Faculty of Sport Science of the Ruhr-Universität Bochum
Department of Movement Science & Biomechanics

Master thesis in Sport Science

Is muscle lengthening during force reduction in fixed-end contractions an eccentric contraction?

(„Ist die Muskelverlängerung bei Kraftverringerung in Kontraktionen mit konstanter Länge der Muskel-Sehnen-Einheit eine exzentrische Kontraktion?")

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Abstract

Background: Steady-state contractions require less muscle activity when they were immediately preceded by active lengthening. This has usually been investigated with a stretch of the whole muscle-tendon-unit (MTU). Yet, the muscle can also lengthen when contraction intensity is reduced during fixed-end contractions.

Aim: The effects of different magnitudes of torque reduction on activity of m. tibialis anterior (TA) during post-reduction steady-states were investigated, in voluntary fixed-end dorsiflexion contractions.

Methods: Ten participants matched predefined torque courses involving an initial torque rise to 85% MVC followed by torque reduction to 45%, 30%, and 15% MVC. Also, reference contractions were performed in which torque was raised to and maintained at the respective levels of 45%, 30% and 15% MVC. Dorsiflexion torque, TA electromyography (EMG) and ultrasound images were recorded during contractions.

Results: Greater Dorsiflexion torque reductions to the different lower levels involved greater TA fascicle lengthenings which were all different from each other ($P \leq 0.01$). Reduction to 45% ($P = 0.8$/$P = 0.34$ for early/late post-reduction steady-state, respectively), to 30% ($P = 0.06$/$P = 0.54$) and to 15% MVC ($P = 0.06$/$P = 0.46$) did not alter TA EMG at the respective post-reduction level.

Conclusion: Regardless of the difference between the high initial and the subsequent lower level, the additional contractile depression established during the high torque increase is completely abolished by reduction of contraction intensity. As no indications for stretch-induced contractile enhancement were found, it might be that potentiation mechanisms – triggered by the high initial contraction intensity – are the dominant factor in reducing initially established contractile depression.

Zusammenfassung

Hintergrund: Muskelkontraktionen mit einer konstanten Kraft benötigen weniger elektrische Muskelaktivität, wenn der Muskel sich unmittelbar zuvor aktiv verlängert hat. Dieser Effekt wurde bisher in der Regel mit einer Verlängerung der gesamten Muskel-Sehnen-Einheit (MSE) untersucht. Jedoch kann sich der Muskel auch dann verlängern, wenn während einer Kontraktion mit konstanter Länge der MSE die Intensität verringert wird.

Ziel: Untersucht wurden die Auswirkungen von Drehmomentverringerungen verschiedener Größenordnungen auf die anschließenden Muskelaktivitäten des m. tibialis anterior (TA) bei konstantem Drehmoment, in willentlichen Dorsalflexionen mit konstanter Länge der MSE.

Methoden: 10 Teilnehmer/-innen führten Kontraktionen entsprechend vorgegebenen Drehmoment-Verläufen aus, die einen Drehmomentanstieg auf 85% MVC beinhalteten, gefolgt von einem Abfall auf 45%, 30% und 15% MVC. Zusätzlich wurden Referenzkontraktionen bei konstanten entsprechenden Drehmomenten von 45%, 30% und 15% MVC durchgeführt. Während der Kontraktionen wurden Drehmomente der Dorsalflexion sowie Elektromyogramm (EMG) und Ultraschallbilder des TA aufgezeichnet.

Ergebnisse: Größere Drehmomentverringerungen der Dorsalflexion auf die verschiedenen tieferen Levels brachten größere Verlängerungen der TA-Faszikel mit sich, die sich jeweils untereinander unterschieden ($P \leq 0.01$). Die Verringerung auf 45% ($P = 0.8$/$P = 0.34$ jeweils für die frühe/späte konstante Kontraktion nach Drehmomentabfall), auf 30% ($P = 0.06$/$P = 0.54$) und auf 15% MVC ($P = 0.06$/$P = 0.46$) veränderten das TA EMG auf dem jeweiligen tieferen Level nicht.

Fazit: Ungeachtet des Unterschieds zwischen dem anfänglichen hohen und dem späteren tieferen Level wird die während des hohen Drehmomentanstiegs ausgebildete, zusätzliche kontraktile Verminderung durch Reduktion der Kontraktionsintensität vollständig rückgängig gemacht. Zumal keine Anzeichen für dehnungsinduzierte kontraktile Erhöhung gefunden wurden, könnte es sein, dass Potenzierungsmechanismen – ausgelöst durch die hohe anfängliche Kontraktionsintensität – der dominante Faktor bei der Reduzierung der anfänglich ausgebildeten kontraktilen Verminderung sind.

Contents

1 Introduction

One of the most prominent features of eccentric contractions is that isometric steady-state forces following active lengthening are greater than isometric steady-state forces without preceding lengthening, despite similar activation levels and muscle lengths in both compared conditions (Edman, 2012; Herzog & Leonard, 2013; Seiberl et al., 2015). This phenomenon was termed residual force enhancement (rFE) (Edman et al., 1982) and was observed in isolated animal muscle preparations of different orders of magnitude (Abbott & Aubert, 1952; Edman et al., 1978; Herzog & Leonard, 2002; Joumaa et al., 2007; Julian & Morgan, 1979; Pinnell et al., 2019), down to the level of single sarcomeres (Leonard et al., 2010) – proving it to be an intrinsic property of muscle actively lengthening. Several studies on human muscle contractions in vivo showed rFE to occur in maximal and submaximal contractions at different activation levels ranging down to 10% of maximal voluntary contraction (MVC), within electrically stimulated as well as voluntary contractions and in a variety of muscles (De Ruiter et al., 2000; Lee & Herzog, 2002; Oskouei & Herzog, 2006; Pinniger & Cresswell, 2007; Tilp et al., 2009).

In contrast to rFE, steady-state forces following active muscle shortening are decreased relative to isometric references with the same muscle length and activation level (Abbott & Aubert, 1952; Edman, 1976; Lee & Herzog, 2003), which is termed residual force depression (rFD). RFD also occurred across isolated muscle preparations of all structural levels (Abbott & Aubert, 1952; Granzier & Pollack, 1989; Maréchal & Plaghki, 1979; Pinnell et al., 2019), as well as following voluntary and electrically-induced muscle shortenings of different human muscles in vivo (De Ruiter et al., 1998; Lee et al., 1999; Power et al., 2014; Rousanoglou et al., 2007; Tilp et al., 2009).

When posteccentric or postconcentric steady-states were matched to the force level of the isometric reference contraction, reduced (activation reduction, AR) or increased activations (activation increase, AI) were found following muscle lengthening or shortening, respectively, when compared to the isometric reference (Altenburg et al., 2008; Jones et al., 2016; Oskouei & Herzog, 2005; Paquin & Power, 2018; Rousanoglou et al., 2007). AR and AI were interpreted as manifestations of lengthening-induced force enhanced states and shortening-induced force depressed states, respectively, as lower (AR) or higher (AI) activities are necessary in those states to produce the same force as in the reference contraction (Hahn et al., 2007; Oskouei & Herzog, 2005; Rousanoglou et al., 2007).

In previous in vivo studies, rFE and AR have typically been induced by actively stretching the whole muscle-tendon-unit (MTU) via changes of the joint angle. The question arises if active muscle lengthening occurring in fixed-end contractions (constant MTU-length) might also trigger rFE-related mechanisms, thus influencing the subsequent contractile output. It is well known that in fixed-end contractions, the muscle shortens during force rise, stretching tendons and aponeuroses of the series elastic component (Fukunaga et al., 1997; Griffiths, 1991; Ito et al., 1998). This happens more pronounced in relatively compliant MTUs like that of the m. tibialis anterior (TA), where shortening of muscle fibers, accompanied by an increase in pennation angle, was observed during fixed-end force ramps (Ito et al., 1998; Pasquet et al., 2005; Raiteri et al., 2015; Reeves & Narici, 2003). Obviously, when the muscle reduces its activity following such a force rise, the fascicles would lengthen back, but this aspect of fixed-end contractions was not given much attention to in previous studies. This behavior was shown for example in m. tibialis posterior, which induces fascicle lengthening by lowering its activity during the early stance phase of gait, while the MTU length remains relatively constant (Maharaj et al., 2016).

One study has recently addressed the question if fascicle shortening and lengthening during fixed-end force rise and reduction might induce rFD and rFE, respectively, influencing contraction history and thus the resulting steady-state contractile output (Raiteri & Hahn, 2019). There, force was raised to 60% MVC and subsequently reduced to 40% MVC (High-Mod condition), in voluntary fixed-end dorsiflexions. The steady-state following this force reduction yielded a similar TA activation level as the reference contraction, where force was raised to and maintained at the level of 40% MVC, while TA fascicle lengths and pennation angles were similar in both steady-states (Raiteri & Hahn, 2019). This study also included an activation-controlled experiment of similar contraction intensities with the same outcome, i.e., that activation reduction from 30% to 20% MVC led to a similar force level as the reference of 20% MVC. The results were explained with initial shortening-induced rFD/AI and subsequent contractile enhancement, possibly via stretch-induced rFE-related mechanisms and/or other potentiating mechanisms – triggered by the previous greater contraction intensity – in the High-Mod condition; the initially established higher rFD/AI level was thought to be thus abolished, so that the resulting activation or force in the steady-states following intensity reduction was similar to the reference contraction, in which fascicles haven´t undergone the additional shortening and subsequent stretch before arriving at the reference level (Raiteri & Hahn, 2019).

The main purpose of the thesis is to investigate how in voluntary fixed-end contractions, various magnitudes of force reduction – proceeding from a common initial high force level – would affect the contractile outputs of the post-reduction steady-states. Greater rises above the reference level in the ramp phase should lead to increasing levels of additional rFD/AI, because rFD has been shown to rise with shortening amplitude (Edman, 1975; Granzier & Pollack, 1989; Herzog et al., 1998). The corresponding subsequent greater torque reductions back to the reference level could similarly lead to rising levels of rFD/AI-diminution, based on the possible contribution of two mechanisms; the further lengthening of muscle fibers might induce greater rFE (Abbott and Aubert, 1952; Cook & McDonagh, 1995; Herzog & Leonard, 2002), and potentiation mechanisms could increase contractile outputs of lower post-reduction levels to a greater relative extent (Abbate et al., 2000; MacIntosh & Willis, 2000; Vandenboom et al., 1993). Thus, it is possible that forces or activation levels of the references could be matched at the final steady-state of various "High-Mod" contractions, regardless of the intensity reduction that they involve. The thesis also aims at assessing the possible contribution of rFE-related mechanisms in this context. A decay of contractile output during the post-reduction steady-state that is greater than in the reference contraction could indicate that certain cross-bridge mechanisms have been triggered, which are known to transiently enhance forces following active lengthening (De Ruiter et al., 2000; Herzog et al., 2006; Noble, 1992).

To accomplish these aims, a torque-controlled experiment was conducted which involved voluntary, submaximal, fixed-end dorsiflexions – with a high initial ramp followed by varying magnitudes of torque reduction. Changes in muscle architecture (i.e., fascicle lengths and pennation angles) and electromyography (EMG) of the TA muscle during contractions were assessed. Firstly, it is hypothesized that greater dorsiflexion torque reduction from 85% MVC – to 45%, 30% and 15% MVC, respectively – will lead to greater fascicle lengthening and greater decrease in pennation angle; secondly, that TA muscle activations during the early and late steady-state following these torque reductions will be similar to those of corresponding reference contractions, in which torque is raised to and maintained at the respective level of 45%, 30% and 15% MVC.

2 Methods

2.1 Subjects

Twelve healthy and recreationally active participants were tested in the study, of which three female and seven male were included in data analysis (age: 25 ± 1 years (mean ± SD), body mass: 72 ± 9 kg, height: 178 ± 7 cm). All participants gave their informed consent before participation. The study was approved by the Ethics Committee of the Faculty of Sport Science of Ruhr University Bochum and conducted in accordance with the Declaration of Helsinki.

2.2 Experimental setup

2.2.1 Torque measurements

Fixed-end dorsiflexion torques were recorded using a dynamometer (IsoMed2000, D&R Ferstl GmbH, Germany). The signal was sampled at a rate of 2kHz using a 16-bit Power-3 1401 and Spike2 data collection system (Cambridge Electronics Design, Cambridge, UK).

2.2.2 Ultrasonography

Ultrasound images from the mid-sagittal plane of the TA muscle were obtained for all test contractions using real-time B-mode ultrasonography (LS128 CEXT – 1Z, Telemed, Vilnius, Lithuania) with a flat, linear, 128-element transducer (LV7.5/60/128Z-2, Telemed). From TA´s superficial compartment, fascicle lengths and pennation angles relative to the central aponeurosis were assessed. The sonogram was recorded at a frequency of 6 MHz, with a field of view of 60 × 50 mm (width × depth) and a frame rate of ~34 Hz and displayed on a computer screen using Echowave II software (Telemed). Water-soluble transmission gel was spread on the transducer, to allow for acoustic contact to the participant´s skin. The system was used to assess the dimensions of the participant´s TA muscle (right leg) as well as the direction of its fascicles, and an appropriate place for the later attachment of EMG electrodes in the distal portion of the TA muscle belly was determined. Proximal to this place, the ultrasound transducer was positioned along the mid-sagittal plane of the TA muscle belly. The transducer was contained in a custom-made polystyrene frame, to minimize local muscle compression and alleviate the participant´s discomfort. The ultrasound image was optically assessed on the computer screen to determine a position where the superficial TA compartment, with its fascicles and their insertions to the adjacent aponeuroses, could clearly be identified. This was done while the participant was performing dorsiflexions of different intensities, to account for movements of the muscle and surrounding tissues during contractions, that could potentially diminish the sonogram´s quality. At the appropriate position, the transducer was securely fixed with adhesive bandage to the participant´s shank, to hold it in place for the duration of the experiment, preventing movement of the transducer relative to the skin.

2.2.3 Electromyography

Surface EMG recordings of the TA muscle were made using bipolar self-adhesive Ag/AgCl electrodes (Covidien, Mansfield, MA, USA) with a recording diameter of 8 mm. These were attached with an inter-electrode distance of 2 cm to the participant´s skin at the previously marked site, above the distal TA portion of the participant´s right leg, away from muscle borders, and aligned in fascicle direction. A reference electrode was placed laterally over the head of the fibula of the participant´s left leg. Before electrodes were attached, the skin was shaved, abraded, and cleansed with an alcohol solution. The attained EMG signals were amplified (NL 844 Pre-Amplifier, Digitimer Ltd., Hertfordshire, UK) and band-pass filtered between 10 and 500 Hz (NeuroLog System, Digitimer Ltd., Hertfordshire, UK), before undergoing analog-to-digital conversion at a sample rate of 2 kHz with the same 1401-Spike2 system as described above.

2.2.4 Positioning

The participant lay in a prone position, with the sole of his right foot put flat against a footplate that was attached to the dynamometer arm. The foot was fixed to the footplate at the participants metatarsals using a custom-built adjustable frame, while the backside of the heel was against the footplate´s firm heel-support. The angular orientation of the footplate was adjusted so that, during dorsiflexion of ~50% of maximum, it was in a 100° position relative to the participant´s shank (10° plantar flexion), which was measured using a digital goniometer (EPT-DAF 380 mm, Conrad Electronic SE, Hirschau, Germany). Maintaining this angle, the ankle joint center (approximated from the lateral malleolus) was optically aligned with the dynamometer´s axis of rotation by adjusting the footplate´s position. The foot was then additionally fastened to the footplate at the participant´s ankle with a nonelastic strap. To improve the participant´s comfort, padding was put between the foot and the frame/heel-support/strap, respectively. Also, a hip strap secured the participant in position during test contractions. The described arrangement, including the positions of footplate and dynamometer arm, were maintained for all contractions of the test procedure.

2.3 Experimental protocol

All participants performed a training session on a separate day prior to the actual test session, in which they absolved the whole procedure that is described in the following.

The TA MTU was preconditioned before testing, with five submaximal (1-second hold, 1-second rest, ~80% of maximum), and one verbally encouraged maximal dorsiflexion. The test protocol started with at least two maximal dorsiflexions (≤ 5% difference), to determine the dorsiflexion MVC torque and maximum TA activation. Between MVCs, a rest of three minutes was given to eliminate fatigue. The participant was provided with visual feedback of the torque tracing in real-time during maximal contractions, and verbally encouraged in a standardized manner.

Following the MVCs, the participant was tasked to match six different types of computer-displayed torque courses, which were calculated based on the previously attained MVC torque, each of them with a duration of 15 seconds. Three of these were the High-Low contractions (ramp-hold-reduce-hold), meaning that after rising to 85% MVC and maintaining this level for a certain time, torque was reduced to 45% (High-Low 45%), to 30% (High-Low 30%), and to 15% MVC (High-Low 15%), respectively (Fig. 1). The other three conditions were the corresponding reference contractions (ramp-hold), ramping up to 45% (Ref 45%), to 30% (Ref 30%) and to 15% MVC (Ref

15%) and maintaining torque at that level, respectively (Fig. 1). All six conditions had the same rate of torque increase as well as reduction, namely 20% MVC/s.

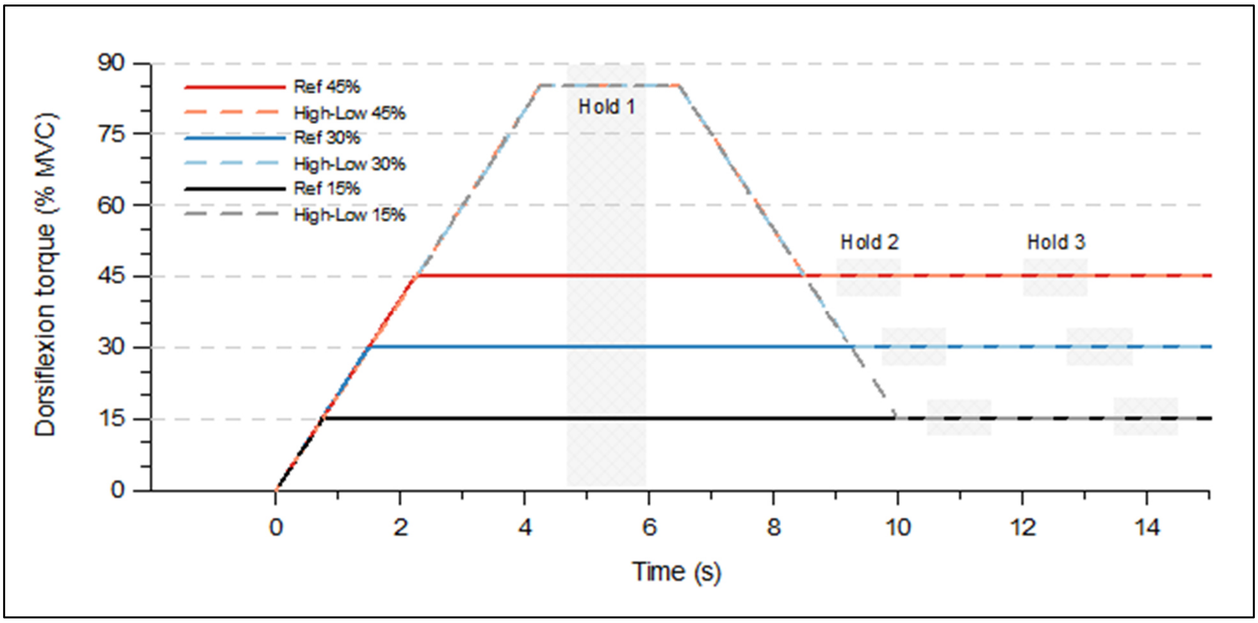

Fig. 1 Torque courses to be matched based on the individual MVC torque

High-Low contractions rise to a high torque level, before returning to the torque level of the corresponding reference contraction (Ref), thus implementing three different magnitudes of torque reduction (see text). Three time-windows representing the initial high steady-state (Hold 1) and the early and late post-reduction steady-states (Hold 2 and Hold 3) from the High-Low contractions were defined for data analysis.

At first, a Ref 45% condition was done, as it served as a reference for the later fatigue test. The participants then performed at least three trials of each of the described conditions, the order of which was randomized, while High-Low and Ref contractions were counter-balanced to minimize possible fatigue. At least two minutes of rest were given between all matching trials, but as much as needed. For each trial, a corridor was drawn on a monitor in front of the participant, the upper/lower line of this corridor being at 4% MVC above/below the particular target torque course of the conditions described above. Participants were then tasked to match the target course with their dorsiflexion effort, staying in the middle of the corridor, as their real-time displayed torque tracing developed. The experimental protocol ended with the performance of another Ref 45% contraction, to assess the fatigue status (see Ch. 2.4).

2.4 Data analysis

All data were processed offline on a personal computer using Spike 2 (Cambridge Electronics Design, 8.17, Cambridge, UK) and custom written scripts in Matlab (Mathworks, 2017b, Natick, MA, USA). Dorsiflexion torques were dual-pass, second-order Butterworth filtered (20 Hz low-pass), and raw TA EMG data had the DC offset removed and were smoothed and rectified with a moving root-mean-square (RMS) calculation (0.25 seconds). Torques and EMGs from all matching trials were normalized to the largest MVC torque and the corresponding maximum TA EMG RMS value, respectively. TA fascicle lengths (FL), determined as the distance from the central to the superficial aponeurosis, and pennation angles relative to the central aponeurosis (PA) were obtained from the ultrasound videos using an updated version of the Matlab add-on UltraTrack, within a semi-automated tracking and digitizing process described elsewhere (Farris & Lichtwark, 2016), the accuracy and reliability of which has been proven (Gillett et al., 2013). In rare cases, when the automatic tracking didn´t precisely match the fascicle or the central aponeurosis, manual corrections were applied.

Mean values of two time-windows were calculated for all test contractions, from 3-1 s prior to ramp onset of the target torque course (rest) and from 4.75-6 s (Hold 1) following ramp onset (Fig. 1). Data was also averaged for two time frames for each of the High-Low contractions and the corresponding reference relative to the end of the reduction phase, that is from 0.5-1.5 s (Hold2) and from 3.5-4.5 s (Hold 3) following the respective steady-state onset (Fig. 1). These rest and Hold time frames allowed for the assessment of changes over the time course of contractions, as well as comparisons between High-Low and corresponding Ref contractions.

Trials were only included in data analysis if they were performed prior to a Ref 45% trial that had the mean EMG RMS amplitude during each of its Holds within 15% relative to the respective Holds of the initially performed Ref 45% contraction, to limit the influence of accumulated fatigue. Also, the maximum torque deviation from the target torque at any point in time, from 5 seconds prior to the ramp start to the end of the target course, wasn´t allowed to be > 10% MVC. Additionally, all trials were excluded in which mean torques during Hold 2 and 3 were more than 2.5% MVC above or below the predefined torque level.

Mean values of all valid trials that were performed per condition were calculated, for all matching contractions (torque, EMG, FL, PA), the calculated rest and Hold means, and the deviations of the produced torque from the predefined course. FL was additionally expressed as the difference to the resting FL prior to each contraction, and for Hold 2 and Hold 3 means, an EMG/torque ratio was calculated.

2.5 Statistics

Statistical analysis of the collected data was done using commercially available software (SPSS v22, IBM Corp., Armonk, NY, USA). Kolmogorov-Smirnov tests were applied to examine data for normality of distribution. T-Tests or Wilcoxon signed-rank tests (for non-parametric data) were used to assess differences between two conditions in Hold means of normalized dorsiflexion forces, normalized EMG, absolute fascicle lengths and pennation angles, and in differences between High-Low and Ref in EMG/torque. Differences across more than two conditions, in Hold means as well as in changes from rest to Hold 1 and from Hold 1 to Hold 2, were assessed using one-way repeated-measures ANOVAs or Friedman tests (for non-parametric data). Mauchly tests for sphericity were applied for the ANOVAs, and if sphericity as violated, a Greenhouse-Geisser correction was applied on the significance of the main effect. When main effects were significant, Bonferroni-corrected t-Tests or Wilcoxon signed-rank tests (for non-parametric data) were used to determine which conditions significantly differed from each other. The level of significance was set at $P \leq 0.05$, and P values were reported rounded to two decimal places.

3 Results

3.1 Valid trials and participant error

Data of two participants were excluded from data analysis, in one case because the recorded sonogram was not evaluable and in the other the predefined torque wasn´t matched closely enough. For the included participants, an average of 2 out of 4 performed trials per condition met the precision criteria described above. Mean deviations from the predefined torque course, from onset to the end of contractions, were 1.0 ± 0.2% MVC and 2.2 ± 0.4% MVC, averaged for the Ref and High-Low conditions, respectively.

3.2 Changes over the time course of test contractions

Mean normalized dorsiflexion torques during Hold 1 were 15.9 ± 0.9% MVC, 30.3 ± 0.9% MVC and 45.5 ± 0.6% MVC for the Ref 15%, Ref 30%, and Ref 45% conditions, and 84.8 ± 2.6% MVC, 83.9 ± 1.6% MVC and 85.0 ± 1.6% MVC for High-Low 15%, High-Low 30% and High-Low 45%, respectively. Accordingly to these torque levels, the increases in mean normalized TA EMG RMS amplitude ($P < 0.01$) and TA pennation angle ($P < 0.01$), and the decreases in TA fascicle length from rest to Hold 1 ($P < 0.01$), significantly differed between Ref conditions (Tab. 1). Also, each High-Low condition had significantly greater changes compared to the Ref conditions from Hold 1 to Hold 2 in EMG (all $P < 0.01$), FL (all $P < 0.01$) and PA (all $P \leq 0.03$), the only exception being that for PA, High-Low 15% did not differ from Ref 45% in this comparison ($P = 0.06$) (Tab. 1). As in all High-Low conditions the same high initial torque level was targeted, there was no significant difference between them in the change from rest to Hold 1 for torque ($P = 0.43$), EMG ($P = 0.16$), FL ($P = 0.32$) and PA ($P = 0.86$).

Tab. 1 Changes in normalized root-mean-square amplitude (RMS amp.) of tibialis anterior (TA) EMG, TA fascicle length and TA pennation angle from rest to Hold 1 and from Hold 1 to Hold 2

	From rest to Hold 1			From Hold 1 to Hold 2		
Contraction Condition	**Δ RMS amp. (% MVC)**	**Δ Fascicle length (mm)**	**Δ Pennation angle (°)**	**Δ RMS amp. (% MVC)**	**Δ Fascicle length (mm)**	**Δ Pennation angle (°)**
Ref 15%	8.3 (2.2) * *	-5.2 (1.7) * *	0.9 (0.5) *	0.3 (0.5) † †	-0.1 (0.2) † †	0.0 (0.1) †
Ref 30 %	14.2 (3.7) * *	-7.7 (2.2) * *	1.2 (0.7) *	1.1 (1.2) † †	-0.1 (0.1) † †	0.0 (0.1) †
Ref 45%	24.0 (5.0) * *	-10.0 (2.5) * *	1.6 (0.9) *	2.2 (0.8) † †	-0.2 (0.2) † †	0.0 (0.1) †
High-Low 15%	70.3 (8.2) * *	-14.1 (3.8) * *	2.0 (1.2)	-60.7 (7.1) †	8.2 (3.1) † †	-1.1 (0.6) †
High-Low 30%	72.4 (8.4) * *	-14.0 (3.5) * *	2.0 (1.0) *	-54.8 (7.8) †	5.8 (1.8) † †	-0.7 (0.4) †
High-Low 45%	74.1 (6.6) * *	-14.3 (3.6) * *	2.0 (1.1) *	-47.7 (6.5) † †	3.8 (1.3) † †	-0.5 (0.4) † †

Values are mean (SD) (n = 10).
* Significantly different to (other) Ref conditions at $P \leq 0.05$ and ** $P \leq 0.01$ after Bonferroni correction.
† Significantly different to (other) High-Low conditions at $P \leq 0.05$ and † † $P \leq 0.01$ after Bonferroni correction.

The torque reductions in the High-Low contractions from Hold 1 to the different Hold 2 levels were accompanied by significantly different decreases in EMG ($P < 0.01$) and PA ($P < 0.01$) and increases in FL ($P < 0.01$) (Tab. 1); the larger the respective torque reduction was, the greater these changes turned out (Tab. 1, Fig. 2). Each Ref contraction also differed in the change from Hold 1 to Hold 2 in comparison with the High-Low conditions, for EMG (all $P < 0.01$), FL (all $P < 0.01$) and PA (all $P \leq 0.03$). Across Ref conditions, the minor changes in torque ($P = 0.29$), FL ($P = 0.19$) and PA ($P = 0.6$) from Hold 1 to Hold 2, where contraction intensity was held at a constant level, were not significantly different. However, EMG changes from Hold 1 to Hold 2 of Ref conditions were different ($P < 0.01$), as Ref 45% showed a significantly greater activation increase than Ref 15% ($P < 0.01$), while Ref 30% did not differ from Ref 15% (P=0.21) and Ref 45% (P=0.11), which shows the greater impact of acute fatigue during Ref 45% contractions (Tab. 1, Fig. 2).

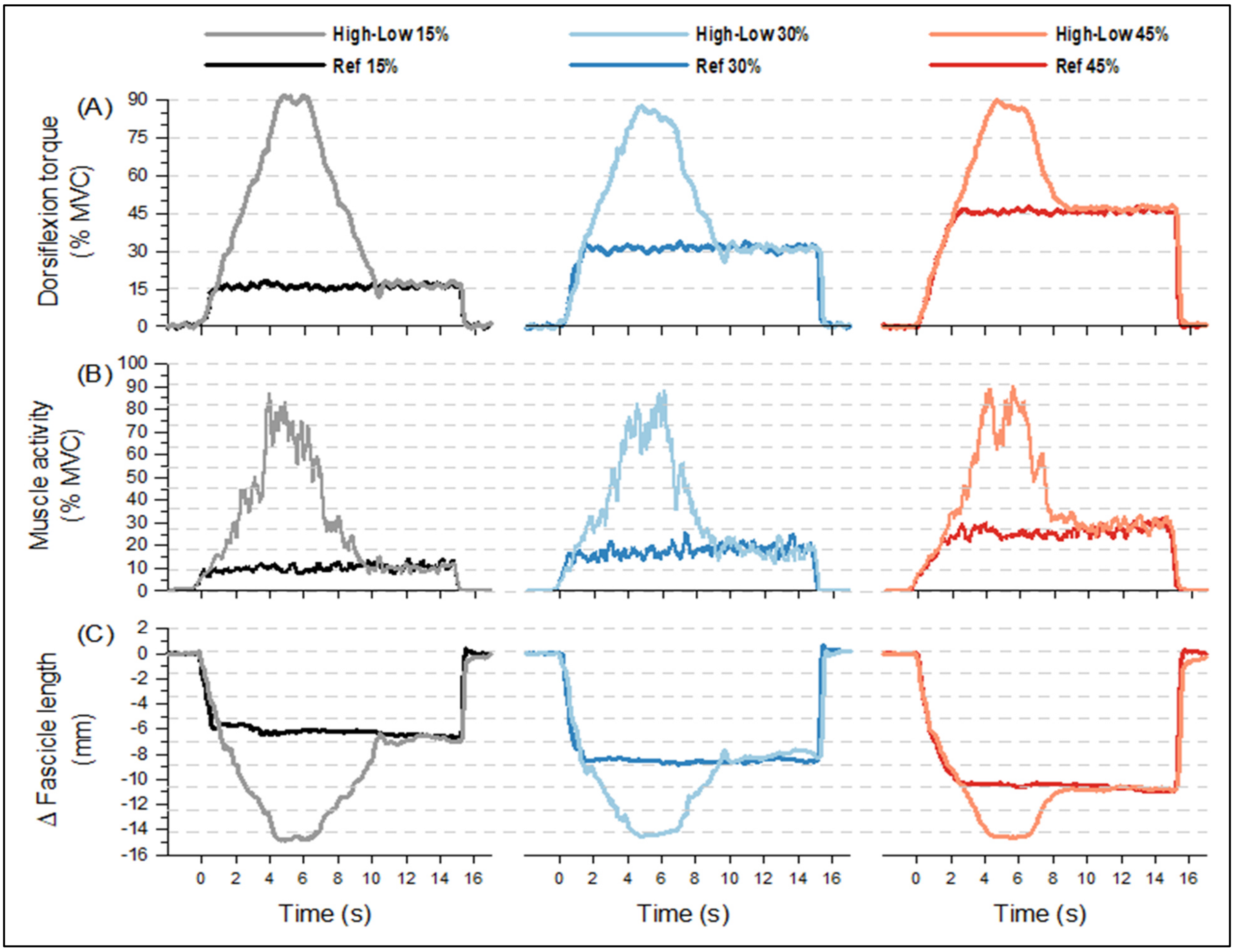

Fig. 2 Time traces of normalized dorsiflexion torque (A), normalized tibialis anterior EMG (B) and tibialis anterior fascicle length changes (C) for one participant during each contraction condition

Data are mean of performed trials per condition. Muscle activity is presented as the root-mean-square amplitude of tibialis anterior EMG, with a 0.25-seconds consecutive moving window calculation. Fascicle length is expressed as the difference to the resting fascicle length prior to contraction onset. Note that after rising to the same high intensity level, all parameters in the High-Low conditions return to a similar level as in the respective reference contraction.

3.3 Comparison between High-Low and Ref contractions during post-reduction steady-states

After torque reduction to the different lower levels, mean normalized torques of High-Low contractions were not different from those of their corresponding Ref contractions during Hold 2 (P = 0.12, P = 0.06, P = 0.06, for the 15%, 30% and 45% MVC level, respectively) and Hold 3 (P = 0.38, P = 0.14, P = 0.1) (Fig. 2, 3). Similarly, mean normalized TA EMG RMS amplitudes were not different between High-Low and Ref conditions at any of the intensity levels during Hold 2 (P = 0.06, P = 0.06, P = 0.8, for the 15%, 30% and 45% MVC level, respectively) and Hold 3 (P = 0.46, P = 0.54, P = 0.34) (Fig. 2, 3). Absolute TA fascicle lengths (all $P \geq 0.27$) and pennation angles (all $P \geq 0.44$) in the High-Low conditions also returned to levels at both Hold 2 and 3 that were not different from the corresponding Ref contractions, the only exception being that FL of High-Low 45% was significantly lower than that of Ref 45% during Hold 2 (mean difference 0.3 mm, 95% CI 0.0 to 0.6 mm; *P* = 0.03) (Fig. 3).

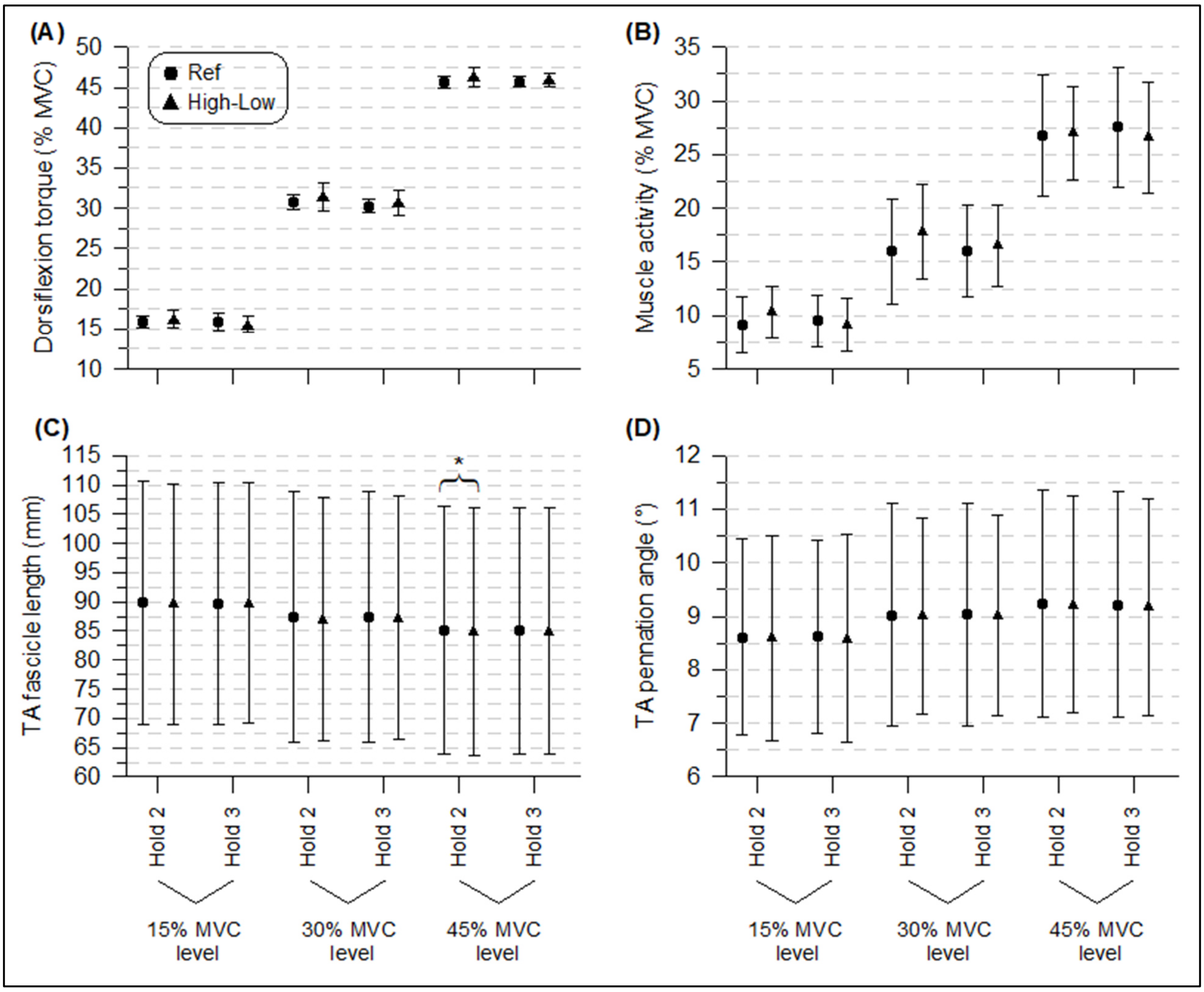

Fig. 3. Mean normalized dorsiflexion torques (A), mean normalized tibialis anterior (TA) root-mean-square EMG (B), mean TA fascicle lengths (C) and mean TA pennation angles (D) during Hold 2 and Hold 3 at each torque level

Circles and triangles represent the mean values during each Hold for Ref and High-Low conditions, respectively, bars represent SD (n = 10). Significant differences at $P \leq 0.05$ between Ref and High-Low values of each Hold were marked (*). Note that at each targeted torque level and Hold, High-Low contractions yield similar values as Ref contractions (with one exception in FL).

The mean dorsiflexion torques during Ref Hold 2, High-Low Hold 2, Ref Hold 3 and High-Low Hold 3 were not different on the 15% MVC (*P* = 0.12) and 45% MVC level (*P* = 0.07), but on the 30% MVC level (*P* < 0.01) they were, as High-Low Hold 2 torque differed significantly from that of Ref Hold 3 (*P* = 0.04) and High-Low Hold 3 (*P* = 0.05). Therefore, to make contractile outputs comparable between hold 2 and 3, an EMG/torque ratio was calculated for both Holds in each contraction condition. The differences of these ratios between High-Low Hold 2 and Ref Hold 2 were then compared to the differences between High-Low Hold 3 and Ref Hold 3 for each torque level, and they were not different for the 30% ($P = 0.14$) and 45% MVC ($P = 0.24$) intensity levels. At the 15% MVC level, however, High-Low Hold 2 yielded a ratio that was 0.1 EMG/torque higher than Ref Hold 2 (meaning that the contractile output was lower), and this was different ($P = 0.01$) compared to Hold 3, where the difference between High-Low and Ref was 0.0 EMG/torque.

4 Discussion

The purpose of this study was to investigate changes in muscle architecture and activity of the human TA during fixed-end dorsiflexions, which involve varying magnitudes of torque reduction proceeding from a high submaximal torque level, and the effects of the varying contraction histories on the post-reduction contractile outputs. As expected, the greater the torque reduction was in the High-Low contractions from Hold 1 to the different lower Hold 2 levels, the more the fascicles shortened and rotated. Also, during the early and late post-reduction steady-states at each of the lower levels, muscle activities of the High-Low contractions were similar to those of the Ref contractions. This means that regardless of how much additional torque increase and subsequent reduction was involved, the different contraction histories of High-Low contractions haven´t altered the contractile outputs compared to the Ref conditions.

4.1 RFD and AI in fixed-end contractions

To reconstruct the emergence of the results based on the proposed effects of contraction history, it is important to consider the additional contractile depression that High-Low contractions likely experienced during the high initial torque increase, compared to their corresponding Ref conditions. Raiteri & Hahn (2019) indirectly demonstrated that rFD and AI, which have typically been shown in MTU-shortening contractions with changes in joint angles (see Chen et al., 2019 for review), can also be established during ramp phases of fixed-end contractions, induced by the concomitant active shortening of muscle fibers. They showed a long-lasting higher rFD level to be present in a fixed-end dorsiflexion of 20% MVC TA activation, by comparing its produced force with that of a contraction to the same activation level, that but included a slight increase in ankle angle during the ramp phase before matching the angle of the fixed-end contraction. With this imposed MTU stretch, TA fascicles shortened 19.8% less in arriving at the target activation level, and the steady-state force 10.5-13 s following contraction onset was increased by 9.6% relative to that of the fixed-end contraction (Raiteri & Hahn, 2019). Furthermore, in a likewise designed force-controlled experiment with similar contraction intensities, the ramp-and-hold contraction with reduced fascicle shortening yielded a steady-state activation that was 13.9% lower relative to the fixed-end contraction, which shows the establishment of greater AI due to greater shortening in the fixed-end contraction (Raiteri & Hahn, 2019).

RFD has been found to increase with the amount of muscle shortening (Edman, 1975; Herzog & Leonard, 1997; Lee & Herzog, 2003; Rousanoglou et al., 2007), and to strongly correlate with the amount of mechanical work (force x displacement) produced during shortening (Granzier & Pollack, 1989; Herzog et al., 2000; Josephson & Stokes, 1999; Kosterina et al., 2009; Leonard & Herzog, 2005). Raiteri & Hahn (2019) found the above mentioned higher rFD/AI levels being induced by a further mean fascicle shortening of 1.7 mm. They therefore concluded for the additional fascicle shortenings of ~1.5 - 2 mm in the force rises to 60/30% MVC (High-Mod conditions) compared to the force ramps to 40/20% MVC (reference conditions), that these would also result in a detectable rFD/AI level. It is plausible that this can similarly be applied to the different additional torque increases of the present study.

In the Ref contractions, muscle activity initially rises to the level that is necessary to produce the respective targeted torque level (i.e., 15%, 30% or 45% MVC). Thereby, a subset of motor units (MUs) is activated in a specific order, once the respective recruitment threshold of each motor unit is reached (Awiszus & Feistner, 1999; Henneman, 1957; Milner-Brown et al., 1973). Thus, once the targeted torque level is arrived at, the associated fibers of the now activated motor units should have experienced AI proportional to their respective fascicle shortening and exerted force contribution since their respective activation onset, which constitute their performed work. In each of the High-Low contraction ramps, activation rises above the corresponding Ref level so that a torque of 85% MVC can be matched, and motor units of higher thresholds are thereby additionally being recruited, while the previously activated fibers continue to shorten, building up additional AI. This AI is additional to the AI established corresponding to the respective Ref level, and it is likely to be lowest in the High-Low 45%, medium in the High-Low 30%, and largest in the High-Low 15% condition, respectively. This is because of the additional mean fascicle shortenings of 4.3 mm, 6.3 mm and 8.9 mm found in the respective High-Low conditions, compared to their corresponding references (Tab. 1).

4.2 Different torque reductions cancel out corresponding additional AI levels

When in the High-Low conditions following Hold 1 activation is reduced to lower torque, derecruitment of motor units takes place in reverse order as they were previously recruited (Henneman et al., 1965; Lauber et al., 2014; Pasquet et al., 2006). Once the Hold 2 torque level – which is the torque level of the respective reference – is reached, only the fibers that were active in the late hold phase of the respective reference should still be activated (Johnson et al., 2017). It is likely that these fibers – that have previously established the above-mentioned additional AI – have by that time been actively lengthened back to a similar length as the fiber length of the corresponding

Ref contraction (Johnson et al., 2017) (Tab. 1). Thus, their respective magnitude of active lengthening is greater, the lower the targeted Hold 2 torque level is. The above made reconstruction of torque increases and reductions is plausible for TA, as this muscle has an upper limit of recruitment thresholds of 90% MVC (Van Cutsem et al., 1997), and thus the various torque levels of this experiment should have been produced by specific corresponding subsets of MUs.

As the post-reduction EMG matched the Ref EMG at each of the intensity levels, it seems that regardless of the magnitude of previously established additional AI, in all cases it was completely abolished by the time that torque has returned to the reference level. This parallels with the study of Raiteri & Hahn (2019), where a complete abolishment occurred, while the force difference between high and low steady-states was smaller (20% MVC) than in the present study (40%, 55% and 70% MVC). That means that the larger this difference was, the more AI-diminution must have occurred, when it is assumed that the additional AI rose with greater differences between the high and low level, as discussed above.

4.3 Possible AI-reducing mechanisms

Raiteri & Hahn (2019) proposed that one of the factors that have potentially diminished the higher level of contractile depression was that the muscle fibers which were active during the post-reduction steady-state have experienced active lengthening during torque reduction. This might have elicited rFE-related mechanisms (Edman, 2012; Herzog & Leonard, 2013; Herzog, 2014, 2018), which underlie stretch-induced rFE and AR on the sarcomere level, such as an increase in the average force of each cycling cross bridge (Leonard et al., 2010; Mehta & Herzog, 2008) or an increase in the number of cycling cross bridges (Brunello et al., 2007; Linari et al., 2000; Mehta & Herzog, 2008) induced by active stretch. Furthermore, non-uniformity of sarcomeres (Edman, 2012; Morgan, 1990) or half-sarcomeres (Campbell et al., 2011) were assumed to account for rFE. The mechanisms related to cross bridge kinetics are thought to be only partly and transiently enhancing post-stretch steady-states, which can be seen as a decay in rFE that lasts up to ~ 10 seconds following steady-state onset (De Ruiter et al., 2000; Herzog et al., 2006; Noble, 1992; Pinniger & Cresswell, 2007). For-long lasting rFE, the contribution of other mechanisms involving the stretch-induced stiffening of the inner-sarcomere titin were found to be responsible (Duvall et al., 2013; Joumaa et al., 2008a; Leonard & Herzog, 2010; Nishikawa et al., 2012; Schappacher-Tilp et al., 2015), as well as for increased passive forces in the resting state following lengthening contractions (Herzog & Leonard, 2002; Joumaa et al., 2008b). In the present experiment however, the differences between contractile outputs of High-Low and Ref contractions during Hold 2 were not different than during Hold 3 on the 30% and 45% MVC level, and on the 15% MVC level, High-Low contractile output at Hold 2 was lower than that of Ref, which significantly differed from the minor difference between High-Low and Ref during Hold 3. Also, as in the study of Raiteri & Hahn (2019), no increases in passive forces were seen following High-Low compared to Ref contractions. Thus, no indications for the influence of rFE-related mechanisms can be drawn from these results, but their contribution cannot be ruled out either and it may be that it is merely too small to be detected with the applied methods (see Raiteri & Hahn, 2019). RFE rises with stretch amplitude in animal muscle preparations and electrically stimulated muscles in vivo (Abbott and Aubert, 1952; Cook & McDonagh, 1995; De Ruiter et al. 2000; Edman et al., 1978), so if it was a factor here, it could have been conducive to greater torque reduction inducing more AI-diminution. However, this correlation has not been consistently observed in voluntary lengthening contractions (Hahn et al., 2007; Lee & Herzog, 2002; Tilp et al., 2009), and it is unclear how different stretch amplitudes would be effective in voluntary fixed-end contractions. Generally, it is questionable if even the highest fascicle stretch amplitude of the present experiment (8.2 mm) was able to induce contractile enhancement to the extent as for example the rFE found with an TA MTU stretch induced by 15° (~10 mm) or 30° (~18 mm) increase in ankle angle during maximal dorsiflexions (Tilp et al., 2011).

A promising AI-reducing factor is that the high contraction intensity attained during Hold 1 might have induced a state of potentiation, which increased the subsequent contractile output at the lower steady-state (Raiteri & Hahn, 2019). Potentiation has previously been demonstrated as an increased twitch contractile response following tetanic stimulation (Brown & von Euler, 1938), as well as following maximal voluntary contraction (postactivation potentiation) (Baudry & Duchateau, 2007; Hamada et al., 2000). Also, high-frequency conditioning stimulations increased subsequent tetanic force production on lower activity levels (Baldwin et al., 2006; MacIntosh & Willis, 2000; Neyroud et al., 2016). Various mechanisms were proposed to be responsible for these effects, including central and spinal factors (Bergquist et al., 2011; Collins, 2007). In terms of intramuscular mechanisms, activation-induced phosphorylation of myosin regulatory light chains, which increases Ca2+-sensitivity of the actin-myosin interaction, is thought to be responsible for the twitch or low-frequency potentiation (Grange et al., 1993; Sweeney & Stull, 1990; Vandenboom et al., 1993). Yet, such increases in lower-level Ca2+-sensitivity were also proposed to be caused by greater initial crossbridge interaction itself, without being mediated by myosin phosphorylation (Cheng et al., 2017). Oskouei & Herzog (2009) investigated lower-level potentiation in the context of fixed-end, voluntary thumb adductions, where activation level of m. adductor pollicis was reduced from 60% and 100% to 30% MVC. The result was that the higher initial contraction intensity induced greater low-level force increase, but some of the subjects didn´t show any potentiation. A reason for the increased forces could be that muscle shortening during the initial force increase would be minor in the adductor pollicis MTU due to its short series elastic component, and the resulting lower additional rFD levels would not only be eradicated by torque reduction, but potentiation would be large enough to even increase post-reduction contractile output compared to the reference, in contrast to what happens in dorsiflexions. However, the findings of Oskouei & Herzog (2009) also emphasize the interindividual differences in voluntary lower-level potentiation, which were also seen in the study of Raiteri & Hahn (2019) as well as in the current experiment.

Since previous studies on potentiation have shown that lower intensity levels are potentiated to a greater relative extent, as Ca2+-sensitivity increases are more effective at low myoplasmic calcium concentrations (Abbate et al., 2000; MacIntosh & Willis, 2000; Rassier & MacIntosh, 2000; Vandenboom et al., 1993), it could be that the lower Hold 2 levels have experienced greater relative enhancement, and this might explain why greater torque reductions apparently induced more AI-diminution. However, some studies found that subjects or muscles with higher percentage of type II fibers show greater postactivation potentiation (Hamada et al., 2000; O´Leary, 1997), and this might contradict the idea that the MUs which are active at the lower levels and therefore have more type I fibers, exhibit greater potentiation. However, this correlation between fiber type distribution and potentiation wasn´t always found (Stuart, 1988). It is unclear if the discussed manifestations of potentiation at various tetanic low-levels similarly apply to voluntary contractions, and what role other potentiating mechanisms like central or spinal contributions play in this context. Raiteri & Hahn (2019) argued that it is difficult to determine the possible application or contribution of the proposed low-frequency potentiating mechanisms for voluntary contractions in which the number of active fibers changes instead of being constant.

4.4 Limitations

Despite the preceding training session, participants showed large deviations of up to 10% MVC from the predefined torque courses in some cases, especially during the great torque rises and reductions in the High-Low conditions, and at the transitions from steady-state to torque increase or decrease and vice versa. On one hand, this could have biased the amount of active fascicle lengthening and contractile enhancement during torque reduction, as participants might have completely deactivated the TA muscle in some cases to follow up with the quick intensity drops or perform very fast active lengthenings referred to as slippage (Fukutani et al., 2019). Then, at the transition to the post-reduction steady-state, a greater EMG impulse might have been necessary to keep torque from falling further, that could also have increased Hold 2 EMG means. These aspects might explain why no decay in contractile output from Hold 1 to Hold 2 was observed, which would be typical for the contribution of rFE-related mechanisms. Also, participant error might have influenced the duration of the initial high-steady state (predefined as lasting 2.25 seconds), which potentially alters the magnitude of potentiation (Tillin & Bishop, 2009). However, as contraction intensities during Hold 1 were not different across High-Low conditions, it is unlikely that a systematic bias has influenced conditions differently, or in a way that preceding contractile depression would have directly diminished subsequent lengthening-induced enhancement, which is thought to only occur if the time interval between shortening and stretch is smaller than ~1 second (Edman et al., 1982; Fortuna et al., 2016; Rassier & Herzog, 2004).

The different rises in activity that occurred from Hold 1 to Hold 2 in the Ref 45% compared with the Ref 15 % condition indicate that acute fatigue was present during contractions. Yet, it is unlikely that acute fatigue has increased activities of the High-Low Hold 2 steady-states to a different extent than those of the corresponding Ref steady-states, as specific subsets of MUs were likely active corresponding to each lower torque level, which were activated to a similar extent over the course of contractions.

It is unclear why FL of High-Low 45% was significantly lower by 0.3 mm than that of Ref 45% during Hold 2, although torques were not different. Raiteri & Hahn (2019) used the same ultrasound system and fascicle tracking method as were used in this study, and they stated that it should be able to detect sub-millimeter fascicle length changes, referring to fascicle measurements of previous studies (Day et al., 2013; Day et al., 2017). However, the tracking process sometimes showed a tendency towards underestimation of FL after the fascicle has shortened and then lengthened, and it could be that manual corrections haven´t precisely compensated for this effect in every case.

Regarding the unlikelihood of changes in synergistic activation across conditions, of antagonist contribution to measured forces and EMGs, and of force fluctuations or fascicle length changes induced by differences in TA moment arm or ankle joint rotation, the reader is referred to the remarks of Raiteri & Hahn (2019) concerning these aspects, which likewise apply to the present experiment.

4.5 Implications

The findings of this study point towards a possible mechanism that in relatively compliant MTUs like that of TA, different intensity reductions induce just the right magnitude of contractile re-enhancement necessary to cancel out previous according depression levels. This could be beneficial for precise motor regulation in different tasks of compliant MTUs, as the contractile output stays similar regardless of preceding intensity changes – at least as long as the MTU-length stays constant. This is also consistent with findings of Raiteri & Hahn (2019), where torque reduction was smaller, preceding from a lower initial intensity level. The higher Hold 1 intensity level in the current experiment should have induced greater relative potentiation (Oskouei & Herzog, 2009; Skurvydas et al., 2019), yet, additional shortening relative to the reference levels was also greater here, and the corresponding higher levels of additional AI have been compensated by the greater potentiation. Thinking of this aspect, it could be that in the context of fixed-end intensity level change, the difference between high and low levels (i.e. the reduction magnitude) is actually a good estimate for the overall influence on the post-reduction contractile output, rather than initial and/or final intensity level, but this remains to be seen.

As discussed above using the example of m. adductor pollicis, the effect of force reduction on post-reduction steady-states could turn out differently in other muscles, due to different MTU compliances – specifically other tendon-to-fiber length ratios, or other activity-force relationships. While the contribution of the different proposed AI-reducing mechanisms remains unclear for the contractions of the present study, it may be that under different circumstances the influence of rFE-related mechanisms might be detectable, for example with intensity reductions following maximal contractions in very compliant MTUs. Also, the method of manipulating fascicle length changes during the initial force rise applied by Raiteri & Hahn (2019) may be used to get a clearer picture regarding the effects of different intensity increases and decreases. On one hand, the depressing effect of different shortenings may be assessed in this way; on the other hand, the enhancing effect of intensity reductions could be isolated, by performing them following "truly isometric" force rises, in which muscle shortening was eliminated.

Regarding the transfer to everyday human movement, Raiteri & Hahn mentioned two examples where force reduction might play a beneficial role. One was the fascicle lengthening induced by activity reduction of m. tibialis posterior during the early stance phase of gait, while the MTU length remains relatively constant (Maharaj et al., 2016); the other the strategy that humans apply to grasp objects, where the applied force initially peaks and is then being slightly reduced to reach a steady-state (Nowak et al., 2003). In these examples, force reductions are rather small, and it is questionable if large intensity decreases like the ones tested here occur on a regular basis in everyday human movements. It has to be shown if active lengthening plays a crucial role in tasks like grasping, where the involved muscles are commonly used for direct, fine-tuned force transmission, or if this is more relevant for example in locomotion, where more compliant MTUs of the lower limbs are engaged in the storage and release of large forces. As the role of muscle length changes in the context of fixed-end contraction history has largely been overlooked in previous studies (Chen et al., 2019; Raiteri & Hahn, 2019), to advance biomechanical models future research should focus on the in-concert action of rFD- and rFE-related mechanisms during fixed-end contractions, while also considering the influence of potentiation mechanisms.

4.6 Conclusion

In the fixed-end voluntary dorsiflexions of this study involving high initial torque rises and subsequent reduction to different lower levels, TA EMGs during early and late post-reduction steady-states matched the EMGs of corresponding reference contractions without intensity reduction. Thus, it appears that the higher levels of AI which likely rose with additional torque increase above the reference level, were in each case canceled out by the correspondingly greater torque reductions, with larger reductions inducing greater relative enhancement.

Even with the largest stretch amplitude of TA fascicles during torque reduction, no typical indicator for the involvement of rFE-related mechanisms was found. Therefore, it might be that potentiation mechanisms – triggered by the high initial contraction intensity – played the dominant role in abolishing additional AI, and lower-post-reduction steady-states might have been enhanced to a greater relative extent because of higher susceptibility to potentiation. Further research is necessary regarding the depressing and enhancing mechanisms involved in fixed-end torque changes, and how different conditions determine their contribution.

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