European Journal of Applied Physiology (2019) 119:2287–2299
https://doi.org/10.1007/s00421-019-04212-8

ORIGINAL ARTICLE

The loss of muscle force production after muscle stretching is not accompanied by altered corticospinal excitability

Timothy S. Pulverenti[1] · Gabriel S. Trajano[2] · Benjamin J. C. Kirk[1] · Anthony J. Blazevich[1]

Received: 18 March 2019 / Accepted: 14 August 2019 / Published online: 27 August 2019

Abstract

Purpose The aim of the present study was to determine whether depression of maximal muscular force and neural drive subsequent to prolonged ($\geq$60 s) passive muscle stretching is associated with altered corticospinal excitability or intracortical ($GABA_B$-mediated) inhibition.

Methods Fourteen healthy adult males were tested before and after 5 min (5×60-s stretches) of intense, passive static stretching of the plantar flexor muscles. Two protocols (A and B) were conducted in a randomized order. Transcranial magnetic stimulation was delivered to the contralateral motor cortex at rest (Protocol A) and during maximal voluntary contractions (Protocol B). Changes in maximal voluntary isometric torque, voluntary surface electromyographic activity of triceps surae muscles (normalized to M-wave; EMG/*M*), motor-evoked potentials (MEP), and cortical silent period (cSP; Protocol B) in soleus elicited by transcranial magnetic stimulation were examined 10 min after stretch.

Results In both protocols A and B, significant decreases were observed immediately after stretching in maximal voluntary plantar flexion torque ($-20.1 \pm 15.9\%$, $P=0.004$; and $-17.2 \pm 13.5\%$, $P=0.006$) and EMG/*M* ($-18.0 \pm 18.2\%$, $P=0.023$; and $-13.0 \pm 9.3\%$, $P=0.003$). Decreases in torque and EMG/*M* were highly correlated ($r=0.67$–0.85, $P<0.05$). However, no changes were observed in MEP amplitudes during rest ($+29.3 \pm 50.0\%$) or maximum voluntary contraction ($+1.9 \pm 16.8\%$), or in cSP ($+2.1 \pm 15.1\%$).

Conclusions Impaired neural drive contributed to the stretch-induced force loss; however, changes in corticospinal excitability and intracortical inhibition could not explain the phenomenon.

Keywords Static stretching · Maximum voluntary contraction · Transcranial magnetic stimulation · Corticospinal excitability

Abbreviations

CI	Confidence interval
cSP	Cortical silent period
EMG	Electromyography
ICC	Intraclass correlation
MANOVA	Multivariate analysis of variance
MEP	Motor-evoked potential
MG	Medial gastrocnemius
MG_{RMS}	Medial gastrocnemius root-mean square
M_{max}	Maximal motor-wave
MSO	Maximum stimulator output
MVC	Maximal voluntary contraction
RMS	Root-mean square
RMT	Resting motor threshold
SOL	Soleus
SOL_{RMS}	Soleus root-mean square
TA	Tibialis anterior
TA_{RMS}	Tibialis anterior root-mean square
TMS	Transcranial magnetic stimulation
T_{peak}	Peak torque
TS	Triceps surae

Communicated by Olivier Seynnes.

✉ Anthony J. Blazevich
a.blazevich@ecu.edu.au

[1] Centre for Exercise and Sports Science Research (CESSR), School of Medical and Health Sciences, Edith Cowan University, 270 Joondalup Drive, Joondalup, WA 6027, Australia

[2] School of Exercise and Nutrition Sciences, Institute of Health and Biomedical Innovation, Queensland University of Technology (QUT), Brisbane, Australia

Introduction

After prolonged (e.g. ≥ 60 s) stretching of relaxed (i.e., passive) human skeletal muscle held at a (near-) constant length causes a short-lasting, but significant, reduction in maximal voluntary force and power production (Kay and Blazevich 2012; Simic et al. 2013; Behm et al. 2016). There is strong evidence that this stretch-induced impairment of force output results predominately from decreases in neural drive to the muscle (Behm et al. 2001; Trajano et al. 2017). This proposition has been corroborated by the simultaneous and highly correlated stretch-induced decreases (and then recovery) in voluntary force and both electromyogram (EMG) and V-wave amplitudes and voluntary muscle activation assessed using the interpolated twitch technique (Fowles et al. 2000; Behm et al. 2001; Kay and Blazevich 2009; Trajano et al. 2013, 2014a). Potentially, the reduced neural drive may be caused partly by a stretch-induced reduction in spinal motoneuron facilitation mediated by a withdrawal of excitatory muscle spindle feedback (Trajano et al. 2014b). However, it is not known if other parallel changes within the central nervous system after prolonged stretching contribute to the reduction in neural drive, because the methods used in the previous studies have not targeted specific sites or processes within the nervous system (e.g., corticospinal processes).

One possibility is that decreases in neural drive after stretching result from stretch-induced reductions in corticospinal excitability that outlast the period of muscle stretch. Corticospinal excitability, describing the ability of the corticospinal–motoneuronal pathway to receive, initiate, and transmit excitatory synaptic input, is particularly sensitive to the net excitation and inhibition from sensory/afferent feedback (Chen 2004) and can be estimated by measuring the amplitude/area of motor-evoked potentials (MEPs) elicited non-invasively by transcranial magnetic stimulation (TMS; Rothwell 1997; Rothwell et al. 1999). Several sensory/afferent receptors are responsive to slow, passive muscle stretch, depending on stretch amplitude (Stuart et al. 1970; Kniffki et al. 1978; Burke et al. 1988; Gandevia 2011), and can influence corticospinal excitability through direct and trans-cortical projections to cortical motor areas (Hore et al. 1976; Wiesendanger and Miles 1982; Huerta and Pons 1990). Indeed, during slow, passive cyclic stretches of upper and lower limb muscles there are muscle length-dependent reductions in MEP amplitudes, which return to pre-stretch levels once stretch is removed (Guissard et al. 2001; Coxon et al. 2005; Chye et al. 2010). These decreases in MEP amplitude become more clear during large amplitude stretches similar to those imposed during standard static stretching protocols (i.e., near maximum joint range of motion; Lewis et al. 2001; Guissard and Duchateau 2006). Of interest is that sustained somatosensory input through electrical stimulation (Veldman et al. 2014) or stretch-related forms of sensory feedback such as tendon/muscle vibration and acute muscle pain (Kniffki et al. 1978; Gandevia 2011) can induce similar, but short lasting, depressive effects on MEP amplitude and voluntary motor output after stimuli were removed (Marconi et al. 2008; Burns et al. 2016). It is not yet known, however, whether the stretch-dependent reduction in MEP amplitude (i.e., corticospinal excitability) previously observed during muscle stretching is exacerbated by prolonged muscle stretches and outlasts the period of stretch and contributes to impairment in voluntary neural drive to the muscle during subsequent muscle contractions.

When TMS is superimposed on a voluntary contraction, there is a period of EMG silence following the MEP, known as the cortical silent period (cSP), with at least the latter part (> 50 ms) of the cSP reflecting a form of intracortical inhibition of cortical motor output in both upper and lower limbs, which is thought to be mediated by gamma-aminobutyric acid $(GABA)_B$-receptors (Ziemann et al. 1993; Siebner et al. 1998). The effects of sustained stretch-induced somatosensory feedback on the cSP, and thus intracortical inhibitory mechanisms, are unknown. During muscle vibration, which selectively activates stretch sensitive Ia afferent feedback, $GABA_B$-mediated intracortical inhibition increases, as indicated by larger long-interval intracortical inhibition (LICI; Rosenkranz and Rothwell 2003). However, sustained somatosensory electrical stimulation does not appear to alter $GABA_B$-mediated inhibition, with no changes in either cSP or LICI being observed (Veldman et al. 2014). Nonetheless, the effect of prolonged stretching and its resulting sensory/afferent feedback on cSP is unknown.

The purpose of the present study was to investigate stretch-induced changes in corticospinal excitability and intracortical inhibition which were assessed by measuring MEP amplitude and cSP duration, respectively. Resting measurements were made to examine the balance of inhibition and excitation within the corticospinal pathway without the confounding influences of afferent feedback or volitional drive, and then active measurements were made to test the pathway under modulatory influences, e.g., from afferent pathways that are activated during muscle contractions similar to those during voluntary muscle contraction. It was hypothesized that acute passive muscle stretching would reduce corticospinal excitability, evidenced by decreased MEP amplitudes and prolongation of the cSP and that these changes would coincide and correlate with reductions in both plantar flexor voluntary peak torque and maximal voluntary EMG amplitudes.

Methods

Subjects

Fourteen recreationally trained men (mean ± SD: age, 28.5 ± 4.7 years; height, 1.76 ± 0.59 m; body mass, 80.5 ± 12.9 kg) volunteered to participate. All participants completed a Physical Activity Readiness Questionnaire (PAR-Q) and a TMS Readiness Questionnaire and reported no lower limb neuromuscular disorders or contraindications to the safe use of TMS (i.e., no neurological disorders, medical implants, or use of medications that alter neuronal activity). All participants reported no engagement in flexibility-training programs for at least 6 months prior to the study and refrained from muscle-stretching activities during data collection. Written informed consent was obtained from all participants and all procedures in the study were conducted in accordance with the Declaration of Helsinki and were approved by the Edith Cowan University Human Research Ethics Committee.

Study overview

Participants visited the laboratory at the same time of day on three different occasions, separated by at least 48 h. Participants were asked to abstain from consuming stimulants and depressants for 12 h and from performing intense exercise for 48 h, prior to all visits. The first visit included a full familiarization of the experimental procedures, including: (1) plantar flexor muscle stretching; (2) TMS; (3) tibial nerve stimulation; and (4) maximal voluntary isometric contractions (MVC) of the plantar flexor muscles. At each of the two subsequent visits, participants performed one of the two different protocols that were randomly allocated, *Protocol A* and *Protocol B* (explained below), and two experimental conditions were completed in each session: (1) a 5-min passive rest control period and (2) a prolonged static muscle-stretching protocol (Fig. 1).

During the familiarization session, participants completed three maximum tolerable passive stretches of the plantar flexors to measure maximum ankle dorsiflexion range of motion and maximum passive joint torque. This followed a standardized warm-up of 5 min of stationary cycling at 60 rpm with a 1-kg load followed by five voluntary isometric plantar flexions in the dynamometer ranging from

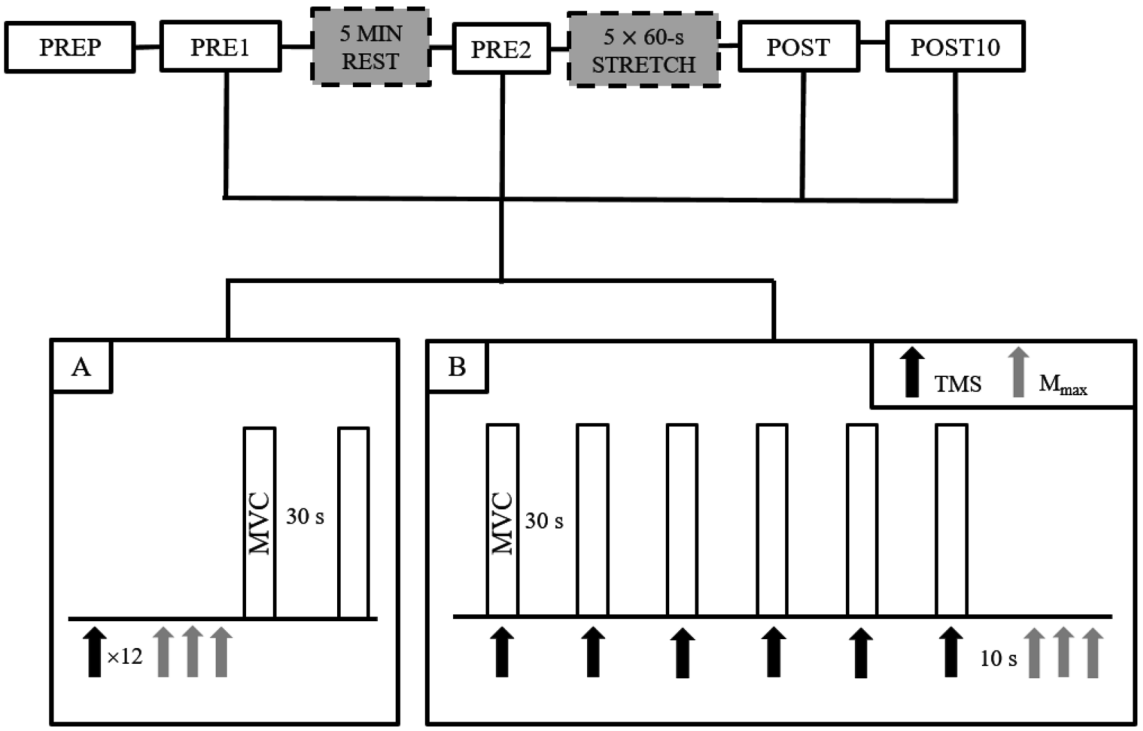

Fig. 1 Study 1 design. Participants completed PREP, which included participant setup and defining stimulus parameters, and then tested immediately before (PRE1) and after (PRE2) a 5-min control period and immediately (POST) and 10-min after (POST10) stretching (STR; 5×60-s stretching). *Protocol A*: 12 resting TMS and three M_{max} measurements were completed at an ankle joint angle of 0° (neutral) followed by two isometric plantar flexion MVCs. *Protocol B*: six MVCs; superimposed with TMS (MEP and SP); and three resting M_{max} measurements

20 to 100% perceived maximum effort with 30 s of rest between contractions. Two-minute rest intervals separated each stretch. The greatest maximum passive joint torque was used to calculate the stretch intensity for all participants (see "Stretching protocol"). Protocols A and B included a preparation period consisting of the standardized warm-up followed by the determination of stimulation sites and intensities for TMS (see "Transcranial magnetic stimulation") and tibial nerve stimulation (see "Tibial nerve stimulation"). Participants were then assessed immediately before (PRE1) and after (PRE2) the control period. Following a 5-min rest after PRE2, participants completed the stretching protocol and were then assessed immediately (POST) and 10-min (POST10) after stretching. All measurements were completed on the plantar flexors of the right leg, while seated in a slightly reclined position in an isokinetic dynamometer (Biodex System 4 Pro, Biodex Medical Systems, NY, USA). The knee joint was fully extended (0°), ankle joint in a neutral position (0°), so the sole of the foot was perpendicular to the shank, and the lateral malleolus aligned with the centre of rotation of the dynamometer. The upper body, knee, and foot were firmly secured to the dynamometer with straps and the contralateral leg rested on a stool to avoid unwanted movements. Visual feedback of torque data was provided by a television monitor placed ~ 2 m in front of participants. All data were recorded synchronously at 2000 Hz on a personal computer running LabChart software (version 7.1.3, ADInstruments, NSW, Australia) using a 16-bit analogue-to-digital converter (PowerLab 16/35, ADInstruments).

Protocol A At each timepoint (PRE1, PRE2, POST, POST10), 12 single TMS pulses were applied at 0.2 Hz (5-s inter-stimulus interval) with the participants relaxed followed by three supramaximal tibial nerve electrical stimulations to elicit maximal motor wave responses (M_{max}; 10-s inter-stimulus interval). A larger number of MEPs may have provided higher test reliability. However, this would also have increased the time required for the measurements to be taken, which is problematic in studies like the present one where neurophysiological changes are expected to occur rapidly. Thus, we increased sample size above that used in other studies in order to maintain statistical power. Participants also performed two isometric plantar flexion MVCs following stimulation procedures, a 30-s passive rest separated each MVC. A total of sixty stimulations, including TMS (total = 48) and tibial nerve (total = 12), were imposed in each session.

Protocol B At each timepoint, the participants performed six 3-s isometric MVCs of the plantar flexors with a single-TMS pulse superimposed on each isometric MVC when voluntary torque reached a plateau (30-s rest intervals between MVCs) followed by three supramaximal tibial nerve stimulations at rest to elicit M_{max} (10-s inter-stimulus interval). A total of 36 stimulations, including TMS (total = 24) and tibial nerve (total = 12), were imposed in each session.

Stretching protocol

Passive stretching of the plantar flexors was performed in the isokinetic dynamometer. While relaxed, the plantar flexors were stretched by rotating the ankle joint at $5°s^{-1}$ into dorsiflexion from a 15° plantar flexion position until a passive torque resistance equal to 90% of the maximum tolerable passive stretch torque recorded during familiarization was attained (Fig. 2). Because the muscle–tendon unit exhibits a viscoelastic stress relaxation response during stretching, which steadily decreases the passive stretch torque and thus stretch intensity when the joint position is held constant (Magnusson 1998), the joint angle was continually adjusted (i.e., increased dorsiflexion), so that passive torque was always within a range of 85–90% of the maximum tolerable passive torque level. This also ensured that the intensity of stretch was similar for all participants. A total of five 60-s stretches were performed (5 × 60-s stretches) with 15-s non-stretch intervals; the ankle joint was returned to the plantar flexed position (15°) between stretches. The total stretching duration was 300 s (i.e., 5 min). The stretching protocol was chosen based on findings of Trajano and colleagues (Trajano et al. 2013, 2014a) which showed larger stretch-induced reductions in muscular force and neural drive than continuous stretch.

Maximal voluntary contraction (MVC)

To assess muscular strength following the control period and stretching intervention, peak isometric plantar flexor torque (T_{peak}) was quantified during MVCs. At PRE1, PRE2, POST, and POST10, participants performed either two (*Protocol A*) or six (*Protocol B*) 3–5 s MVCs separated by 30 s while seated in the isokinetic dynamometer with the ankle in the neutral position (0°). Participants were instructed to perform the MVCs 'as fast and as hard as possible'. Strong verbal encouragement was given to participants during each MVC. At each measurement timepoint, T_{peak} during each MVC was averaged and used for subsequent analysis. T_{peak} data from PRE1, POST, and POST10 are expressed relative to PRE2 (i.e., pre-stretch) data. All torque data were low-pass filtered at 25 Hz.

Electromyography (EMG)

Surface EMG was used to record muscle activity as well as evoked responses to TMS (i.e., MEP and cSP) and tibial nerve stimulation (M_{max}). Voluntary EMG activity from medial gastrocnemius (MG) and tibialis anterior (TA) of

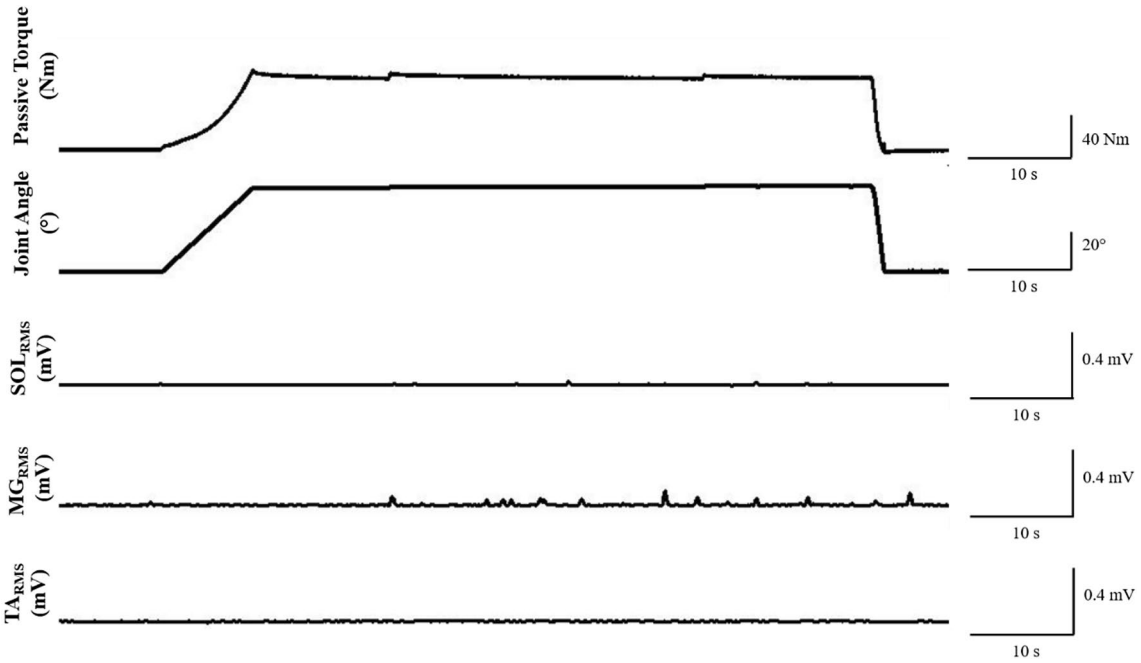

Fig. 2 Traces from a single subject during a 60-s plantar flexor stretch of the passive stretch protocol of the passive torque (first row) and joint angle (second row) as well as soleus (third row), medial gastrocnemius (fourth row), and tibialis anterior (fifth row) root-mean square EMG

the right leg was recorded during MVCs and stretching using bipolar configurations of two Ag/AgCl self-adhesive electrodes (inter-electrode distance of 2 cm; Blue Sensor N-00-S, 28 mm^2, Ambu, Ballerup, Denmark). MG electrodes were positioned over the most prominent area of MG when isometrically contracted and TA electrodes were placed 1/3 of the distance between the lateral epicondyle of the tibia and the medial malleolus. A pseudo-monopolar EMG configuration was used over SOL to record voluntary EMG activity and evoked responses (i.e., MEP, cSP, and M_{max}) with better signal-to-noise ratios with one electrode placed ~ 3 cm distal to the head of the MG and the second electrode placed ~ 3 cm superior to the medial malleolus and over the Achilles tendon-soleus muscle–tendon junction (Blazevich et al. 2012). EMG was sampled at 2000 Hz and bandpass filtered at 10–1000 Hz (BioAmp EMG System, ADInstruments, NSW, Australia; gain = 1000, input impedance = 200 MΩ, common mode rejection ratio ≥ 85 dB at 1–60 Hz, noise input ≤ 1.3 μV RMS).

Voluntary EMG activities of SOL, MG, and TA were smoothed post-hoc using a symmetric root-mean square filter using a 100-ms averaging window (SOL$_{RMS}$, MG$_{RMS}$, and TA$_{RMS}$). Maximal SOL$_{RMS}$, MG$_{RMS,}$ and TA$_{RMS}$ were collected from a 500-ms epoch around the T_{peak} values for *Protocol A* and from 500-ms epoch just prior to the TMS pulse in *Protocol B* for each measurement timepoint. SOL$_{RMS}$ and MG$_{RMS}$ were averaged to obtain a global measure of triceps surae EMG (TS$_{RMS}$; Trajano et al. 2013). All maximal EMG$_{RMS}$ amplitudes were normalized to M_{max} amplitude of the corresponding muscle (EMG$_{RMS}$/*M*; see "Tibial nerve stimulation") to account for potential influence of peripheral changes in fibre membrane characteristics (Arabadzhiev et al. 2010). EMG$_{RMS}$/*M* ratios at PRE1, POST, and POST10 are expressed relative to the PRE2 EMG$_{RMS}$/*M* ratio, and any changes in this ratio are considered to reflect alterations in central drive to the muscle (Neyroud et al. 2015). To ensure that stretching was imposed on resting muscle (i.e., passive), SOL and MG EMG were monitored, so that EMG activity was < 10% of maximum measured during MVCs in PRE, and TA EMG was also monitored to ensure that voluntary or reflexive activation was not present (see Fig. 2).

Tibial nerve stimulation

Percutaneous tibial nerve stimulation was used to evoke *M*-waves to assess potential changes in peripheral (muscle) function. A constant-current electrical stimulator (DS7A, Digitimer Ltd., Welwyn Garden City, UK) was used to deliver supramaximal single square-wave pulses (200-μs duration, 400 V) to the tibial nerve of the right leg. A self-adhesive circular cathode (pick-up area 77 mm^2; Unilect 4535 M, Ag/AgCl, Unomedical Ltd, Redditch, UK) was placed over the tibial nerve in the popliteal fossa and a rectangular self-adhesive anode (5 × 9 cm; Dura-Stick Plus, DJO Global LLC., Vista, USA) was placed just proximal to the patella. An elastic band was placed around the knee over the cathode to hold the cathode in position and to apply constant pressure throughout testing sessions.

The stimulation site that elicited the greatest SOL M-wave response at a submaximal stimulation intensity was located by a hand-held cathode electrode pen (Compex Motor Point Pen, DJO Global LLC., Vista, USA). The stimulation intensity for SOL M_{max} was determined by increasing stimulator intensity in 5–10 mA increments from a sub-motor threshold intensity, where no evoked response was observed, until the *M*-wave amplitude plateaued, with 10-s intervals between stimuli, while the participants were at rest in the isokinetic dynamometer. The stimulus intensity eliciting resting SOL M_{max} was then increased by 50% to ensure a supramaximal stimulus intensity (150%) was used (*Protocol A*: 103.1 ± 48.0 mA; *Protocol B*: 104.1 ± 50.3 mA). The supramaximal stimulus intensity for M_{max} was held constant throughout the experiment.

Transcranial magnetic stimulation

To assess changes in corticospinal excitability during both *Protocol A* and *Protocol B*, magnetic stimulation was provided by a double-cone coil (110 mm diameter) delivering single magnetic stimuli from a Magstim 200^2 stimulator (Magstim, Whitland, UK) to the motor cortex area contralateral to the plantar flexors of the right leg. The coil was orientated to induce a posterior-anterior current. The optimal stimulation site was found by moving the coil over the contralateral motor cortex area in 1-cm increments laterally, anteriorly, and posteriorly to the vertex using a pre-defined grid marked on a cap worn by the participants which was set according to the intersection of lines between the inion and nasion and the left and right ear tragus. The position eliciting the greatest average of three SOL MEPs while evoking minimal TA response (< 50% of SOL MEP) was then marked on the cap to ensure positioning throughout experimental sessions. A custom-made coil holder and an experienced investigator maintained coil placement throughout the experiments. Using Parameter Estimation by Sequential Testing (Awiszus 2003) procedures through the TMS Motor Threshold Assessment Tool software (MTAT 2.0; Awiszus and Brockhardt 2011; Silbert et al. 2013), resting motor threshold (RMT) was determined as the lowest TMS intensity that yielded a peak-to-peak resting SOL MEP amplitude of at least 60 μV [mean ± SD; *Protocol A*: 52.5 ± 10.9% of maximal stimulator output (MSO); *Protocol B*: 46.3 ± 14.5% MSO]. TMS intensity was then set at 120% of RMT (*Protocol A*: 63.0 ± 13.1% MSO; *Protocol B*: 55.6 ± 17.4% MSO) and this intensity was kept constant throughout the sessions (Soto et al. 2006).

SOL MEP peak-to-peak amplitudes were measured and normalized to M_{max} amplitude (MEP/*M*). In *Protocol B*, the TMS-induced EMG cortical silent period (cSP) duration was measured manually by quantifying the time (ms) from MEP onset to a constant (> 1 s) return of voluntary EMG activity that was greater than 2 standard deviations of the pre-stimulus average SOL EMG (Damron et al. 2008). The individual MEP/M ratios and cSP durations were averaged and are expressed relative to those obtained at PRE2. Changes in MEP/*M* ratio were used to assess corticospinal excitability. The cSP was used to assess intracortical inhibition. While some recent evidence suggests that cSP is mediated by both spinal and cortical mechanisms at long durations (> 50 ms; Yacyshyn et al. 2016), these data were collected during stimulations with greater intensities than used in the current study, and spinal excitability was tested during a period without descending drive and compared to a control condition with descending drive. Respectively, several direct recordings, clinical population, and pharmacological studies strongly suggest a critical role for $GABA_B$-mediated intracortical inhibition affecting the cSP at durations longer than 50 ms (Roick et al. 1993; Ziemann et al. 1993; Siebner et al. 1998; Chen et al. 1999). Given that changes in neurophysiological function were expected to occur rapidly, measurements at each time point had to be collected in the shortest time possible and thus additional measurements, including test of short- and long-interval intracortical inhibition, could not be included.

Data and statistical analysis

Data are presented in text and figures as mean ± standard deviation (SD). Normality of all data was checked by Shapiro-Wilk test. Separate one-way repeated measures ANOVAs were performed to compare changes T_{peak}, MEP/*M*, cSP, joint angle, TA_{RMS}, and a one-way repeated measures MANOVA were used for changes in SOL_{RMS}/M, MG_{RMS}/M, and TS_{RMS}/M over time, using SPSS statistical software (IBM Corporation, NY, USA). When Mauchly's Test of Sphericity was violated, Greenhouse–Geisser corrections were used. Bonferroni-corrected pairwise comparisons were performed when significant time effects were detected. Pearson's product-moment correlations were calculated to determine relationships between changes in torque, EMG, MEPs, and cSP immediately and 10 min after stretching. The 90% confidence intervals (CI) are also presented for statistically significant correlations. Intraclass correlations (ICC) and coefficient of variation were calculated for reliability of experimental variables (Table 1). Statistical significance was set at an α level of 0.05.

Results

There were no significant changes after the control period in any voluntary or TMS measure for either Protocol A (Table 2) or B (Table 3).

Table 1 Intrasession reliability and coefficients of variation (CV) of experimental variables

	ICC	CV (%)
T_{peak}	0.98	5.3
SOL_{RMS}	0.98	12.2
MG_{RMS}	0.96	10.9
TS_{RMS}	0.98	9.3
MEPA	0.95	17.0
MEPB	0.95	11.4
MEP_A latency	0.98	1.0
MEP_B latency	0.77	2.3
cSP	0.99	4.0
SOL_{PSU} M_{max}	0.99	2.1
SOL_{BP} M_{max}	0.99	4.7
MG M_{max}	0.99	6.4

T_{peak} peak MVC torque, *SOL_{RMS}* soleus RMS EMG, *MG_{RMS}* medial gastrocnemius RMS EMG, *TS_{RMS}* triceps surae RMS EMG, *MEP_A*, motor-evoked potential during rest Protocol A, *MEP_B* motor-evoked potential during MVC Protocol B, *cSP* cortical silent period, *SOL_{PSU} M_{max}* soleus pseudo-monopolar EMG maximal M-wave, *SOL_{BP} M_{max}* soleus bipolar EMG maximal M-wave, *MG M_{max}* medial gastrocnemius maximal M-wave

There were significant time effects for joint angle during the stretching from the end of the first minute to end of the fifth minute for both Protocols A ($F_{4,48}=35.56$, $P<0.001$) and B ($F_{1.97,23.65}=3.98$, $P=0.033$) with increases of $8.2\pm3.6\%$ ($30.5\pm6.5°$ vs. $33.1\pm6.8°$; $P<0.001$) and $5.8\pm7.7\%$ ($30.8\pm7.9°$ vs. $32.5\pm8.2°$; $P=0.033$), respectively.

There were significant time effects for T_{peak} in both Protocols A ($F_{1.95,23.47}=11.16$, $P<0.001$) and B ($F_{1.47,17.64}=17.63$, $P<0.001$) with significant decreases of $20.1\pm15.9\%$ ($P=0.004$) and $17.2\pm13.5\%$ ($P=0.006$), respectively (Fig. 3). The decreases in T_{peak} recovered by 10 min, and no significant differences were observed relative to immediately before stretching (PRE2).

In Protocol A, there were significant time effects for maximal SOL_{RMS}/M ($F_{3,36}=7.94$, $P<0.001$) and TS_{RMS}/M ($F_{1.87,22.48}=5.14$, $P=0.016$) during MVCs with decreases of $19.1\pm19.2\%$ ($P=0.023$) and $18.0\pm18.2\%$ ($P=0.023$) immediately after stretching, respectively (Fig. 5). A non-significant decrease of $17.7\pm21.2\%$ in MG_{RMS}/M was observed immediately after stretching ($P=0.066$). In Protocol B, there were also significant time effects for SOL_{RMS}/M ($F_{3,36}=11.57$, $P<0.001$), MG_{RMS}/M ($F_{3,36}=6.67$, $P=0.001$), and TS_{RMS}/M ($F_{3,36}=10.28$, $P<0.001$) with decreases immediately after stretching of $13.6\pm9.0\%$ ($P<0.001$), $14.9\pm13.0\%$ ($P<0.01$), and $13.0\pm9.3\%$ ($P<0.01$), respectively. In both Protocols A and B, all EMG amplitudes returned to PRE2 levels by 10 min. There were no significant changes in TA_{RMS} during MVCs for either Protocol A or B.

There were significant moderate–strong correlations between the decreases in T_{peak} and SOL_{RMS}/M [$r=0.94$ (CI 0.87–0.98), $P<0.001$], MG_{RMS}/M [$r=0.70$ (CI 0.36–0.88), $P=0.008$) and TS_{RMS}/M [$r=0.85$ (CI 0.64–0.94], $P<0.001$] in Protocol A when measured immediately after stretching. For Protocol B, there were also moderate–strong correlations between decreases in T_{peak} and SOL_{RMS}/M [$r=0.81$ (CI 0.56–0.93), $P=0.001$], MG_{RMS}/M [$r=0.47$ (CI 0.01–0.76), $P=0.102$], and TS_{RMS}/M [$r=0.67$ (CI 0.30–0.86), $P=0.012$]. During the recovery period from POST to POST10 in Protocol A, the recovery of T_{peak} was correlated with the recovery of SOL_{RMS}/M [$r=0.83$

Table 2 Plantar flexor torque, EMG, and TMS data measured in Protocol A

	PRE1	PRE2	POST	POST10
T_{peak} (Nm)	194.3 ± 54.1	198.9 ± 52.2	162.2 ± 63.1**	191.7 ± 58.0
SOLRMS/*M*	0.033 ± 0.013	0.035 ± 0.016	0.028 ± 0.012*	0.035 ± 0.014
MG_{RMS}/M	0.041 ± 0.014	0.041 ± 0.018	0.036 ± 0.018	0.041 ± 0.019
TS_{RMS}/M	0.077 ± 0.042	0.076 ± 0.039	0.063 ± 0.030*	0.074 ± 0.034
TA_{RMS} (mV)	0.055 ± 0.037	0.062 ± 0.044	0.055 ± 0.050	0.062 ± 0.057
MEP/*M*	0.041 ± 0.021	0.036 ± 0.017	0.045 ± 0.024	0.037 ± 0.021
MEP Latency (ms)	34.1 ± 2.1	33.9 ± 1.9	34.2 ± 2.0	34.0 ± 1.9
SOL M_{max} (mV)	20.5 ± 5.4	20.1 ± 5.3	20.6 ± 5.4	20.0 ± 5.2
MG M_{max} (mV)	8.2 ± 3.5	8.1 ± 3.6	7.9 ± 3.5	7.7 ± 3.5

Values are mean ± SD

T_{peak} peak torque during maximal voluntary isometric plantarflexion, *SOL_{RMS}/M*, *MG_{RMS}/M*, *TS_{RMS}/M*, soleus, medial gastrocnemius and triceps surae (SOL + MG) root-mean square EMG normalized to maximal M-wave amplitude, *TA_{RMS}* tibialis anterior root-mean square EMG, *MEP/M* motor-evoked potential amplitude normalized to M_{max}, *SOL* soleus, *MG* medial gastrocnemius

*Changes significantly different from PRE2; *$P<0.05$, **$P<0.001$

Table 3 Plantar flexor torque, EMG and TMS data from Protocol B

	PRE1	PRE2	POST	POST10
T_{peak} (Nm)	204.9 ± 42.0	207.3 ± 38.2	177.2 ± 46.3**	202.4 ± 37.2
SOLRMS/M	0.037 ± 0.017	0.038 ± 0.018	0.033 ± 0.015**	0.039 ± 0.018
MG_{RMS}/M	0.042 ± 0.017	0.045 ± 0.020	0.037 ± 0.013*	0.045 ± 0.021
TS_{RMS}/M	0.082 ± 0.047	0.086 ± 0.049	0.075 ± 0.045**	0.085 ± 0.048
TA_{RMS} (mV)	0.037 ± 0.01	0.037 ± 0.009	0.033 ± 0.014	0.039 ± 0.012
MEP/*M*	0.19 ± 0.06	0.18 ± 0.05	0.18 ± 0.06	0.18 ± 0.06
MEP latency (ms)	30.1 ± 1.0	30.0 ± 1.2	30.0 ± 1.0	30.0 ± 0.5
cSP (ms)	93.0 ± 32.2	93.3 ± 37.3	96.6 ± 51.2	90.1 ± 41.6
SOL M_{max} (mV)	20.2 ± 4.1	20.0 ± 4.3	19.9 ± 4.5	19.5 ± 4.7
MG M_{max} (mV)	8.3 ± 4.6	8.0 ± 4.4	7.7 ± 4.3	7.7 ± 4.6

Values are mean ± SD

T_{peak} peak torque during maximal voluntary isometric plantarflexion, SOL_{RMS}/M, MG_{RMS}/M, TS_{RMS}/M soleus, medial gastrocnemius and triceps surae (SOL + MG) root-mean square EMG normalized to maximal M-wave amplitude, TA_{RMS} tibialis anterior root-mean square EMG, *MEP/M* motor-evoked potential amplitude normalized to M_{max}, *cSP* cortical silent period, *SOL* soleus, *MG* medial gastrocnemius

*Changes significantly different from PRE2; *$P < 0.05$, **$P < 0.001$

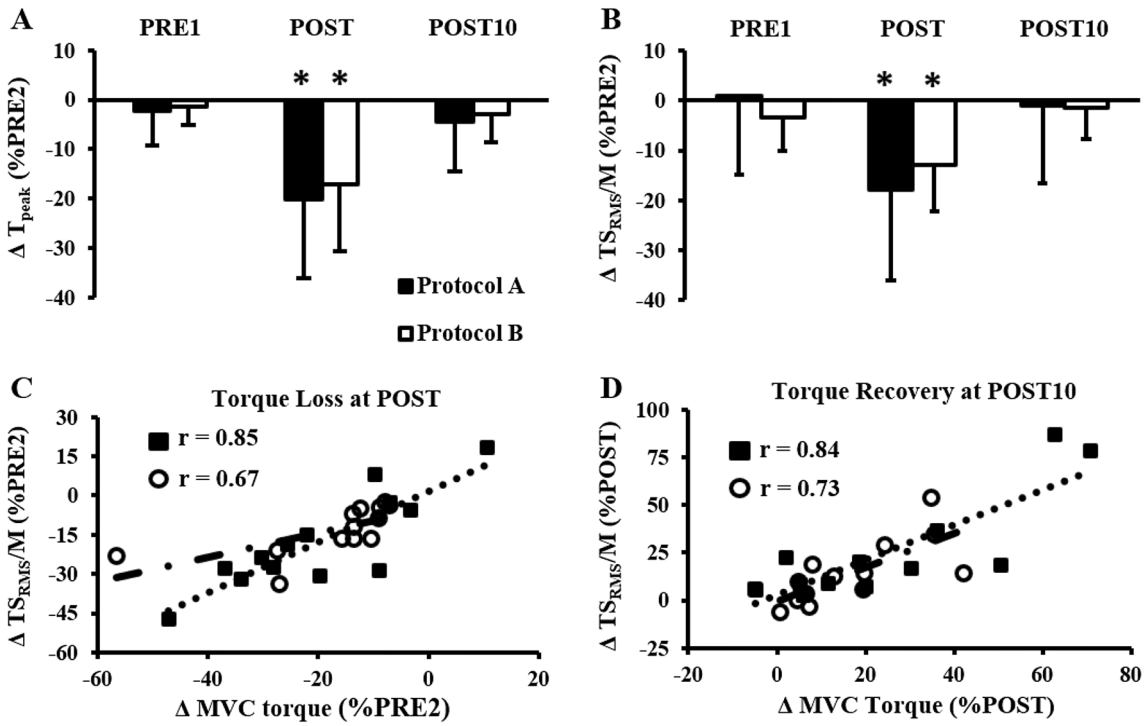

Fig. 3 Changes in and correlations of torque and EMG measured before and after stretching. **a** Changes in torque from immediately before stretch (PRE2) to immediately (POST) and 10 min after (POST10) stretching. **b** Changes in triceps surae RMS EMG relative to immediately before stretching (PRE2). **c** Correlation between changes in MVC torque and triceps surae EMG_{RMS} normalized to M_{max} from immediately before stretching (PRE2) to after (POST) stretching. **d** Correlation between recovery of MVC torque and triceps surae EMG_{RMS} normalized to M_{max} from immediately (POST) to 10 min after (POST10) stretching; (black square) *Protocol A* (open circle) *Protocol B*. (*) Changes significantly different from PRE2, *$P < 0.05$

(CI 0.60–0.93), $P < 0.001$], MG_{RMS}/M [$r = 0.81$ (CI 0.56 – 0.93), $P = 0.001$], and TS_{RMS}/M [$r = 0.84$ (CI 0.62–0.94), $P < 0.001$]. Similarly, during Protocol B POST-to-POST10 recovery period, the recovery of T_{peak} was correlated with recovery of SOL_{RMS}/M [$r = 0.57$ (CI 0.15–0.82), $P < 0.05$], MG_{RMS}/M [$r = 0.62$ [CI 0.23–0.84], $P < 0.05$], and TS_{RMS}/M [$r = 0.73$ (CI 0.41–0.89), $P < 0.01$].

There were no significant time effects for MEP/*M* in either Protocol A ($F_{1.67,20.08} = 2.71$, $P = 0.098$) or B ($F_{3,36} = 0.171$, $P = 0.915$; Fig. 4). Similarly, there were no significant time

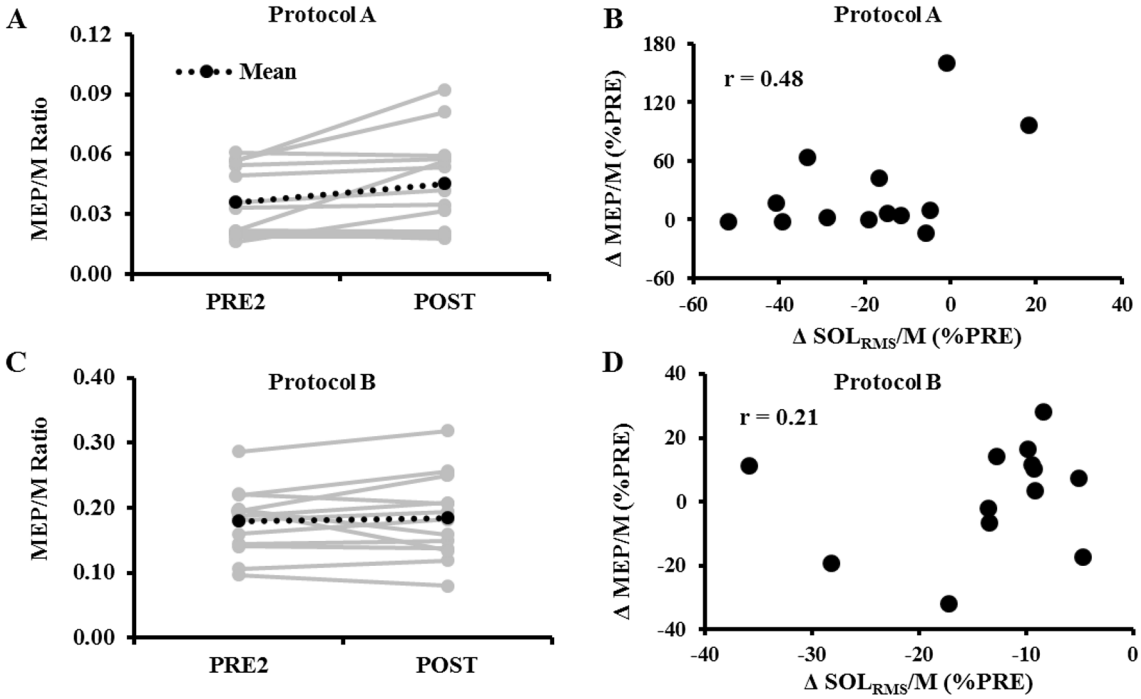

Fig. 4 Changes in MEP/*M* and correlations with SOL_{RMS}/M after stretching in Protocols A and B. **a** Individual (grey) and mean (black) changes in MEP/*M* from Protocol A. **b** Relation between changes in SOL_{RMS}/M and MEP/*M* from immediately before (PRE2) to after stretching (POST) from Protocol A. **c** Individual (grey) and mean (black) changes in MEP/*M* ratio from Protocol B. **d** Relation between changes in SOL_{RMS}/M and MEP/*M* from immediately before (PRE2) to after stretching (POST) from Protocol B

effects for cSP in Protocol B ($F_{1.41,16.93} = 1.26$, $P = 0.294$). No correlations were observed between changes in TMS variables and changes in either T_{peak} or EMG. No changes were observed in M_{max} amplitude for either Protocol A or B (Fig. 5).

Discussion

The present study examined the potential contribution of changes in corticospinal excitability to the decreases in neural drive and voluntary force following acute passive, static muscle stretching in the human plantar flexors. As in the previous studies, decreases in EMG activity were correlated with the reductions and the recovery of voluntary torque production in both protocols after the muscle stretching (Kay and Blazevich 2009; Trajano et al. 2013, 2014a). The mechanism(s) underpinning this change are not currently clear; however, both motor-evoked potential amplitude, measured in resting and maximally active conditions, and cortical silent period duration remained unchanged after the stretching in the current study. Thus, the current evidence suggests that the stretch-induced attenuation of neural drive and voluntary force output was not mediated by changes in corticospinal excitability or intracortical inhibitory mechanisms.

The finding of clear and correlated changes in SOL and TS EMG_{RMS}/M ratios and voluntary peak torque following stretching is consistent with several earlier studies (Fowles et al. 2000; Behm et al. 2001; Kay and Blazevich 2009; Trajano et al. 2013, 2014a; Ryan et al. 2014) and supports the view that impaired voluntary muscular force production after an acute bout of stretching largely results from a reduction in neural drive. Although the use of changes in EMG amplitude as a measure of neural drive alterations should be done with caution, even when normalized to the maximal M-wave amplitude (Enoka and Duchateau 2015), the magnitude and temporal profile of the stretch-induced changes in both EMG and voluntary torque in the present study closely mirror those reported in previous studies that have also shown depressions in other measures of neural drive (i.e., V-wave amplitude and voluntary activation assessed using the interpolated twitch technique; Fowles et al. 2000; Behm et al. 2001; Trajano et al. 2013, 2014a; Ryan et al. 2014). Therefore, the present results are consistent with earlier research and suggestive of a decrease in neural drive to muscle being a major causative factor to voluntary torque loss after an acute bout of prolonged passive, static muscle stretching.

The mechanisms underlying this decrease in neural drive after muscle stretching are not well described, although reductions in corticospinal excitability might be hypothesized to play a role. There is good evidence for phasic reductions in MEP amplitudes during maintenance of a muscle stretch, indicating a muscle length-dependent decrease in

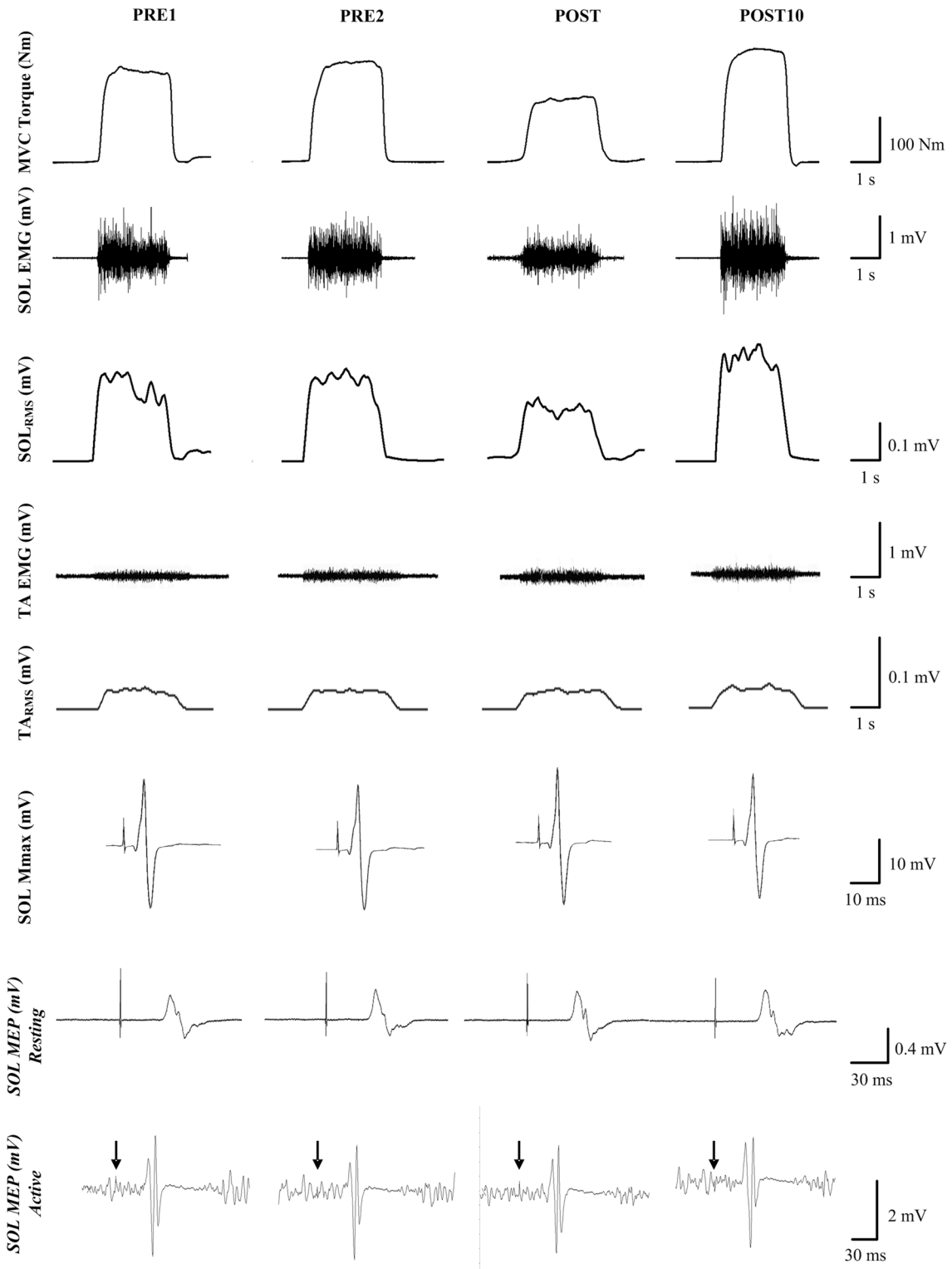

Fig. 5 Traces of raw data. Example data obtained from a single subject immediately before (PRE1) and after (PRE2) the control period and immediately (POST) and 10 min after (POST10) the stretching protocol. A decrease in MVC torque (first row), a decrease in raw soleus EMG (second row), and a decrease in triceps surae root-mean square EMG amplitudes (third row) are all clearly visible immediately after the stretching protocol. Fourth-tosixth rows, average SOL maximal M-wave traces from three trials, average SOL MEP traces from 12 trials during rest from Protocol A, and average SOL MEP traces from six trials during MVC from Protocol B. Arrows denote when TMS pulse was applied

corticospinal excitability (Guissard et al. 2001; Coxon et al. 2005). Additionally, sustained sensory/afferent stimulationcan induce lasting depressive effects on corticospinal excitability and voluntary motor output (Marconi et al. 2008; Burns et al. 2016). However, despite our findings of both impaired neural drive (EMG_{RMS}/M ratio) and voluntary force output, we found no changes in MEP amplitudes during rest or maximally active conditions nor any relation between changes in MEP amplitude and either maximal EMG amplitudes or peak torque during MVCs. In addition, we did not find a change in the duration of the cortical silent period after stretching when TMS was superimposed on an MVC. Taken to together, these results suggest a lack of stretch-induced shift in inhibitory and excitatory influences after prolonged muscle stretching, which thus suggests that these mechanisms are unlikely to play a key regulatory role in neural drive impairments after stretching.

It should be noted that the lack of change in MEP amplitudes after muscle stretching does not completely rule out the possibility of changes in the corticospinal pathway. It is known that the MEP is influenced by both cortical and spinal mechanisms (Taylor and Gandevia 2004) and that the TMS-elicited MEP is composed of corticospinal volleys in both direct monosynaptic and indirect polysynaptic pathways when targeting the soleus muscle (Brouwer and Ashby 1992; Nielsen and Petersen 1995a, b). Thus, divergent changes at any of these sites could obscure potential stretch-induced changes in MEP amplitude. For example, this potentially was displayed by the decrease in EMG activity and lack of change in MEP amplitude after stretching, where the lack of change in MEP could be explained by a potential decrease in spinal excitability (implied by a decrease in EMG activity) cancelling out a possible strectch-induced increase in cortical excitability. However, the reduced, and significantly different, EMG activity immediately after stretching may have had confounding implications on MEP responses and the interpretation of the effects of stretching on corticospinal excitability given that ongoing EMG activity directly influences MEP amplitude (Oya et al. 2008). Therefore, it may be of interest in future studies to control spinal excitability during neurophysiological measurements to better interrogate mechanisms contributing to the stretch-induced neural drive and force loss. In addition, because measurements at each time point had to be collected in the shortest time possible, and thus paired-pulse TMS examinations of short- and long-interval intracortical inhibition could not be included, should be done in future studies. Additional testing of the integration of afferent information at the spinal level, through H-reflex and other tests (i.e. reciprocal inhibition and presynaptic inhibition), might also provide a more rigorous inspection of neurophysiological changes in response to acute static stretching. Furthermore, a non-significant trend towards an increase in MEP amplitude measured in soleus was observed in Protocol A (see Fig. 4a; Table 2), indicating the possibility for change, at least in some individuals. Examination of the individual data (Fig. 4a) shows that this trend resulted from increases in 4 of the 14 participants, and that these changes were not correlated with the changes in torque production. The interpretation of the current data may be constrained, however, as only a limited number of MEPs were evoked in each protocol (Protocol A: 12 MEPs and Protocol B: 6 MEPs). Though the current data appears to be reliable (see Table 1) studies with greater number of MEPs should be completed to confirm the current results. Nonetheless, the current data do not provide reasonable evidence for changes in corticospinal excitability being a factor influencing force production after muscle stretching.

Previous researchers have speculated that stretch-induced impairment in neural drive may result from alterations in the recruitment and/or firing frequency of higher threshold motoneurons (Ryan et al. 2014). However, it is also noteworthy that MEP amplitudes in the present study were approximately 4% (Protocol A) and 20% (Protocol B) of M_{max}, with voluntary contraction facilitating MEP amplitudes by increasing spinal motoneuron excitability (Oya et al. 2008). If M_{max} amplitude is an electrophysiological estimate of complete motoneuron pool activation (Pierrot-Deseilligny and Burke 2012) and TMS recruits motoneurons in a physiological order (Bawa and Lemon 1993) then, comparatively, only a sub-population of motoneurons (i.e., those with lower activation thresholds) were examined by TMS in the current study. This inability to test a larger number of motoneurons may have been due to: (1) a scarcity of monosynaptic corticospinal projections and larger degree of indirect polysynaptic pathways from motor cortex to soleus (Brouwer and Ashby 1992), and/or (2) activation of inhibitory neurons, as they are activated at lower TMS intensities than excitatory neurons for lower leg muscles (Nielsen et al. 1993), and/or (3) higher discharge rates of motoneurons during maximal contractions might have increased the proportion of motoneurons at refractory periods due to after-hyperpolarization (Matthews 1999).

It has been suggested that the stretch-induced neural drive impairment mainly reflects changes in the properties of motoneurons due to a withdrawal of facilitatory muscle spindle feedback that would reduce maximal motoneuron recruitment and firing rates (Ryan et al. 2014; Trajano et al. 2014b). Despite evidence of a change in facilitatory muscle spindle feedback being presented previously (Trajano et al. 2014b), it is uncertain whether this disfacilitation could be able to influence MEP amplitudes. When MEPs are measured during rest, the rapid multiple volleys caused by TMS-excitatory input might not be sensitive to alterations in slow-activated calcium persistent inward currents at the (spinal) motoneurons which have been observed in a previous study (e.g., Trajano et al. 2014b). Calcium PICs are generated slowly by L-type Ca^{2+} channels and exhibit progressively larger Ca^{2+} currents in response to repetitive activation (Heckman

and Enoka 2012). It is also important to note that PICs are strongly initiated by excitatory monosynaptic input from Ia afferents (Heckman and Enoka 2012) and these inputs generate larger depolarizations in slow-type motoneurons. On the other hand, MEPs are generated by input from the descending pathways, and these inputs favour the activation of high-threshold fast units (Johnson et al. 2017). For instance, it is known that corticospinal input is nine times larger in high-threshold fast units compared with low-threshold slow units (Johnson et al. 2017). Therefore, because synaptic input is non-uniformly distributed across lower and higher threshold motor units, it is possible that the different tests used could be testing distinct motoneuron pools. Thus, an interesting question remains as to whether muscle stretching might differentially modulate cortical and spinal responses, and this requires examination in future studies.

In conclusion, the current results support the view that the attenuation of voluntary torque output after prolonged passive, static stretching is predominately mediated by a reduced neural drive to the muscle, as evidenced by simultaneous and correlated reductions in, and recoveries of, normalized EMG amplitudes and maximal muscle force. However, prolonged stretching had no consistent effect on corticospinal excitability or intracortical inhibitory mechanisms, and changes in corticospinal excitability and silent period were not related to the post-stretch reductions (or recoveries) in neural drive (EMG_{RMS}/M) or maximal torque production. Given that the previous evidence is suggestive that a change in motoneuron facilitation might occur after muscle stretching (Herda et al. 2010; Trajano et al. 2014b), future work might focus on examining discrete (divergent) changes in cortical vs. spinal processes to elucidate the mechanisms underpinning the stretch-induced force loss. Such information may thus aid the development of interventions to minimize the impairment in neuromuscular function after acute muscle stretching.

Author contributions TSP, AJB, and GST conceived and designed the research. TSP and BJCK conducted experiments. TSP analyzed data. TSP and AJB wrote the manuscript. All authors read and approved the manuscript.

Funding ECU International Post Graduate Research Scholarship.

Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

References

Arabadzhiev TI, Dimitrov VG, Dimitrova NA, Dimitrov GV (2010) Interpretation of EMG integral or RMS and estimates of "neuromuscular efficiency" can be misleading in fatiguing contraction. J Electromyogr Kinesiol 20:223–232. https://doi.org/10.1016/j.jelekin.2009.01.008

Awiszus F (2003) TMS and threshold hunting. Suppl Clin Neurophysiol 56:13–23

Awiszus F, Brockhardt JJ (2011) TMS motor threshold assessment tool (MTAT 2.0). https://clinicalresearcher.org/software.htm

Bawa P, Lemon RN (1993) Recruitment of motor units in response to transcranial magnetic stimulation in man. J Physiol 471:445–464

Behm DG, Button DC, Butt JC (2001) Factors affecting force loss with prolonged stretching. Can J Appl Physiol 26:261–272

Behm DG, Blazevich AJ, Kay AD, McHugh M (2016) Acute effects of muscle stretching on physical performance, range of motion, and injury incidence in healthy active individuals: a systematic review. Appl Physiol Nutr Metab 41:1–11. https://doi.org/10.1139/apnm-2015-0235

Blazevich AJ, Kay AD, Waugh C et al (2012) Plantarflexor stretch training increases reciprocal inhibition measured during voluntary dorsiflexion. J Neurophysiol 107:250–256. https://doi.org/10.1152/jn.00407.2011

Brouwer B, Ashby P (1992) Corticospinal projections to lower limb motoneurons in man. Exp brain Res 89:649–654

Burke D, Gandevia SC, Macefield G (1988) Responses to passive movement of receptors in joint, skin and muscle of the human hand. J Physiol 402:347–361

Burns E, Chipchase LS, Schabrun SM (2016) Primary sensory and motor cortex function in response to acute muscle pain: a systematic review and meta-analysis. Eur J Pain 20:1203–1213. https://doi.org/10.1002/ejp.859

Chen R (2004) Interactions between inhibitory and excitatory circuits in the human motor cortex. Exp Brain Res 154:1–10. https://doi.org/10.1007/s00221-003-1684-1

Chen R, Lozano AM, Ashby P (1999) Mechanism of the silent period following transcranial magnetic stimulation. Evidence from epidural recordings. Exp Brain Res 128:539–542. https://doi.org/10.1007/s002210050878

Chye L, Nosaka K, Murray L et al (2010) Corticomotor excitability of wrist flexor and extensor muscles during active and passive movement. Hum Mov Sci 29:494–501. https://doi.org/10.1016/j.humov.2010.03.003

Coxon JP, Stinear JW, Byblow WD (2005) Amplitude of muscle stretch modulates corticomotor gain during passive movement. Brain Res 1031:109–117. https://doi.org/10.1016/j.brainres.2004.10.062

Damron LA, Dearth DJ, Hoffman RL, Clark BC (2008) Quantification of the corticospinal silent period evoked via transcranial magnetic stimulation. J Neurosci Methods 173:121–128. https://doi.org/10.1016/j.jneumeth.2008.06.001

Enoka RM, Duchateau J (2015) Inappropriate interpretation of surface EMG signals and muscle fiber characteristics impedes understanding of the control of neuromuscular function. J Appl Physiol 119:1516–1518. https://doi.org/10.1152/japplphysiol.00280.2015

Fowles JR, Sale DG, Macdougall JD (2000) Reduced strength after passive stretch of the human plantarflexors. J Appl Physiol 89:1179–1188

Gandevia SC (2011) Kinesthesia: roles for afferent signals and motor commands. In: Comprehensive physiology. Wiley, Hoboken

Guissard N, Duchateau J (2006) Neural aspects of muscle stretching. Exerc Sport Sci Rev 34:154–158. https://doi.org/10.1249/01.jes.0000240023.30373.eb

Guissard N, Duchateau J, Hainaut K (2001) Mechanisms of decreased motoneurone excitation during passive muscle stretching. Exp brain Res 137:163–169

Heckman CJ, Enoka RM (2012) Motor Unit. Comprehensive physiology. Wiley, Hoboken, pp 2629–2682

Herda TJ, Ryan ED, Costa PB et al (2010) Acute effects of passive stretching and vibration on the electromechanical delay and musculotendinous stiffness of the plantar flexors. Electromyogr Clin Neurophysiol 50:277–288

Hore J, Preston JB, Cheney PD (1976) Responses of cortical neurons (areas 3a and 4) to ramp stretch of hindlimb muscles in the baboon

Huerta MF, Pons TP (1990) Primary motor cortex receives input from area 3a in macaques. Brain Res 537:367–371

Johnson MD, Thompson CK, Tysseling VM et al (2017) The potential for understanding the synaptic organization of human motor commands via the firing patterns of motoneurons. J Neurophysiol 118:520–531. https://doi.org/10.1152/jn.00018.2017

Kay AD, Blazevich AJ (2009) Moderate-duration static stretch reduces active and passive plantar flexor moment but not Achilles tendon stiffness or active muscle length. J Appl Physiol 106:1249–1256. https://doi.org/10.1152/japplphysiol.91476.2008

Kay AD, Blazevich AJ (2012) Effect of acute static stretch on maximal muscle performance. Med Sci Sport Exerc 44:154–164. https://doi.org/10.1249/MSS.0b013e318225cb27

Kniffki KD, Mense S, Schmidt RF (1978) Responses of group IV afferent units from skeletal muscle to stretch, contraction and chemical stimulation. Exp Brain Res 31:511–522. https://doi.org/10.1007/BF00239809

Lewis GN, Byblow WD, Carson RG (2001) Phasic modulation of corticomotor excitability during passive movement of the upper limb: effects of movement frequency and muscle specificity. Brain Res 900:282–294

Magnusson SP (1998) Passive properties of human skeletal muscle during stretch maneuvers. A review. Scand J Med Sci Sports 8:65–77

Marconi B, Filippi GM, Koch G et al (2008) Long-term effects on motor cortical excitability induced by repeated muscle vibration during contraction in healthy subjects. J Neurol Sci 275:51–59. https://doi.org/10.1016/j.jns.2008.07.025

Matthews PB (1999) The effect of firing on the excitability of a model motoneurone and its implications for cortical stimulation. J Physiol 518(Pt 3):867–882

Neyroud D, Kayser B, Place N (2015) Commentaries on Viewpoint: Inappropriate interpretation of surface EMG signals and muscle fiber characteristics impedes understanding of the control of neuromuscular function. J Appl Physiol 119:1519–1519. https://doi.org/10.1152/japplphysiol.00753.2015

Nielsen J, Petersen N (1995a) Evidence favouring different descending pathways to soleus motoneurones activated by magnetic brain stimulation in man. J Physiol 486(Pt 3):779–788

Nielsen J, Petersen N (1995b) Changes in the effect of magnetic brain stimulation accompanying voluntary dynamic contraction in man. J Physiol 484(Pt 3):777–789

Nielsen J, Petersen N, Deuschl G, Ballegaard M (1993) Task-related changes in the effect of magnetic brain stimulation on spinal neurones in man. J Physiol 471:223–243

Oya T, Hoffman BW, Cresswell AG (2008) Corticospinal-evoked responses in lower limb muscles during voluntary contractions at varying strengths. J Appl Physiol 105:1527–1532. https://doi.org/10.1152/japplphysiol.90586.2008

Pierrot-Deseilligny E, Burke DC (2012) The circuitry of the human spinal cord: spinal and corticospinal mechanisms of movement. Cambridge University Press, Cambridge

Proske U, Morgan DL, Gregory JE (1993) Thixotropy in skeletal muscle and in muscle spindles: a review. Prog Neurobiol 41:705–721

Roick H, von Giesen HJ, Benecke R (1993) On the origin of the postexcitatory inhibition seen after transcranial magnetic brain stimulation in awake human subjects. Exp Brain Res 94(3):489–498

Rosenkranz K, Rothwell JC (2003) Differential effect of muscle vibration on intracortical inhibitory circuits in humans. J Physiol 551:649–660. https://doi.org/10.1113/jphysiol.2003.043752

Rothwell JC (1997) Techniques and mechanisms of action of transcranial stimulation of the human motor cortex. J Neurosci Methods 74:113–122

Rothwell JC, Hallett M, Berardelli A et al (1999) Magnetic stimulation: motor evoked potentials. the international federation of clinical neurophysiology. Electroencephalogr Clin Neurophysiol Suppl 52:97–103

Ryan ED, Herda TJ, Costa PB et al (2014) Acute effects of passive stretching of the plantarflexor muscles on neuromuscular function: the influence of age. Age (Omaha) 36:9672. https://doi.org/10.1007/s11357-014-9672-x

Siebner HR, Dressnandt J, Auer C, Conrad B (1998) Continuous intrathecal baclofen infusions induced a marked increase of the transcranially evoked silent period in a patient with generalized dystonia. Muscle Nerve 21:1209–1212. https://doi.org/10.1002/(SICI)1097-4598(199809)21:9<1209::AID-MUS15>3.0.CO;2-M

Silbert BI, Patterson HI, Pevcic DD et al (2013) A comparison of relative-frequency and threshold-hunting methods to determine stimulus intensity in transcranial magnetic stimulation. Clin Neurophysiol 124:708–712. https://doi.org/10.1016/j.clinph.2012.09.018

Simic L, Sarabon N, Markovic G (2013) Does pre-exercise static stretching inhibit maximal muscular performance? A meta-analytical review. Scand J Med Sci Sports 23:131–148. https://doi.org/10.1111/j.1600-0838.2012.01444.x

Soto O, Valls-Solé J, Shanahan P, Rothwell J (2006) Reduction of intracortical inhibition in soleus muscle during postural activity. J Neurophysiol 96:1711–1717. https://doi.org/10.1152/jn.00133.2006

Stuart DG, Goslow GE, Mosher CG, Reinking RM (1970) Stretch responsiveness of Golgi tendon organs. Exp brain Res 10:463–476

Taylor JL, Gandevia SC (2004) Noninvasive stimulation of the human corticospinal tract. J Appl Physiol 96:1496–1503. https://doi.org/10.1152/japplphysiol.01116.2003

Trajano GS, Nosaka K, Blazevich AJ (2017) Neurophysiological mechanisms underpinning stretch-induced force loss. Sport Med 1–11

Trajano GS, Seitz L, Nosaka K, Blazevich AJ (2013) Contribution of central vs. peripheral factors to the force loss induced by passive stretch of the human plantar flexors. J Appl Physiol 115:212–218. https://doi.org/10.1152/japplphysiol.00333.2013

Trajano GS, Nosaka K, Seitz LB, Blazevich AJ (2014) Intermittent stretch reduces force and central drive more than continuous stretch. Med Sci Sport Exerc 46:902–910. https://doi.org/10.1249/MSS.0000000000000185

Trajano GS, Seitz LB, Nosaka K, Blazevich AJ (2014) Can passive stretch inhibit motoneuron facilitation in the human plantar flexors? J Appl Physiol 117:1486–1492. https://doi.org/10.1152/japplphysiol.00809.2014

Veldman MP, Maffiuletti NA, Hallett M et al (2014) Direct and crossed effects of somatosensory stimulation on neuronal excitability and motor performance in humans. Neurosci Biobehav Rev 47:22–35. https://doi.org/10.1016/j.neubiorev.2014.07.013

Wiesendanger M, Miles TS (1982) Ascending pathway of low-threshold muscle afferents to the cerebral cortex and its possible role in motor control. Physiol Rev 62:1234–1270

Yacyshyn AF, Woo EJ, Price MC, McNeil CJ (2016) Motoneuron responsiveness to corticospinal tract stimulation during the silent period induced by transcranial magnetic stimulation. Exp Brain Res 234(12):3457–3463

Ziemann U, Netz J, Szelényi A, Hömberg V (1993) Spinal and supraspinal mechanisms contribute to the silent period in the contracting soleus muscle after transcranial magnetic stimulation of human motor cortex. Neurosci Lett 156:167–171