Force Enhancement of Quadriceps Femoris in Vivo and Its Dependence on Stretch-Induced Muscle Architectural Changes

Wolfgang Seiberl, Daniel Hahn, Florian Kreuzpointner, Ansgar Schwirtz, and Uwe Gastmann

The purpose of this study was to investigate if force enhancement (FE) in vivo is influenced by stretch-induced changes of muscle architecture. Therefore, 18 subjects performed maximum voluntary isometric (100° knee flexion angle) and isometric-eccentric-isometric stretch contractions (80°–100°; \( \omega = 60° \cdot s^{-1} \)) whereby pennation angle and fascicle length of vastus lateralis was determined using ultrasonography. We found significant (2-way repeated ANOVA; \( \alpha = 0.05 \)) enhanced torque of 5–10% after stretch as well as significant passive FE but no significant differences in muscle architecture between isometric and stretch contractions at final knee angle. Furthermore, EMG recordings during a follow-up study (\( n = 10 \)) did not show significant differences in activation and mean frequency of contraction conditions. These results indicate that FE in vivo is not influenced by muscle architectural changes due to stretch.

Keywords: ultrasound, fascicle length, stretch contraction, force potentiation, EMG

History dependence of muscle action is a well-described phenomenon in the literature (Abbott & Aubert, 1952; Herzog, 2004; Rassier & Herzog, 2004b) and especially force enhancement (FE) after active muscle stretch is a topic of high scientific interest. In general the latter is defined as enhanced force after active muscle stretch compared with an isometric contraction (FE) at corresponding muscle length and the same amount of muscle activation (Oskouei & Herzog, 2005). Main findings show that residual FE increases with stretch magnitude (Abbott & Aubert, 1952; Cook & McDonagh, 1995; Edman et al., 1978, 1982; Sugi, 1972) and sarcomere length on the ascending limb of the force-length relationship (F-l-r) (Edman et al., 1982). Enhanced forces were also detected on the ascending limb of the F-l-r (Peterson et al., 2004) and are shown to exceed the purely isometric forces on the plateau of the sarcomere F-l-r for optimized stretch conditions (Joumaa et al., 2008a). Force enhancement does not depend on the speed of the stretch (Edman et al., 1978; Sugi & Tsuchiya, 1988) and is known to be long lasting (Abbott & Aubert, 1952). These findings are supposed to be a property of all muscles and preparations (De Ruiter et al., 2000; Edman et al., 1978, 1982; Lee & Herzog, 2002; Sugi & Tsuchiya, 1988). For a detailed overview, we suggest Herzog et al. (2008).

To investigate the phenomenon of FE, an essential requirement is to guarantee equal muscle activation as well as sarcomere, fiber, or muscle lengths when comparing resulting forces or torques of different contraction types. Whereas these conditions can be well controlled for in vitro and in situ experiments, there is the problem with in vivo studies that muscle length is normally controlled by external joint angles. Hence, the question arises how precise muscle length can be controlled by joint kinematics. In this context, Finni et al. (2001) showed that fascicle length after eccentric contractions was longer than that of isometric reference contractions at the same external joint angle. According to the F-l-r (Gordon et al., 1966; Huxley, 1957; Huxley & Simmons, 1971) this must result in different force or torque. In contrast to that, Griffiths (1991) showed that muscle fibers tend to shorten during stretch of the muscle-tendon complex (MTC).

Thus, there is no general explanation for the behavior of fascicle length during and following muscle stretch, and the influence of stretch-induced changes of muscle architecture on FE in vivo is not analyzed yet. Especially...
on the descending limb of the F-l-r, where usually the highest FE is detectable (Edman et al., 1982; Rassier & Herzog, 2004b; Schachar et al., 2002), preceding stretch would either optimize (due to less length change) or impair (due to an increased length change) the ability of a muscle to produce enhanced forces. However, this is in contrast to findings in the literature on single fiber experiments (Edman et al., 1982; Julian & Morgan, 1979; Rassier, 2007; Sugi, 1972), where force enhancement constantly occurred although fiber length is controlled.

Nevertheless, in vivo experiments do show some differences according to the amount and duration of FE compared with in vitro findings (Lee & Herzog, 2002; Pinniger & Cresswell, 2007), especially the unexpected absence of enhanced torques in quadriceps femoris described by Hahn et al. (2007). The latter was assumed to be caused by an observed reduction in voluntary activation after stretch of 14–18%. In addition, a further explanatory approach presented by Hahn et al. (2007) is that the absence of significantly enhanced torques following stretch could partly originate from a difference of external knee flexion angle and real fascicle length changes, respectively. That means there might be a discrepancy between test and reference fascicle lengths at equal knee joint angles that possibly influences FE after stretch of MTC in vivo.

Therefore, by means of ultrasonography this work focuses on differences in muscle architecture between purely isometric and isometric contractions preceded by stretch. The corresponding research question is whether potential changes of pennation angle and fascicle length induced by active muscle stretch influence the phenomenon of FE in vivo.

Methods

Subjects

Eighteen moderately active male adults volunteered for this study. Mean values of age, body height, and body mass were 26.0 ± 1.8 years, 178.9 ± 4.8 cm, and 76.3 ± 6.7 kg, respectively. No subject was handicapped by any history of physical or neuronal disease and injury. Free, written informed consent was obtained from all subjects, and the study was conducted according to the Declaration of Helsinki.

Torque and Angle Measurements

Knee torque, knee angle, and angular displacement were measured by a rotational isokinetic dynamometer (Model Isomed2000, D&R Ferstl GmbH, Germany). Data sampling frequency was 200 Hz.

Measurements of Muscle Architecture

To obtain information of inner-body muscle movement, an ultrasound system (LOGIQ e, GE Healthcare, USA) with a 7.5-MHz linear probe was used. After visibility of echoes of the muscle architectural structures were checked during knee movement, the probe was fixed to the mid-parts vastus lateralis (VL). Motionless positioning of the probe was controlled for each single measurement. The images (20 Hz) were sent in real time to a digital video camera (50 Hz). The software Vicon Motus (Vicon Motion Systems, Oxford, U.K.) was used to synchronize ultrasound measurements with analog data. As probe width was 4.5 cm, an estimation model according to Finni et al. (2001, 2003) served for calculation of the whole fascicle lengths (Figure 1).

Measurements of Muscle Activation

(Follow-Up Study)

Muscle activation of rectus femoris (RF), vastus medialis (VM), and VL was obtained using surface electromyography (EMG). Bipolar surface electrodes (AMBU Blue Sensor P, Germany) with an interelectrode distance of 2 cm were used for EMG recording. A single reference electrode was attached to the patella. The EMG signals

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**Figure 1** — Estimation model used for determination of fascicle length (FL). FL was calculated with tracked length \(l_1\) (gray line) added to calculated length \(l_2\) (dashed line) \(\frac{h}{\sin(s)}\). Parameters \(l_1\), \(h\), and \(s\) are means measured over 500-ms intervals using a 4-point model with Vicon Motus motion capture software (Vicon Motion Systems, GB). For model details see Finni et al. (2001, 2003).
were collected at a sampling rate of 1000 Hz and band-pass filtered at 10 Hz and 500 Hz. Subject preparation and placement of electrodes was conducted following the guidelines of the SENIAM group (Hermens et al., 1999).

**Experimental Settings and Protocol**

Subjects were placed on the dynamometer according to the experimental setting published by Hahn et al. (2007). Initial start position for stretch was 80° of knee flexion, and the end position 100° of knee flexion, resulting in a total of 20° of stretch amplitude, with 0° corresponding to full knee extension. Comparisons of purely isometric and isometric torques following stretch were done at the final knee flexion angle of 100°. All angles of isometric and stretch contractions correspond to the descending limb of the torque–angle relationship (t–a–r) of the quadriceps femoris (Hahn et al., 2007; Pincivero et al., 2004). Before the actual test protocol was performed, every subject took part in at least two training sessions to minimize learning effects during tests. At test day, after a standardized warm-up program, all subjects had to perform three maximal voluntary isometric contractions (MVCs) at initial 80° of knee flexion angle to define a 95% start trigger for dynamic muscle action trials. Thereafter, three isometric–eccentric–isometric (20° stretch, 60° s−1 angular velocity) and three purely isometric (100° knee flexion angle) muscle contractions were performed in a randomized order. The 95% start trigger was used to control maximal voluntary contraction level and fatigue because lever arm did not start stretch contractions when 95% of individual isometric maximum were not reached. In this case, additional time to recover was required (Hahn et al., 2007). In addition, to distinguish between passive and active torques, purely passive trials were assessed for isometric (100° of knee flexion) and stretch conditions of the deactivated muscle. Subsequently, these data were subtracted from the total torque of active trials. In the follow-up study, no pure passive curves were measured and subtracted from the torque data because data analysis of the results showed that relative torque was not affected by the influence of passive forces. Every subject got as much rest as needed, but a minimum of 3 min was enforced. Every trial was accompanied by maximal verbal motivation.

**Data Analysis**

Analyzed parameters were torque, pennation angle (PA) of muscle fascicles, and calculated fascicle length (FL). In addition, muscle activation, mean frequency, and torque of n = 10 subjects were analyzed in a follow-up study. These parameters were assessed for an interval of 500 ms at five specific time segments (Figure 2): before stretch (BS); 0.5–1, 1.5–2, and 2.5–3 s after stretch (AS 1–3); and 1.5–2 s after deactivation (AD), respectively.

Torque was normalized to isometric reference MVC (except passive torque) at corresponding time segments to standardize contraction time and to determine possible FE. Concerning EMG, root mean square (RMS) and mean absolute value (MAV), both over an interval of 500 ms, were determined for statistical analysis of differences between action conditions. In addition, Fourier transformation was used to analyze the mean frequency of muscle action for the contraction time of 0.5–3 s AS.

Five laboratory assistants performed analysis of ultrasound data in parallel to minimize error in estimation of muscle architectural structures. To control comparability of the investigators, intraclass correlation (ICC) factors were calculated.

Mean values of the measured variables for each testing condition across all subjects were used for calculations. After control on normality (Kolmogorov–Smirnov test) two-way repeated-measures ANOVA and Bonferroni post hoc comparisons served for statistical analysis (α = 0.05).

**Results**

**Torque**

Our results (n = 18) showed significant (p < .001) enhanced torque of 108.6 ± 5.3% (171.1 ± 22.8 N·m to 158.0 ± 23.2 N·m) at AS1 and 105.5 ± 6.3% (159.6 ± 21.7 N·m to 151.2 ± 22.5 N·m) at AS2 (p < .01). For the time period AS3, mean values are slightly enhanced (102.3 ± 8.0%), but torque of 149.2 ± 20.8 N·m after active stretch was not significantly different compared with isometric reference of 145.8 ± 21.2 N·m anymore (Figure 3). Torque after deactivation of a purely isometric MVC was 0.12 ± 0.9 N·m and 0.88 ± 0.83 N·m after deactivation of an isometric–eccentric–isometric contraction. Thus, we found a significant (p < .001) passive force enhancement (FEpassive) after deactivation of quadriceps femoris (Figure 4).
Pennation Angle (PA) and Fascicle Length (FL)

Ultrasound data of three subjects were discarded from analysis because muscle architectural characteristics could not be defined with adequate accuracy. Therefore, analysis of muscle architecture was made with $n = 15$ subjects. When the knee was stretched from 80° to 100° of knee flexion, the PA of VL significantly ($p < .05$) decreased from 15.4 ± 1.6° to 14.2 ± 1.4° and FL significantly ($p < .05$) increased from 63.6 ± 6.5 mm to 68.6 ± 6.0 mm. At final external knee flexion angle of 100°, no significant difference between action conditions and within duration of contraction from AS1 to AS3 was found. After deactivation there was a significant ($p < .05$) increase of PA from 14.0 ± 1.7° to 15.0 ± 2.3° but no change in FL (Figure 5). Detailed data of PA and FL can be found in Table 1.

Electromyography

Muscle activation of VL, VM, and RF did not show any significant differences between the two contraction conditions in AS1–3 and AD. Furthermore, there was no difference in mean frequency of EMG during isometric contractions after stretch- and purely isometric contractions (Table 2).

Discussion

Besides the indirect detection of Hahn et al. (2007), we could give the first evidence for FE of maximal voluntarily activated quadriceps femoris. Because most in vivo investigations concentrated on small muscles, such as the adductor pollicis of the human hand (Lee & Herzog, 2002; Oskouei & Herzog, 2005), or submaximal forces of bigger muscles (Pinniger & Cresswell, 2007), these findings enrich the knowledge concerning the phenomenon of FE in vivo. However, not all of our results agree with literature, especially data concerning duration and amount of FE.

Torque

We found significantly enhanced torque of the quadriceps femoris after active muscle stretch of about 6–9% (Figure 3). Thus, it appeared to be a bit smaller compared with former findings on in vivo human muscles, such as 12% (De Ruiter et al., 2000; Pinniger & Cresswell, 2007) and up to 17% in Lee & Herzog (2002). A possible reason might be activation inhibition during stretch of activated large human muscles (Babault et al., 2001; Beltman et al., 2004), as Hahn et al. (2007) found activation of quadriceps femoris to be 14–18% reduced in comparison with a purely isometric reference contractions.

In contrast to expectations from literature (Abbott & Aubert, 1952), significant enhanced torque could just be detected for AS1 and AS2, which reflects a time period of only 2 s. For the stretch condition, the decrease of torque was significant over time of contraction from AS1 to AS2...
for this phenomenon. Thus, we assume that the absent long lastingness of FE is possibly due to differences between responders and nonresponders (Abbott & Aubert, 1952). Nevertheless, the characteristics that torque during stretch signifi-

cantly decreased more than during isometric contractions from the literature (Hahn et al., 2007; Lee & Herzog, 2002), we did not find significant (α = .05) differences in activation level between stretch and isometric contractions (Figure 6). Furthermore, the mean frequency of both contraction conditions did not show any significant differences. Concerning force enhancement, significantly enhanced torque of 10.4 ± 6.5%, 7.5 ± 6.3%, and 5.1 ± 7.4% could be detected throughout the whole contraction time including AS1, AS2, and AS3. Therefore, the results of this follow-up do not match the data of the original study and give new aspects to this work. First, activation is shown to be equal for the different test conditions, thereby disproving the assumption that FE in vivo might be influenced by stretch-induced neuronal inhibition (Babault et al., 2001). Second, FE could be detected beyond 2.5 s after stretch, at least in a subset of original subjects.

Although recruitment of subjects for the follow-up happened by accident and there were absolutely no special selection criteria to get as many original subjects as possible, 7 out of the 9 subjects that participated in both studies correspond to the 12 “responders” in the original study. In addition to the responder who took part only in the follow-up, there were one-fifth nonresponders in the follow-up compared with one-third in the original study. This might explain the enhancement in AS3. Comparing torque responses after stretch, the 9 subjects who participated in both studies did not show significant differences in means of FE at AS1, AS2 and AS3 (Figure 7). Nevertheless, the characteristics that torque during stretch contraction significantly decreased more than during isometric contraction remained the same. This leads to the assumption that post-eccentric torque might reach or even drop below isometric level when contraction is maintained for longer time. Hence, the long-lasting effect as described in literature (Abbott & Aubert, 1952) still has to be questioned for in vivo muscle actions. As EMG data did not show any differences between contraction conditions, muscle activation cannot provide answers to this behavior.

Table 1  Means and standard deviation (SD) of pennation angle and fascicle length of vastus lateralis

<table>
<thead>
<tr>
<th>Time</th>
<th>Pennation Angle (degrees)</th>
<th>Fascicle Length (millimeters)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Isometric (SD)</td>
<td>Stretch (SD)</td>
</tr>
<tr>
<td>BS</td>
<td>15.4 (1.6)</td>
<td>63.6 (6.5)</td>
</tr>
<tr>
<td>0.5–1 s AS</td>
<td>13.9 (1.9)</td>
<td>14.2 (1.4)</td>
</tr>
<tr>
<td>1.5–2 s AS</td>
<td>13.8 (1.9)</td>
<td>14.0 (1.7)</td>
</tr>
<tr>
<td>2.5–3 s AS</td>
<td>13.7 (2.0)</td>
<td>14.0 (1.7)</td>
</tr>
<tr>
<td>1.5–2 s AD</td>
<td>15.2 (2.5)</td>
<td>15.0 (2.3)</td>
</tr>
</tbody>
</table>

Note. Means are measured during isometric-eccentric-isometric contractions (20° stretch, 60° · s⁻¹ angular velocity, 100° final knee flexion angle) and purely isometric contractions (100° knee flexion angle) at five instances in time over an interval of 500 ms: Before stretch (BS), 0.5–1 s after stretch (AS), 1.5–2 s AS, 2.5–3 s AS, and 1.5–2 s after deactivation (AD) of the muscle.

(p < .001) and AS2 to AS3 (p < .01). At AS3, normalized torque after stretch almost reached isometric reference (102.1%) and no significant difference remained (Figure 3). Because fatigue was controlled with the 95% trigger constant contraction times, and a randomized test protocol, it is not supposed to be an explanation for this phenomenon.

Furthermore, the idea that FE might partly be associated with a responder and nonresponder affinity (Hahn et al., 2008; Oskouei & Herzog, 2005) could also apply for this study. According to this theory only responders react to an active stretch with enhanced force or torque whereas nonresponders do not show differences between isometric and stretch contractions. Thus, not all subjects in this study reacted to stretch with enhanced torque, whereas others showed enhancement of up to 20%. Thus, the mixture of responders and nonresponders might be a reason for the absence of an obvious FE in AS3. According to this, one-third (6 out of 18 subjects) reached or dropped below the isometric reference and therefore did not show enhanced torque continuously throughout contraction time. However, the question remains why torque after stretch decreased more than torque of purely isometric contractions, so that we cannot support characteristics of long lastingness of FE proposed in the literature for over 50 years (Abbott & Aubert, 1952). The significant decrease throughout the time segments AS1 to AS3 found in this study is not compatible with findings in the literature, where FE appears as long as activation is maintained (Abbott & Aubert, 1952). However, there is evidence that the properties of FE in vivo differ between studies that used electrical stimulation and voluntary activation, respectively (Cook & McDonagh, 1995; Lee & Herzog, 2002). Thus, we assume that the absent long lastingness of FE is possibly due to differences between artificial and voluntary activation, especially when using maximum activation. But for all that, without control of muscle activation we cannot provide a final explanation for this phenomenon.

Therefore a follow-up study concentrating on muscle activation was arranged with 10 subjects (9 out of the original sample). Surprisingly, against expectations from the literature (Hahn et al., 2007; Lee & Herzog, 2002), we did not find significant (α = .05) differences in activation level between stretch and isometric contractions (Figure 6). Furthermore, the mean frequency of both contraction conditions did not show any significant differences. Concerning force enhancement, significantly enhanced torque of 10.4 ± 6.5%, 7.5 ± 6.3%, and 5.1 ± 7.4% could be detected throughout the whole contraction time including AS1, AS2, and AS3. Therefore, the results of this follow-up do not match the data of the original study and give new aspects to this work. First, activation is shown to be equal for the different test conditions, thereby disproving the assumption that FE in vivo might be influenced by stretch-induced neuronal inhibition (Babault et al., 2001). Second, FE could be detected beyond 2.5 s after stretch, at least in a subset of original subjects.
Table 2  Mean absolute value (in volts) and standard deviation (SD) of muscle activation of vastus lateralis, vastus medialis, and rectus femoris

<table>
<thead>
<tr>
<th>Time</th>
<th>Vastus Lateralis</th>
<th></th>
<th>Vastus Medialis</th>
<th></th>
<th>Rectus Femoris</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Iso (± SD)</td>
<td></td>
<td>SC (± SD)</td>
<td></td>
<td>Iso (± SD)</td>
<td></td>
</tr>
<tr>
<td>0.5–1 s AS</td>
<td>0.84 (0.41)</td>
<td>0.91 (0.47)</td>
<td>0.60 (0.46)</td>
<td>0.69 (0.61)</td>
<td>0.52 (0.25)</td>
<td>0.51 (0.18)</td>
</tr>
<tr>
<td>1.5–2 s AS</td>
<td>0.86 (0.41)</td>
<td>0.90 (0.48)</td>
<td>0.60 (0.46)</td>
<td>0.61 (0.50)</td>
<td>0.54 (0.25)</td>
<td>0.52 (0.21)</td>
</tr>
<tr>
<td>2.5–3 s AS</td>
<td>0.87 (0.44)</td>
<td>0.90 (0.48)</td>
<td>0.60 (0.44)</td>
<td>0.63 (0.49)</td>
<td>0.54 (0.25)</td>
<td>0.51 (0.18)</td>
</tr>
<tr>
<td>1.5–2 s AD</td>
<td>0.008 (0.004)</td>
<td>0.007 (0.003)</td>
<td>0.009 (0.006)</td>
<td>0.008 (0.006)</td>
<td>0.005 (0.003)</td>
<td>0.005 (0.002)</td>
</tr>
<tr>
<td>MF (0.5–3 s AS)</td>
<td>76.18 (9.56)</td>
<td>76.26 (9.20)</td>
<td>77.40 (15.02)</td>
<td>74.81 (14.41)</td>
<td>71.89 (6.85)</td>
<td>72.51 (8.30)</td>
</tr>
</tbody>
</table>

Note. Means are measured during purely isometric contractions (Iso; 100° knee flexion angle) and stretch contractions (SC; 20° stretch, 60°·s⁻¹ angular velocity, 100° final knee flexion angle) at four instances in time over an interval of 500 ms: 0.5–1 s after stretch (AS), 1.5–2 s AS, 2.5–3 s AS, and 1.5–2 s after deactivation (AD) of the muscle. Mean frequency (MF) of both contraction conditions is analyzed over the contraction period of 0.5–3 s AS.

![Figure 5](image_url) — Fascicle length (FL) and pennation angle (PA) of vastus lateralis before and following stretch. Parameters according to muscle action conditions of PA (▲ isometric, ▼ isometric-eccentric-isometric) and FL (® isometric, • isometric-eccentric-isometric) were calculated as means measured during stretch contractions (SC) at four instances in time over an interval of 500 ms: 0.5–1 s AS, 1.5–2 s AS, 2.5–3 s AS, and 1.5–2 s after deactivation (AD) of the muscle.

![Figure 6](image_url) — Muscle activation [MVC] and mean absolute value (MAV; in volts) and standard deviation of muscle activation of vastus lateralis (squares), vastus medialis (triangles), and rectus femoris (circles). Mean absolute value measured during purely isometric contractions (iso) (100° knee flexion angle) and isometric-eccentric-isometric contractions (20° stretch, 60°/s angular velocity, 100° final knee flexion angle) at four instances in time over an interval of 500 ms: 0.5–1 s AS, 1.5–2 s AS, 2.5–3 s AS, and 1.5–2 s after deactivation (AD) of the muscle.
The follow-up, we found post-eccentric contraction time in the original study. For (Herzog & Leonard, 2002, 2005), it is even more surprising that almost all subjects of the follow-up showed FE throughout contraction time in the original study as well as in the follow-up needs further investigation according to responder and nonresponder theories. Fiber type distribution is already discussed as a possible factor for classification (Hahn et al., 2008; Oskouei & Herzog, 2005) but no final answer could be given to this topic yet.

The small but significant passive FE after deactivation of the quadriceps femoris found in the original (Figure 4) as well as in the follow-up study fits in with the common theory of an active and a passive component that together implement enhanced forces after stretch (Bagini et al., 2002; Labeit et al., 2003; Mehta & Herzog, 2008; Peterson et al., 2004; Pinniger et al., 2006). As passive FE is supposed to be already apparent during active FE (Herzog & Leonard, 2002, 2005), it is even more surprising that we did not find active FE throughout the entire post-eccentric contraction time in the original study. For the follow-up, we found significant FE passive within the same amount of torque as measured before. Statistical analysis of EMG signals after deactivation of the muscles showed no differences between action conditions with EMG values around zero (Figure 6). Passive FE was found in single fibers (Lee et al., 2007; Rassier & Herzog, 2004a) and myofibrils (Joumaa et al., 2007) as well as in vivo whole muscles (Hahn et al., 2007; Lee & Herzog, 2002). In the literature, passive FE was mainly associated with the structural protein titin, a molecular spring between the Z-lines and M-band of a sarcomere (Granzier & Labeit, 2004; Horowits, 1992). However, Joumaa et al. (2008b) recently reported that titin can account for only about 25% of FE passive. Therefore, the origin of the major part remains unknown. But because absolute means of FE passive in this study are vanishing small, their influence on active FE in voluntarily activated quadriceps femoris, and therefore its relevance for daily living, must be considered to be very small.

**Fascicle Length and Pennation Angle**

Normally, force enhancement is defined as a property for equal activation and muscle lengths. In this context, in vivo experiments suffer from the problem that internal parameters such as fascicle length cannot be controlled easily. In contrast, several factors may take influence on fascicle length changes during in vivo muscle stretch. First, due to the complexity of the MTC, it is not clear which parts of the muscle (tendon, elastic components in series and in parallel, and fascicles) are stretched more or less. Furthermore, there is probably interdependency between stretch-induced reduction in muscle activation and fascicle length. Based on these problems, we hypothesized that stretch-induced changes in FL could possibly influence FE in vivo.

According to the F-l-r of skeletal muscle, differences in fascicle length should result in different forces or torques, so that on the descending limb of the F-l-r, where all experiments were performed, force decreases with an increase of muscle or fascicle length and vice versa. If FE is influenced by length discrepancies between action conditions, FL after active muscle stretch should be shorter or longer compared with FL of the purely isometric reference.

We did not find significant differences in FL of VL for isometric and stretch contractions although there have been consistently lower means of 2.1–3.2 mm for FL in AS1 to AS3 (i.e., shorter fascicle lengths) in comparison with purely isometric contractions. However, for single cases we found shorter as well as longer FL after stretch, thereby indicating that changes in muscle architecture are not likely to account either for gain or for loss in torque after active muscle stretch in vivo. Furthermore, at AS 3, where no FE was measured, the same amount of fascicle length change was observed, indicating that differences in fascicle length are not responsible for enhanced torque. A correlation of r = .14 (p = .372) between AFL of isometric and stretch contractions and torque after stretch (Figure 8) strengthens this assumption. Moreover, this finding implies that the underlying muscle architecture can vary even though the external joint angle remains the same.

On the one hand these results neglect torque-enhancing influences due to muscle architectural changes during and after stretch for the in vivo muscle action of MTC. Furthermore, if FE in vivo would be related to an optimized working range on the F-l-r and not to contractile mechanism as discussed in the literature (Colombini et al., 2007; Mehta & Herzog, 2008), these results would be totally incongruent with findings in the literature, where FE is demonstrated in single fiber or myofibril preparations (Joumaa et al., 2008a; Peterson et al., 2004; Rassier et al., 2003) and where muscle length and activation can be exactly controlled. Therefore, these results are in good accordance with the prevalent understanding of the
underlying mechanisms. On the other hand, these findings also cannot help to explain the differences according to the amount and duration of FE compared with in vitro findings as reported several times in literature (Hahn et al., 2007; Lee & Herzog, 2002; Pinniger & Cresswell, 2007).

Based on our data, the hypothesis that FE might be influenced by stretch-induced changes of muscle architecture has to be rejected. However, before drawing final conclusions, our method for the determination of fascicle length changes needs to be discussed. First of all, the estimation model presented is a strong simplification, assuming that contractile behavior of the whole quadriceps femoris is represented by VL. Moreover, the change in fascicle length is thought to be linear and the influence of moment arm and joint geometry is neglected. Despite these simplifications, an altered fascicle length at a given joint angle should result in a gain or loss in torque since the influence of joint geometry is eliminated. In addition, despite an investigator ICC of 0.8, the analysis of ultrasound images needs to be discussed as well. Finni et al. (2003) stated errors originating from nonlinear aponeurosis to be 2–7%, but since architectural structures are not as linear as assumed in the used model, our data analysis is potentially more erroneous.

To summarize the main results, this work provides direct evidence for active and passive FE in maximal voluntarily activated quadriceps femoris in vivo. Enhanced torque of 5–10% compared with isometric reference decreased significantly with contraction time. Beyond 2.5 s, AS significant difference between torques of stretch and isometric contractions remained only in a subset of subjects. Additional EMG recordings during a follow-up study did not show any differences between contraction conditions, thereby disproving influences of neuronal inhibition during stretch contractions. Fascicle length of VL significantly increased during 20° stretch on the descending limb of the F-I-r, but there was no difference between isometric-eccentric-isometric and purely isometric contractions at final knee flexion angle (100°). Correlations of torque and fascicle length were calculated and found to be very small (r = .14) for VL. This shows that stretch-induced muscle architectural changes do not contribute to the development of FE in vivo. In fact, fascicle length changes did not show any significance, so their influence on FE in vivo is to be neglected.

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References


