, aponeurotomy was performed by transection of the proximal aponeurosis approximately at its middle. During the physiological experiments after the recovery, two markers (4 and 5) were placed at the location of the sutures and the lengths of both proximal and distal aponeurotic parts were measured ( $la\text{ prox-p}$  and  $la\text{ prox-d}$ ) by determination of the distance between markers 1 and 4 and markers 3 and 5, respectively. The distance between markers 4 and 5 provided the length of the newly aponeurotic tissue.

tetanic stimulation). A microcomputer was used to collect all force data, using an AD converter (Validyne Engineering Corp. UPC601-G, sampling frequency of 1000Hz and resolution of force 0.0071N).

#### Treatment of data

##### Muscle force

Data relating passive force to muscle length were least square fitted using an exponential function. Active muscle force ( $F_{ma}$ ) was calculated by subtracting passive force from total muscle force for the appropriate muscle lengths. The relationship of active muscle force with muscle length was fitted by a polynomial function. The order of the polynomial most adequately describing the relationship was selected (see section statistics). Muscle optimum length ( $l_{mao}$ ) was defined as that length at which the maximum of the selected polynomial curve describing active muscle force was encountered within the muscle length range of the experiment.

##### Lengths of muscle and aponeurosis

Post-experimentally, morphological parameters were estimated by determination of the marker positions on projected photographic images (magnification 15 $\times$ ) using a digitiser tablet (Microgrid III Summagraphics Co., mean error 0.017mm) and a software program (Autocad 12.0). Muscle length ( $l_m$ ) and the length of the proximal aponeurosis ( $la\text{-prox}$ ) were determined for all muscles. In addition, for the AT-recovery muscles, the lengths of the proximal and distal aponeurotic parts were determined as well as the length of the new

aponeurotic tissue. The relationships between the lengths of aponeurotic tissue and active muscle length were fitted with a third-order polynomial.

##### Histology

After the experiments, the muscles were fixed (4% (v/v) formaldehyde, 15% (v/v) alcohol and 1.5g/l thymol) for at least two weeks at room temperature. After this, the muscles tissue was routinely processed, embedded in paraffin and longitudinal sections (4 $\mu$ m thick) of the mid-longitudinal plane were cut and collected on glass slides. Subsequently, sections were stained with hematoxylin and eosin (HE), dehydrated, and mounted in Entellan (Merck). The morphology and histology of the muscle sections was examined with a microscope (AxioskopH, Zeiss), using bright-field illumination. Images were obtained with different objectives ( $\times 5$ ,  $\times 10$  and  $\times 20$ ) and a CCD camera (AxoCam MRc, Zeiss) connected to a frame grabber (MRGrab 1.0).

##### Statistics

To select the polynomial most adequately describing the length-force data, the fitting was started with a first order and the power was increased up to a fifth order. One way ANOVA was performed to select the highest polynomial order that yielded a significant improvement of the length-force data.

Two-way ANOVA was performed to test for the effects of the factor "type of intervention" (between groups) and the factor "muscle length" (repeated measures) on the length changes of segments of the proximal aponeurosis. Post hoc tests were performed using the Bonferroni procedure to locate significant differences ( $P < 0.05$ ).

## Results

### Macroscopic changes after aponeurotomy

During stimulation of the muscle at increasing muscle lengths, a gap developed between the proximal and distal part of the proximal aponeurosis (Fig. 2A). At

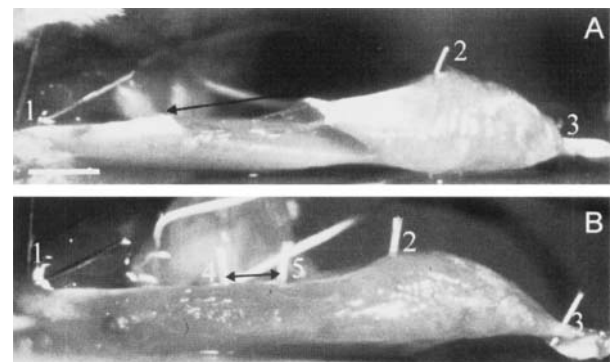


Fig. 2. Muscle morphology after recovery from aponeurotomy. (A) Active muscle at about optimum length acutely after aponeurotomy. After aponeurotomy, during activation of the muscle at high lengths, a gap (arrow) formed between the cut aponeurosis ends. In fact, the intramuscular connective tissue ruptured along the muscle fibres, to deep within the muscle, below the location of the aponeurotomy. The numbers indicate the markers during the physiological experiments (see Fig. 1). (B) Active muscle after recovery from aponeurotomy. During the physiological experiments after the recovery, two markers (4 and 5) were placed at the location of the sutures and the lengths of both proximal and distal aponeurotic parts. The aponeurotic segments are reconnected by newly formed connective tissue. The length of the new aponeurotic tissue is indicated by an arrow. The scale bar indicates 5mm.

muscle optimum length this gap was  $6.3 \pm 0.6$  mm (mean  $\pm$  S.D.). This situation represents the initial condition of the recovery phase. After recovery, both parts of the proximal aponeurosis were reconnected (Fig. 2B). After aponeurotomy and subsequent recovery, longitudinal sections of the muscle show the presence of scar tissue at the location of the gap. In fact, the gap within the aponeurosis is bridged by new aponeurotic tissue over a length of  $3.2 \pm 1.0$  mm (mean  $\pm$  S.D.) at muscle optimum length (Fig. 3A and B). The variation in the length of the new connective tissue was high as the coefficient of variation was 30.5%. The original aponeurotic tissue located proximally and distally to the new tissue exhibits “striation” (probably caused by crimp due to a regular sinusoidal wave pattern of collagen fibres). This is a typical feature of uninjured collagen fibres of tendons and ligaments (Fig. 3B and C). In contrast, the new aponeurotic tissue shows collagen fibres, which are aligned in the direction of the aponeurosis but does not show any crimping (Fig. 3C).

#### Detailed histological changes after aponeurotomy

Microscopic observation of the HE stained longitudinal sections of the different groups revealed that the

proximal aponeurosis as well as the distal muscle fibres in the sham operated muscles and AT-recovery groups were covered with a layer of collagen fibres, containing a high concentration of fibroblasts (Fig. 4). This layer was not observed on the AT-acute muscles, which indicates that the formation of this layer is caused by adaptation of the epimysium and/or the fascia surrounding the muscles in response to dissection of the muscle rather than by the aponeurotomy per se.

Comparison of the original aponeurotic parts of the AT-recovery muscles with that of the aponeuroses of the sham and AT-acute groups shows a high histological similarity between these structures. The aponeurosis consists of thick collagen bundles generally oriented in the longitudinal direction of the aponeurosis (Fig. 4B). In all three groups, the collagen contained spindle-shaped fibroblasts with nuclei that are also oriented in the longitudinal direction of the aponeurosis. The concentration of nuclei is similar for the three groups.

In contrast to the original aponeurotic parts for the AT-recovery muscles, the new tissue bridging the aponeurotic ends consisted of densely packed, thin collagen fibres (Fig. 4B vs. C). The concentration of nuclei was three to five times higher than for the original connective tissue of the proximal aponeurosis, indicating substantial

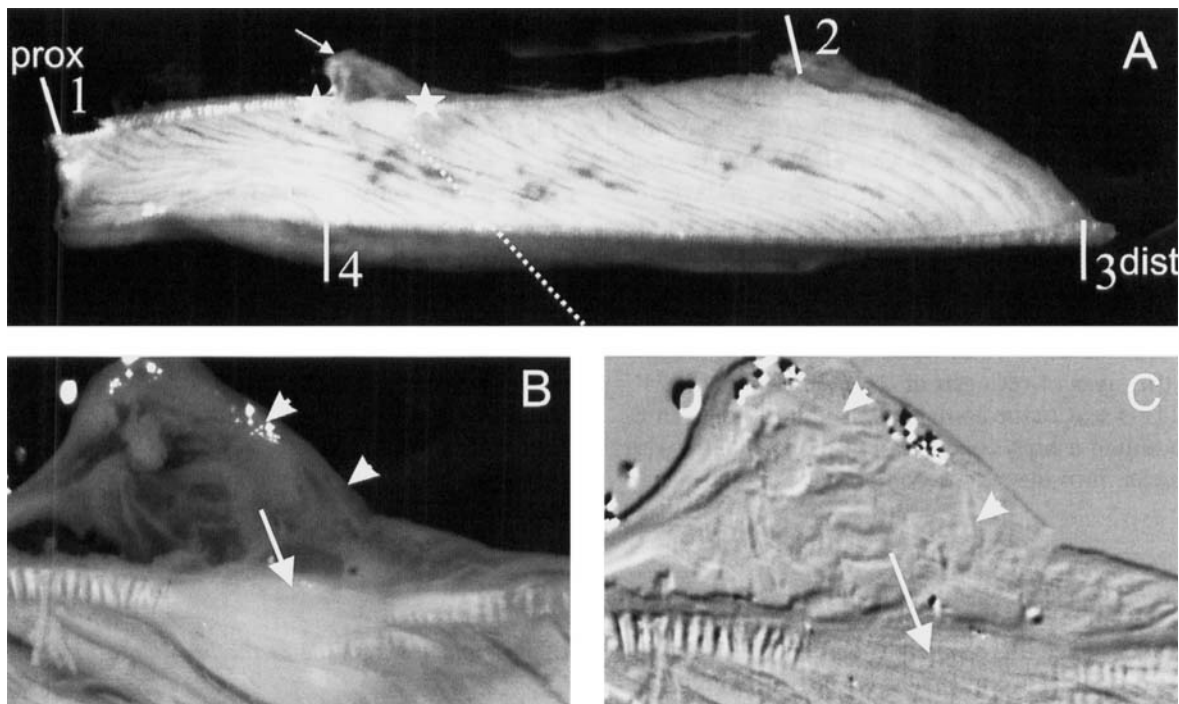


Fig. 3. The muscle and the proximal aponeurosis after recovery from aponeurotomy. (A) Photograph of the mid-longitudinal plane of a whole muscle. Marker 1 and 2 indicate the most proximal and distal ends of the proximal aponeurosis respectively. Markers 3 and 4 indicate the most distal and proximal ends of the distal aponeurosis, respectively. The proximal and distal ends of the new connective tissue, which is filling the gap between the aponeurotic parts of the proximal aponeurosis are indicated by stars. (B) Magnified view of the area of the new aponeurotic tissue within the proximal aponeurosis. Note the crimp in the original aponeurotic tissue on each side of the gap, which is typical for mature collagen type I. Scar tissue is overlying the new aponeurotic tissue (solid arrow heads). (C) Image B after enhancement of the crimp and collagen fibres (embossing). The new aponeurotic tissue (indicated by an arrow) contains collagen fibres, which are aligned in the direction of the aponeurosis.



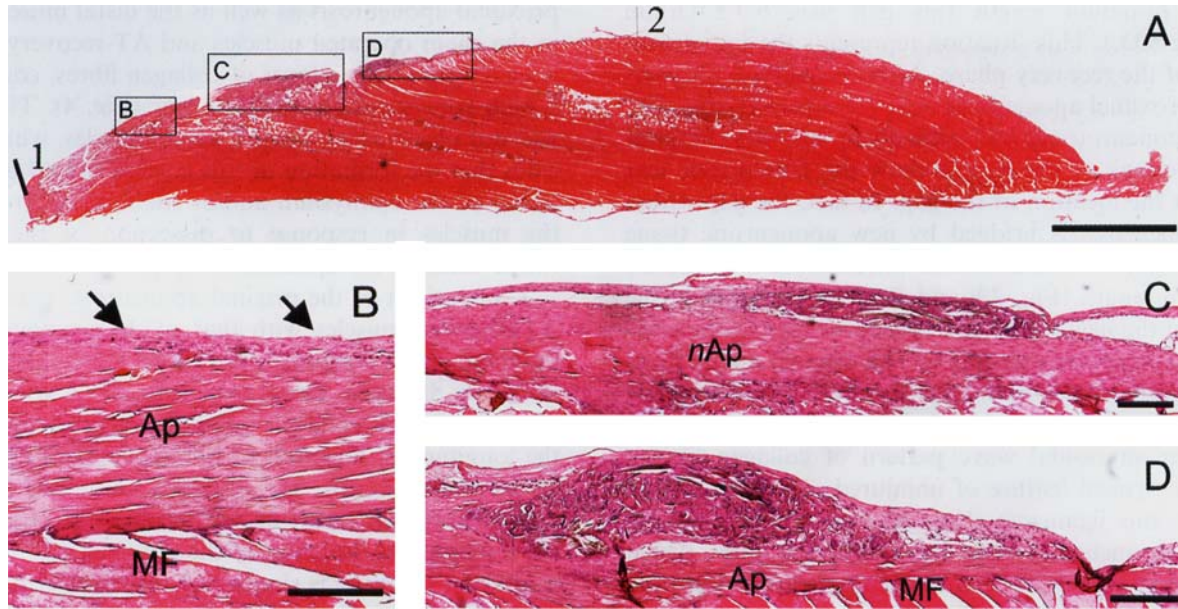


Fig. 4. Histology of muscle and aponeurosis after recovery from aponeurotomy. (A) Image of a section of the mid-longitudinal plane of the muscle after HE staining. The markers 1 and 2 indicate the proximal and distal ends of the proximal aponeurosis, respectively. The ends of the new aponeurotic tissue are marked by stars. Frames indicate areas enlarged in B, C and D. Scale bar: 3 mm. (B) Enlarged view of a part of the original aponeurosis, located proximally to the new tissue. On top there is a layer of connective tissue constituting the epimysium (arrow). Below this layer, the original aponeurotic tissue containing fibroblasts at an identical concentration as for the sham operated and control muscles. (C) Enlarged view of the area containing new aponeurotic tissue inserted in the previous gap. Note, the high concentration of spindle-shaped fibroblasts between the collagen fibres. (D) Enlargement of the scar lying on top of the newly formed collagen. This tissue shows vascularisation and innervation. Ap = original aponeurotic tissue, nAp = new aponeurotic tissue, MF = muscle fibres. Scale bars: B, C and D: 200  $\mu$ m.

proliferation of fibroblasts. In the upper layer of the new aponeurotic tissue, the collagen fibres are aligned in the longitudinal direction of the aponeurosis. The cells of this tissue contain spindle-shaped nuclei, aligned in the same direction as the collagen fibres. In the deeper layer of the aponeurotic tissue, fibroblasts with round nuclei are embedded between collagen fibres interweaving in various directions. In addition, at the location of aponeurotomy (i.e. on the dorsal aspect of the aponeurosis overlying new aponeurotic tissue), scar tissue is shown, which is covered by the layer of collagen of the epimysium (Fig. 4C and D). This scar tissue contained a mesh of collagen fibres containing a high concentration of fibroblasts with round nuclei. Into the scar, blood vessels and nerves had entered.

#### *Effects on the aponeurosis mechanical properties*

At all muscle lengths studied, the aponeuroses of the muscles exposed to aponeurotomy and recovery were significantly longer than in control and sham group (Fig. 5A). This is due to the new aponeurotic tissue added during the recovery process.

Fig. 5B and C show the effects of the sham operation and aponeurotomy on the length change of the old aponeurosis and new aponeurotic tissue. The sham operation did not change the properties of the aponeurosis

compared to that of the control muscle. Neither did aponeurotomy and subsequent recovery significantly alter the properties of the original parts of the aponeurosis (Fig. 5B and C). In contrast, the absolute length change of new connective tissue was substantially lower than that of the original parts of the aponeurosis, particularly at lower muscle lengths (Fig. 5B). Normalisation of the length change of the new aponeurotic tissue by its length (at optimum muscle length) showed that the normalised length change of aponeurotic segments was significantly different from that of the other groups (Fig. 5C). In fact, for the range of muscle lengths from 27 to 30 mm, the normalised length change was two to three times higher than for the original aponeurosis.

#### **Discussion**

##### *New aponeurotic tissue at the location of aponeurotomy*

The present results illustrate the process of healing of the aponeurosis after aponeurotomy within six weeks of recovery. To the best of our knowledge, recovery of lacerated or ruptured aponeurosis has not been reported previously. However, comparison of the flat new aponeurotic tissue and healed severed soft tissues such as round shaped tendons and ligaments for several species

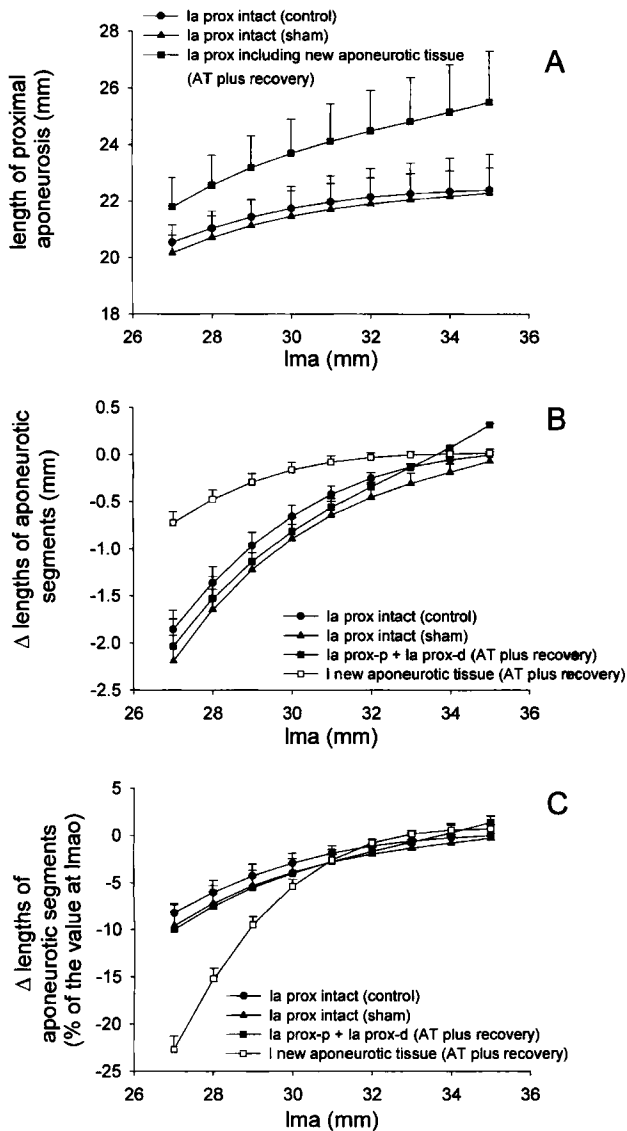


Fig. 5. The effects aponeurotomy and recovery on mechanical properties of aponeurosis segments. (A) The length of the proximal aponeurosis as a function of active muscle length. (B) The length change of the aponeurosis segments as a function of active muscle length, expressed as deviations from their value at optimum muscle length. Note the high length of the new aponeurotic tissue. (C) The change in length of the aponeurosis segments normalised for their lengths at optimum muscle length. All values are presented as means  $\pm$  S.E.M.

such as rat, rabbit and sheep shows a qualitative similarity. Therefore, it is concluded that recovery of flat, aponeurotic tissue occurs in a similar way as round shaped tendons or ligaments. In case of injury of tendons and ligaments, the gap between the ligament or tendon ends becomes filled initially with scar tissue, in which vascularisation and subsequently invasion of proliferating fibroblast are seen (e.g. [5,6,16,18]). After four days of recovery, collagen fibrils had been laid down in the scar in an irregular pattern and cells with round shaped nu-

clei were present between the collagen fibres. The compliance of this new tissue is reported to be about five times lower than that of the original tissue, which is associated with an increase in the collagen type III:type I ratio (e.g. [5,7,14]). As recovering tendons or ligaments are loaded, the newly formed collagen fibres become aligned with the direction of the force exerted on the whole structure, whereas the compliance of the new tissue is reduced [7,31]. In addition, the fibroblast nuclei become spindle-shaped and are aligned with the collagen fibres. These adaptations suggest that force exerted on the new collagen tissue may have enhanced the remodeling of the connective tissue. After aponeurotomy, force is exerted onto the new aponeurotic tissue, because also the muscle fibres, which are attached to the distal segment of the aponeurosis (i.e. distally to the location of aponeurotomy), contribute even acutely after aponeurotomy to total muscle force [4,13].

As the compliance of the new aponeurotic tissue was much higher than that of the original aponeurosis and muscle force after recovery was similar to that of intact muscles [4], it yields higher length changes. Long-term investigation (i.e. >6 weeks) of the healing of sheep and rabbit tendons and ligaments for up to two years after injury, showed that after about six months the histological appearance of new tissue resembles that of original tissues. However, the biomechanical properties remain inferior for at least two years [5,14]. Therefore, it is expected that the present situation is not an end point of the recovery and that the new aponeurotic tissue after aponeurotomy will change into mature, normal collagen aponeurotic tissue, albeit with a higher compliance than that of the original aponeurotic tissue.

#### Mechanisms affecting muscle function

Acutely after aponeurotomy, the distal muscle fibres are reported to become short due to the substantial gap appearing in the aponeurosis [12,13]. This causes a substantial increase in the distribution of the fibres mean sarcomere lengths [13], which is accompanied by an increase of both muscle active slack length and muscle optimum length (i.e. shift of the length–force curve to higher lengths).

During recovery from aponeurotomy, inserting of new connective tissue into this gap yields a longer and therefore more compliant aponeurosis compared to that in untreated muscle. This would be true even if the material inserted had the same material properties as the original aponeurosis. However, the new aponeurotic tissue itself is also more compliant than that of the regular aponeurosis (Fig. 5). Due to such increases in compliance, for any muscle length, the muscle fibres, which insert at the most distal segment of the proximal aponeurosis (i.e. distally from the new aponeurotic

tissue), are expected to remain active at lower lengths than the same fibres within an untreated muscle. After recovery from aponeurotomy, an increase in the muscle active slack length was found, similar to the situation acutely after aponeurotomy [12,13]. In addition, at the ascending limb of the length–force curve, active force was 20–80% lower than for untreated muscles at the identical muscle lengths, implicating a shift of this ascending limb to higher muscle lengths by about 5% [3]. Such a shift is in accordance with an enhanced distribution of the fibre mean sarcomere lengths.

Based on the finding that the distribution of the fibre mean sarcomere lengths correlates positively with the muscle length range of active force exertion [10,27], after recovery from aponeurotomy, an increased length range between optimum and active slack lengths (i.e. length range of active force exertion) was expected. However, this length range was reported not to be changed significantly [4]. It is concluded that additional mechanisms may be active (see for review [10,15]): (1) lower fibre lengths are associated with smaller fibre angles with the aponeurosis and muscle line of pull. Therefore, during recovery from aponeurotomy, a reduction in the fibre angles will contribute to a reduction in the muscle length range of active force exertion; (2) adaptation of the number of sarcomeres in series within muscle fibres or (3) changes in the stiffness of the distal aponeurosis may occur during the recovery.

Alternatively, the above discrepancy may be explained by the fact that in the study by Brunner et al. [4], tetanic force may have been measured over too limited a range of muscle lengths (e.g. if multiple optimum lengths would occur). Further investigation is required in order to resolve this issue (e.g. of possible changes in muscle geometry).

#### *Considerations regarding effects of the surgical operation*

In cerebral palsy, the range of motion at particular joints may be limited due to spastic control (causing overactivity) of muscles with functional and later structural shortness of antagonistic muscles of the movement. Our present results may help to improve understanding of the fundamental mechanisms that are manipulated during the operation. A limited range of joint motion due to spasticity can in principle be treated in two ways: (1) a reduction of force of the spastic antagonist muscle of the movement will allow the force of the agonist muscle of the movement to be more effective (e.g. [20,24]), and (2) in case of structural shortness, lengthening of those muscles and/or increasing their length range of active force exertion (e.g. [8,25]).

After six weeks of recovery, the effects of newly inserted aponeurotic tissue may involve aspects of both types of restoring a full range of joint motion: (1) the shift of the ascending limb of the length–force curve,

after recovery from aponeurotomy [3,4] implies a reduction of muscle force by 20–80% for the muscle lengths of the ascending limb [4]. Such weakening of spastic muscle will increase the relative strength of its antagonist muscle, allowing the spastic muscle to be stretched more; (2) inserting new, relatively compliant, aponeurotic tissue will increase muscle length and shift the length force relationship to higher lengths (see section discussion above), and thereby directly shifting the available length range of active muscle force exertion to a more preferable range of joint angles.

Any understanding of functional effects of aponeurotomy will fail if post-operative adaptations of collagenous and muscle tissue are not taken into account. Based on the finding that even after two years of healing of injured tendons and ligaments, the new collagenous tissue did not return to the material properties of uninjured tissue (see above, [5,14]), it is expected that the positive effects of the new aponeurotic insertions with a higher compliance may be lasting for several years. However, further adaptation of the connective tissue (e.g. increase in stiffness) is expected. In addition, the number of sarcomeres in series within muscles fibres may adapt in response to the new muscle fibre length range, as has been shown after joint immobilisation (e.g. [28]). Such adaptation is determined by post-operative conditions and treatments and may counteract the positive effects of the operation. Our present results indicate already a high variation in the length of the new aponeurotic tissue after six weeks of recovery. Variable clinical post-operative treatment is expected to further affect the functional outcome of the intervention. This may explain the reported variation in the effectiveness of muscle lengthening procedures and be reflected in the recurrence rate of 2–42% (e.g. [19,21,23]). Long-term evaluation of the effects of aponeurotomy, albeit in combination with different after-treatments is required to determine whether and how lengthening of the muscle is maintained.

In conclusion, the present study shows that during six weeks of recovery following proximal aponeurotomy of rat GM, new aponeurotic tissue is laid down within the transected aponeurosis. This new tissue has different histological characteristics (e.g. different collagen appearance), and has substantially higher compliance than that of the original aponeurosis. The presence of the new connective tissue may favour an enhanced range of joint motion, but long-term adaptations of the new aponeurotic tissue as well as of muscle tissue are expected to reduce these effects.

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