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Muscle lengthening surgery causes differential acute mechanical effects in both targeted and non-targeted synergistic muscles

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ABSTRACT

Epimuscular myofascial force transmission (EMFT) is a major determinant of muscle force exerted, as well as length range of force exertion. Therefore, EMFT is of importance in remedial surgery performed, e.g., in spastic paresis. We aimed to test the following hypotheses: (1) muscle lengthening surgery (involving preparatory dissection (PD) and subsequent proximal aponeurotomy (AT)) affects the target muscle force exerted at its distal and proximal tendons differentially, (2) forces of non-operated synergistic muscles are affected as well, (3) PD causes some of these effects.

In three conditions (control, post-PD, and post-AT exclusively on m. extensor digitorum longus (EDL)), forces exerted by rat anterior crural muscles were measured simultaneously. Our results confirm hypotheses (1–2), and hypothesis (3) in part: Reduction of EDL maximal force differed by location (i.e. 26.3% when tested distally and 44.5% when tested proximally). EDL length range of active force exertion increased only distally. Force reductions were shown also for non-operated tibialis anterior (by 11.9%), as well as for extensor hallucis longus (by 8.4%) muscles. In tibialis anterior only, part of the force reduction (4.9%) is attributable to PD. Due to EMFT, remedial surgery should be considered to have differential effects for targeted and non-targeted synergistic muscles.

1. Introduction

In remedial surgery, known under various names (muscle recession (Strayer, 1950; Rush et al., 2006), muscle release (Khot et al., 2008), muscle lengthening (Nene et al., 1993; Sutherland et al., 1997; Borton et al., 2001; Ma et al., 2006; Park et al., 2009), aponeurotomy (AT) (Baumann and Koch, 1989) involves cutting of an intramuscular aponeurosis transversely. Preparatory dissection (PD) is performed first to reach the target muscle (Saraph et al., 2000), then AT is used for correction of problems of range of movement and joint position in spastic paresis. The most important acute effect allowing lengthening of muscle is creation of a gap within the muscle. Enhancing the compromised joint range of motion is a primary goal (Baddar et al., 2002), but reduction of muscle force to correct imbalances of force between antagonistic muscles (Nather et al., 1984) is an additional clinical aim.

The myotendinous junction is widely considered as the main (Tidball, 1991), or implicitly even the sole site (many studies in biomechanics e.g. Hawkins and Bey, 1997) for transmission of forces generated within sarcomeres onto the tendon and from there to bone. However, force transmission is possible also via trans-sarcolemmal proteins connecting muscle fibers along their full periphery of their length to the collagen reinforced extracellular matrix (ECM) (for a review see Berthier and Blaineau, 1997). As a consequence, muscle fibers have been shown to interact mechanically with the ECM and with each other within a bundle (Street and Ramsey, 1965; Street, 1983). Later, for whole muscle, such transmission has been named *myofascial force transmission* (*MFT*) (Huijing, 1999).

Moreover, muscle functioning within its normal context of connective tissues is connected to surrounding muscles and non-muscular structures and epimuscular myofascial force transmission (EMFT) occurs via such connections (e.g. Yucesoy and Huijing, 2007; Huijing, 2009). EMFT has been shown to cause asymmetric effects at muscle's origin and insertion and dependency of muscle characteristics on mechanical conditions within which it functions (Huijing, 2009; Yucesoy, 2010). Evidence for EMFT is accumulating for human muscles *in vivo* (Oda et al., 2007; Bojsen-Moller et al., 2010; Huijing et al., 2011; Carvalhais et al., 2013).

It is quite conceivable that PD (e.g. opening of the compartment) may affect MFT within a compartment, and because of mechanical interaction between muscles, acute mechanical effects of AT could also be present in muscles other than those on which the main

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surgical intervention was performed. Post-operative effects of AT have been investigated at the joint level by using e.g., gait analysis (Baddar et al., 2002; Zwick et al., 2002). However, effects on the target and non-targeted muscles were not studied explicitly.

Therefore, we designed the present study to test for such effects of MFT and test the following hypotheses for muscles within the anterior crural compartment of the rat: (1) effects of muscle lengthening surgery on the target muscle are different at distal and proximal tendons, (2) forces of non-targeted synergistic muscles are affected as well, and (3) PD is responsible from some of these effects.

2. Methods

2.1. Surgical procedures and preparation for experiments

Surgical and experimental procedures were approved by the Committee on Ethics of Animal Experimentation at Boğaziçi University. Young adult male Wistar rats ($n = 8$, mean body mass 327.0 g (SD 18.4 g)) were anesthetized using intraperitoneal injection of urethane (1.2 ml/100 g body mass). Additional doses were given if necessary (maximally 0.5 ml). The animals were placed on a heated pad (Harvard Apparatus) during surgery and data collection. Left hind limb skin and biceps femoris muscle were removed and the anterior crural compartment, containing extensor digitorum longus (EDL), tibialis anterior (TA) and extensor hallucis longus (EHL) muscles was exposed. Only limited distal fasciotomy was performed to remove the retinaculae, leaving fully intact connective tissues at the muscle. At a *reference position* selected (knee and ankle joint angles of 90° and 100° respectively), the four distal tendons of EDL muscle were tied together using silk thread. Matching markers were placed on distal tendons of EDL, TA and EHL muscles, as well as on the lower leg. Subsequently, the distal tendons of EDL as well as TA and EHL were cut as distally as possible. The proximal EDL tendon was cut from the femur, with a small piece of the lateral femur condyle still attached. To allow connection to force transducers, Kevlar threads were sutured to all cut tendons. Within the femoral compartment, the sciatic nerve was dissected and cut as proximal as possible. All its branches to muscles of that compartment were cut.

2.2. Experimental conditions and procedure

The rat was mounted in the experimental set-up (Fig. 1). The femur and foot were clamped, such that the knee was kept at 90° and the ankle in maximal plantar flexion (180°) to allow for free passage of Kevlar threads. Each Kevlar thread was connected to a force transducer (BLH Electronics Inc., Canton MA). The distal end of the sciatic nerve was placed on a bipolar silver electrode. Room temperature was kept at 26 °C. Muscle and tendon tissues were irrigated regularly by isotonic saline to prevent dehydration during the experiment.

Markers on EDL proximal tendon and distal tendons of TA and EHL were kept in their reference positions at all times. EDL length was changed by moving its distal force transducer (in steps of 1 mm), until 2 mm over distal optimum length, and EDL length-force data were collected at proximal and distal EDL tendons. Distal forces of TA and EHL were measured.

Muscles were activated maximally by supramaximal stimulation of the sciatic nerve at a constant current of 2 mA (Biopac Systems stimulator, STMISOC: square pulse width 0.1 ms, pulse train 400 ms, stimulation frequency 100 Hz). After setting EDL to a new length, two twitches were evoked and the muscles were tetanized 300 ms after the second twitch. At 200 ms after cessation of stimulation, another twitch was evoked. After these contractions, muscles were allowed to recover for 2 min. (EDL at short length, others at ℓref).

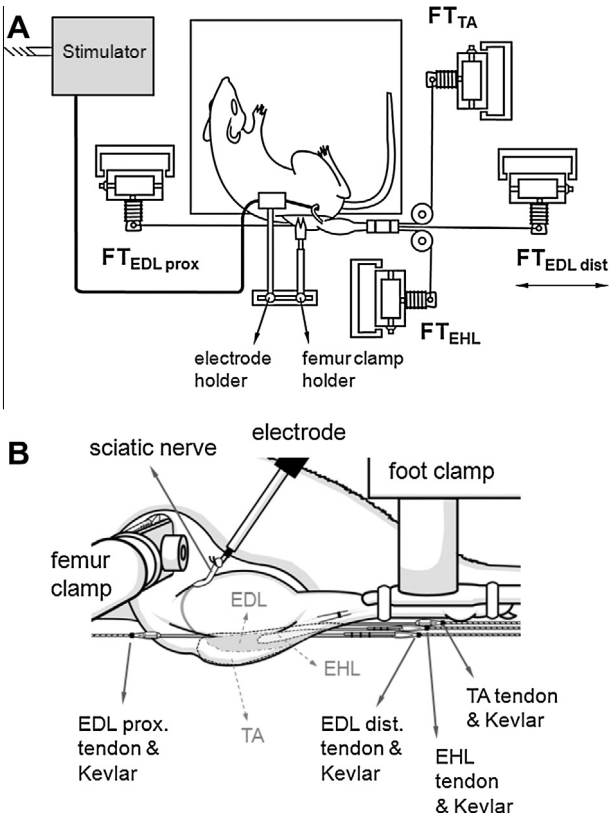

Fig. 1. Schematic view of the experimental setup. (A) The following tendon (–groups) were connected to a separate force transducer (FT): (1) proximal EDL tendon, (2) the tied distal tendons of the EDL, (3) the distal tendon of TA (4) the distal tendon of EHL. Throughout the experiment, TA and EHL muscles were kept at constant muscle-tendon complex lengths. Exclusively, the distal force transducer of EDL was repositioned to progressively increase EDL length, at each of which isometric contractions were performed. (B) The femur and foot were fixed by metal clamps. The sciatic nerve was placed on a bipolar silver electrode. Kevlar threads (hatched lines) were sutured to tendons (solid lines) to provide connection to their respective FT.

2.3. Experimental protocol

Before acquiring data, muscle-tendon complexes and their epimuscular connections were preconditioned by isometric contractions, alternatingly at long and short EDL lengths, until forces at short EDL length were reproducible (i.e. effects of previous activity at long length (Huijing and Baan, 2001) are removed).

Three conditions were studied: Firstly, the anterior crural compartment was intact. Secondly, PD was performed to gain access to the target muscle. This procedure includes the following steps. Laterally, a probe was inserted into the anterior crural compartment between EDL and TA muscles. Starting from the proximal end of the compartment, the probe tip was moved until the middle of the compartment was reached (i.e., approximately for 12 mm). Using a micro-scissor, half of the anterior crural compartmental fascia was cut (partial fasciotomy). Using a cotton swab intermuscular connections between EDL and TA along half of EDL length were removed (blunt dissection). Thirdly, using a scalpel blade (Surgeon, number 11) EDL proximal aponeurosis was transected at its middle, perpendicular to its longitudinal direction (AT). Subsequently, the muscles were activated with EDL at optimum length of the first condition. This causes tearing of the ECM along muscle fibers located below the site of AT. Effects of PD, AT and subsequent tearing of the ECM together are referred to as *cumulative effects of*