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RESEARCH ARTICLE

Central contributions to torque depression: an antagonist perspective

Caleb T. Sypkes[1] · Vincenzo S. Contento[1] · Leah R. Bent[1] · Chris J. McNeil[2] · Geoffrey A. Power[1]

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Abstract

Torque depression (TD) is the reduction in steady-state isometric torque following active muscle shortening when compared to an isometric reference contraction at the same muscle length and activation level. Central nervous system excitability differs in the TD state. While torque production about a joint is influenced by both agonist and antagonist muscle activation, investigations of corticospinal excitability have focused on agonist muscle groups. Hence, it is unknown how the TD state affects spinal and supraspinal excitability of an antagonist muscle. Eight participants (~ 24y, three female) performed 14 submaximal dorsiflexion contractions at the intensity needed to maintain a level of integrated electromyographic activity in the soleus equivalent to 15% of that recorded during a maximum plantar flexion contraction. The seven contractions of the TD protocol included a 2 s isometric phase at an ankle angle of 140°, a 1 s shortening phase at 40°/s, and a 7 s isometric phase at an angle of 100°. The seven isometric reference contractions were performed at an ankle angle of 100° for 10 s. Motor evoked potentials (MEPs), cervicomedullary motor evoked potentials (CMEPs), and maximal M-waves (Mmax) were recorded from the soleus in both conditions. In the TD compared to isometric reference state, a 13% reduction in dorsiflexor torque was accompanied by 10% lower spinal excitability (normalized CMEP amplitude; CMEP/Mmax), and 17% greater supraspinal excitability (normalized MEP amplitude; MEP/CMEP) for the soleus muscle. These findings demonstrate a neuromechanical coupling following active muscle shortening and indicate that the underlying mechanisms of TD influence antagonist activation during voluntary force production.

Keywords Integrated electromyography iEMG · Transcranial magnetic stimulation TMS · Motor evoked potential MEP · Cervicomedullary motor evoked potential CMEP

Introduction

The reduction in steady-state isometric torque following active muscle shortening when compared to a purely isometric contraction at the same muscle length and level of activation is termed torque depression (TD) (Herzog 2004). TD is associated with a concomitant reduction in muscle stiffness, is abolished by a brief period of muscle relaxation, and appears to be dependent on the work of shortening (Abbot and Aubert 1952; Herzog et al. 2000). These observations have been attributed to a decline in the proportion of strongly bound cross-bridges in the newly formed actin–myosin overlap zone after shortening, resulting from a stress-induced angular deformation of the actin filament (Marechal and Plaghki 1979). Since its initial discovery (Abbott and Aubert 1952), this history-dependent property has been demonstrated to occur at every functional level of muscle for both electrically stimulated and voluntary contractions (De Ruiter et al. 1998; Lee and Herzog 2003; Herzog 2004; Power et al. 2014). Recently, our lab has shown altered corticospinal excitability of the agonist motor neuron pool in the TD compared to reference isometric state during maximal (Grant et al. 2017) and submaximal (Sypkes et al. 2017) voluntary contractions. Net torque production about a joint, however, is the result of both agonist and antagonist

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✉ Geoffrey A. Power
gapower@uoguelph.ca

[1] Neuromechanical Performance Research Laboratory, Department of Human Health and Nutritional Sciences, College of Biological Sciences, University of Guelph, Guelph, ON, Canada

[2] Centre for Heart, Lung and Vascular Health, School of Health and Exercise Sciences, University of British Columbia, Kelowna, BC, Canada

muscle activation, and it remains unclear how the TD state may influence excitability of the antagonist motor neuron pool and how this might influence voluntary control of torque production.

It is well established that changes in muscle force production capacity can influence descending and peripheral inputs to the motor neuron pool and lead to subsequent alterations in muscle activation (e.g., Rousanoglou et al. 2007; Duchateau and Enoka 2008; Jones et al. 2016). Only more recently, however, have specific sites of modulation of corticospinal excitability been investigated in the force enhanced (Hahn et al. 2012; Sypkes et al. 2018), and TD state after voluntary maximal (Grant et al. 2017) and submaximal (Sypkes et al. 2017) contractions. Motor evoked potentials (MEPs) and cervicomedullary motor evoked potentials (CMEPs) were recorded and normalized to the downstream response (MEP/CMEP and CMEP/maximal M-wave, Mmax) to detect differences in neural excitability within the motor cortex and spinal cord, respectively (Grant et al. 2017; Sypkes et al. 2017). During submaximal contractions, reduced torque in the TD state was shown to be associated with greater excitability of the spinal cord (i.e., normalized CMEP amplitude) and equivalent excitability of the motor cortex (i.e., normalized MEP amplitude), as compared to the reference isometric condition (Sypkes et al. 2017). However, this did not occur in all participants, indicating differences in corticospinal excitability may be influenced by a non-responder phenomenon. It was proposed that greater spinal excitability during a condition of impaired muscle force production capacity may be due to reduced firing of the Golgi tendon organ (GTO), a peripheral mechanoreceptor that provides inhibitory sensory feedback via Ib afferents to the agonist motor neuron pool in a tension-dependent manner. Cortical and spinal excitability have also been considered following shortening contractions at maximal intensity. While there were no significant differences in excitability between the TD and purely isometric conditions, a significant negative relationship between normalized CMEP and normalized MEP amplitudes was found, whereby higher or lower spinal excitability was matched by an opposite finding at the motor cortex (Grant et al. 2017). Together, these findings demonstrate that while the underlying mechanisms of TD are indeed attributed to intrinsic muscle contractile properties, their effects influence voluntary control of movement.

In these previous studies investigating the influence of TD on voluntary neuromuscular control, central nervous system excitability has been measured only for the agonist muscle. However, given that TD describes the reduction in net torque about a joint, it is important to consider the contributions of both agonist and antagonist muscles in voluntary torque production. Further, antagonist muscle groups are closely linked to their counterpart agonist muscle groups in the descending and peripheral neural inputs they receive. For example, while the Ib afferent delivers inhibitory feedback to the agonist motor neuron pool, it also delivers excitatory feedback to the antagonist motor neuron pool (Jami 1992). Although the agonist and antagonist muscles are closely linked in their contributions to net joint torque production, it remains unclear how changes in agonist muscle force production capacity in the TD state may influence antagonist motor neuron pool excitability.

The purpose of the present study was to assess differences in spinal and supraspinal excitability of an antagonist muscle in the TD state following a shortening contraction at a submaximal intensity, as compared to a reference isometric contraction at the same muscle length and level of activation. A series of dorsiflexion contractions were performed while antagonist co-activation (integrated electromyography; iEMG) of the soleus was held constant at 15% of that recorded during a maximal plantar flexion contraction, and evoked potentials were generated at the cortical (i.e., MEP), and spinal (i.e., CMEP) level to assess central nervous system excitability in the two muscle states. Owing to a reduced capacity to generate torque in the TD state and a suspected reduction in Ib afferent firing, we hypothesized that the TD state would be accompanied by lower antagonist muscle group spinal excitability when compared to the purely isometric condition. Further, because of a design that required matching of antagonist muscle group motor neuron output (i.e., iEMG), we hypothesized that the TD state would also be associated with greater cortical excitability to overcome lower spinal excitability.

Methods

Participants

Eight healthy participants (5 male, 3 female) with a mean age of 24 ± 5 years, height of 173 ± 7 cm, and mass of 70.3 ± 15.4 kg were recruited from the university population for participation in the study. All had no prior history of neuromuscular disease or ankle joint injuries. Data were collected within a single session. Participants gave written informed consent prior to testing and all procedures were approved by the human Research Ethics Board of the University of Guelph (REB: 15NV008) and, with the exception of registration in a database, conformed to the Declaration of Helsinki.

Experimental set-up

A HUMAC NORM dynamometer (CSMi Medical Solutions, Stoughton, MA) was used to record torque, angular velocity and position. Each participant sat with their right hip and knee angles set at 110° and 140° (180°; straight),

respectively. Joint angles were measured using a goniometer. The right knee was immobilized with the dynamometer's leg restraint (superior) and a malleable cushion (inferior), while movement at the torso was restricted with a four-point seatbelt harness. The right foot was fixed to the dorsi/plantar flexor adaptor with one inelastic strap placed over the ankle and another at the mid-distal portion of the metatarsals. The dynamometer's maximum ankle dorsiflexion and plantar flexion angles were set to 100° and 140° (90°; neutral), respectively, allowing for 40° of ankle excursion. Isometric reference contractions were performed at 100° whereas TD contractions started at 140°, were maintained during ankle rotation to 100°, and continued at this angle (see the "Experimental procedures" section below for specific details).

Locations for the EMG electrodes were prepared by shaving and cleaning the skin with alcohol swabs. The active electrode was placed over the tibialis anterior approximately 7 cm inferior and 2 cm lateral to the tibial tuberosity and a reference electrode was placed over the distal tendon of the tibialis anterior, at the level of the malleoli. To record antagonist activity, the active electrode was placed on the soleus, along the midline of the leg approximately 2 cm inferior to the border of the heads of the gastrocnemii, and a reference electrode was placed on the calcaneal tendon. A single ground electrode was centered on the patella. Silver–silver chloride (Ag/AgCl) electrodes (1.5 × 1 cm: Kendall, Mansfield, MA) were used for all recordings.

Raw EMG, torque, angular velocity, joint angle and stimulus trigger data were converted to digital format using a 12-bit analog-to-digital converter (PowerLab System 16/35, ADInstruments, Bella Vista, Australia), and analyzed with Labchart software (Labchart, Pro Modules 2014, version 8). Torque and EMG data were recorded at a sampling rate of 1000 Hz and 2000 Hz, respectively. EMG data were band-pass filtered using a digital filter (3–1000 Hz). Figure 1 depicts the joint angle, torque, and iEMG traces for a single trial.

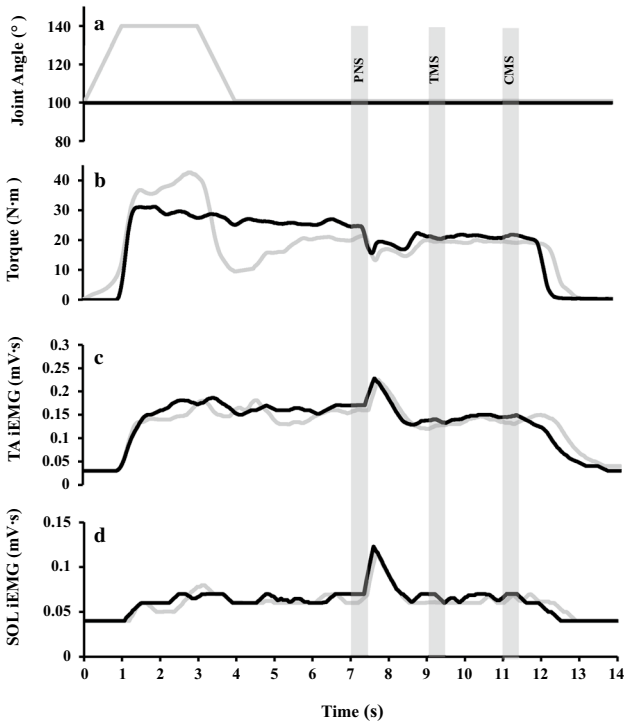

Fig. 1 Ankle angle (**a**), dorsiflexor torque (**b**), tibialis anterior iEMG (**c**), and soleus iEMG (**d**) traces during TD (grey) and ISO (black) contractions for a representative participant. During TD trials, a dorsiflexion contraction corresponding to 15% soleus iEMG was initiated for 2 s at 140° PF before the dynamometer arm rotated the ankle at 40°/s to an angle of 100° PF. A PNS was delivered to the tibial nerve at the 6th second of the contraction (to elicit an Mmax), a TMS pulse was delivered at the 8th second (to elicit an MEP), and a CMS pulse was administered at approximately the 10th second (to elicit a CMEP). During isometric reference trials, the same protocol was in effect, with the exception that the ankle was fixed at an angle of 100° PF. *PNS* peripheral nerve stimulus, *TMS* transcranial magnetic stimulation, *CMS* cervicomedullary stimulation

Peripheral nerve stimulation

To test the voluntary activation of the dorsiflexors (see "Maximum voluntary contraction and voluntary activation" section) and obtain Mmax from tibialis anterior and soleus, peripheral nerve stimulation (PNS) was delivered transcutaneously with a standard clinical bar electrode (Empi, St Paul, Minnesota, USA) coated in conductive gel. The deep fibular nerve, innervating the dorsiflexor muscles, was located by palpating the head of the fibula and moving posteroinferiorly until the nerve was identified. Stimulation distal to the bifurcation of the common fibular nerve was ensured to limit activation of the peroneal muscles. The tibial nerve, innervating the plantar flexor muscles, was found by locating the distal tendon of the semitendinosus muscle and moving laterally while palpating deep into the popliteal fossa. All PNS was delivered as a single pulse from a constant current, high voltage stimulator (model DS7AH, Digitimer, Welwyn Garden City, Hertfordshire, UK). Voltage was set to a maximum of 400 V and pulse width to 200 µs. Current was incrementally elevated until a plateau was reached for the peak-to-peak amplitude of the resting M-wave (Mmax). Figure 2a shows the soleus Mmax for a single participant. To ensure consistent activation of all motor neurons throughout the experiment, the current was increased to a supramaximal level, equivalent to 110% of that required to generate Mmax (90–200 mA and 150–300 mA for deep fibular and tibial nerves, respectively).

Maximum voluntary contraction and voluntary activation

Voluntary activation of the dorsiflexors and plantar flexors was assessed during brief maximum voluntary contractions

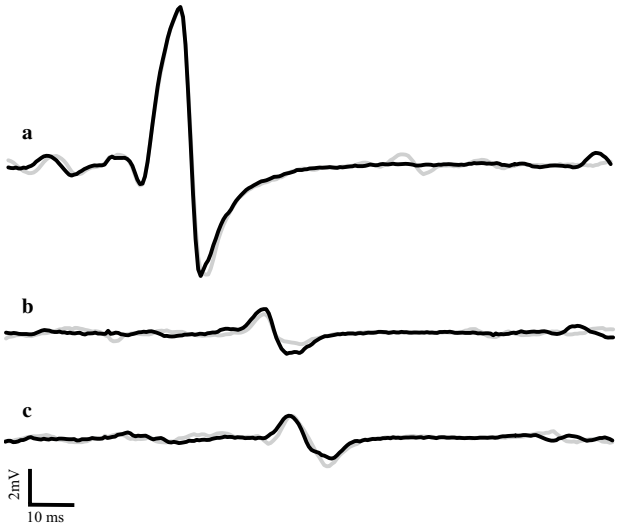

Fig. 2 Raw data traces of an Mmax (**a**), CMEP (**b**), and MEP (**c**) recorded from the soleus following PNS, CMS, and TMS respectively, in the TD (grey) and ISO (black) states

performed both prior to and following the experimental trials. The interpolated twitch technique was used to evaluate voluntary activation during these maximum voluntary contractions (Belanger and McComas 1981; Power et al. 2014). The torque resulting from PNS delivered during the plateau phase of the maximum voluntary contraction was compared to a resting twitch evoked 1–2 s after relaxation. The level of voluntary activation was calculated as: voluntary activation (%) = [1 − (interpolated twitch torque/resting twitch torque)] × 100%. The participants were verbally encouraged during all maximum voluntary contractions and the torque trace was visible during each contraction (Gandevia 2001). All participants were required to reach a minimum of 95% voluntary activation for both the dorsiflexors and plantar flexors and were given 5 min of rest before continuing with the experiment.

Determining submaximal muscle activation

To determine the submaximal iEMG target, participants were instructed to perform a 9 s maximal plantar flexion contraction at an ankle angle of 100°, an angle equivalent to that set during the proceeding experimental trials (see "Experimental procedures"). The average iEMG collected between 6 and 8 s was then used to determine the 15% submaximal antagonist co-activation iEMG target. $A \pm 5\%$ window was calculated about this 15% target, and for all subsequent activation-controlled contractions, participants were instructed to activate their ankle dorsiflexors (agonists) to the level that maintained their soleus (antagonist) iEMG within guidelines marking this target window. This level of antagonist co-activation was chosen because it allowed a reasonable proportion of the MN pool (CMEPs and MEPs of ~ 10% Mmax) to be recorded in all participants. A lower level of co-activation would have led to evoked responses that were more variable and less discernable from the background EMG. A higher level of co-activation would have been unachievable for some participants because of the ceiling effect of the agonist activation. Based on pilot testing, for the average participant to achieve 15% antagonist co-activation, an effort of ~ 65% MVC was required.

Cervicomedullary stimulation

Ag/AgCl electrodes (10 mm diameter; Cleartrace 1700-030, ConMed Corporation, Utica, New York, United States) were used for cervicomedullary stimulation (CMS) to generate CMEPs by passing a current across the spinal cord at the level of the mastoids. Electrodes were placed at a location approximately 2 cm superior and medial to the mastoid processes (Ugawa et al. 1991). Single stimuli were presented (anode on right side and cathode on left) with a constant current, high voltage stimulator (DS7AH). Voltage was set to a maximum of 400 V and pulse width to 200 µs. Current was adjusted to produce a soleus CMEP with an amplitude equivalent to ~ 10% of resting Mmax (Fig. 2b) while the participant performed a dorsiflexion isometric contraction corresponding to 15% soleus iEMG. This current (200–350 mA) was used for the remainder of the experiment.

Transcranial magnetic stimulation

MEPs were elicited by transcranial magnetic stimulation (TMS) of the motor cortex. Stimulation was delivered with a double cone coil (110 mm) linked to two Magstim 200^2 stimulators via a BiStim module (Magstim, Dyfed, UK). To determine the ideal location for coil placement, single stimuli were delivered at 20% of stimulator output while the participant performed brief dorsiflexion contractions at an intensity that produced 15% soleus iEMG. The coil was originally placed at the vertex and was moved in 1 cm increments to the left as well as forward and backward. The placement which yielded the largest soleus MEP was marked on the participant's scalp and used throughout the duration of testing. The centre of the coil was flush with the head and the wings of the coil were perpendicular to the midline of the head (i.e., oriented left to right rather than on an angle). The position of the coil was maintained by visual inspection. Stimulus intensity was adjusted until the soleus MEP amplitude was equivalent to that of the CMEP (i.e., ~ 10% of resting Mmax; Fig. 2c) during a brief dorsiflexion contraction corresponding to 15% soleus iEMG. This stimulus intensity (25–60% of stimulator output) was used for the remainder of the experiment.

Experimental procedures

Each TD trial was followed by an isometric reference (ISO) trial, and this sequence was repeated seven times for a total of 14 dorsiflexion contractions at 15% soleus iEMG (i.e., matching antagonist co-activation). Participants were given visual feedback of the soleus iEMG amplitude on the computer monitor and were verbally encouraged to match the target as closely as possible during all contractions. 3 min of rest separated all submaximal contractions throughout the experiment to prevent muscle fatigue.

Stimulus protocol: assessment of cortical, spinal, and peripheral excitability

Each TD trial consisted of a 10 s dorsiflexion contraction at an intensity sufficient to produce 15% antagonist co-activation (i.e., soleus iEMG). The contractions involved a 2 s isometric phase at an ankle angle of 140°, a 1 s isokinetic shortening phase (40°/s) and a 7 s isometric phase at 100°. Although the dynamometer arm rotated the ankle at 40°/s, the angular velocity was not constant throughout the entire range of motion, particularly at the start, owing to an acceleration phase. This acceleration phase was constant across participants (2000°/s^2) and the phase length was minimized to limit its effects. However, its presence reduces the velocity of the ankle rotation, increasing dorsiflexion work and potentially enhancing TD, which would be consistent for all participants. A PNS pulse was delivered to the tibial nerve at the 6th second (time point 1) and a TMS pulse was administered at the 8th second (time point 2). Due to the disruptive nature of the TMS pulse, a CMS pulse was manually delivered when the participant returned their soleus iEMG to within the ± 5% window at approximately the 10th second (time point 3). During ISO trials, an isometric dorsiflexion contraction corresponding to 15% soleus iEMG was performed for 10 s at an angle of 100°, with stimulation delivered as described for TD trials.

Data analysis and statistics

Mean dorsiflexor torque and root mean squared EMG (EMG_{RMS}) of both tibialis anterior and soleus were calculated in the 500 ms window prior to each stimulus. A paired *t* test was performed to compare the mean torque data for each participant between TD and ISO trials to validate the presence of TD at the time of stimulation. As in similar studies investigating TD, non-responders were considered participants who present with no observable TD on average. There were no non-responders identified in the present study. To assess motor neuron excitability in the TD and ISO states, each evoked CMEP was normalized to the corresponding Mmax (CMEP/Mmax) to control for possible changes in peripheral excitability. To assess motor cortical excitability in the TD and ISO states, each evoked MEP was normalized to the corresponding CMEP (MEP/CMEP) to control for possible changes in subcortical excitability. The EMG_{RMS} of the resting Mmax recorded at the tibialis anterior and soleus was used to normalize the voluntary tibialis anterior and soleus EMG, respectively.

To detect and remove outliers from the data set, the mean normalized amplitude for both MEPs and CMEPs were calculated for each participant and any response which fell more than two standard deviations above or below the mean was rejected. Using this method, 3 of 112 responses were removed from the data set.

A paired *t* test was performed for Mmax, normalized CMEP and normalized MEP data between the TD and ISO states to elucidate differences in peripheral, spinal and cortical excitability in the TD state. The same test was also used to detect any differences in the torque produced during maximum voluntary contractions performed before and after the experiment to assess if fatigue was induced by the experimental protocol. A two-way ANOVA was also performed to compare EMG_{RMS} data between the TD and ISO states, and between each time point during the experimental protocol for both the tibialis anterior and soleus. Descriptive data found in the text are reported as means ± standard deviation (SD), and data presented in figures are reported as means ± standard error of the mean (SE). Significance was determined based on a p value of < 0.05.

Results

Maximum voluntary contraction and voluntary activation

Mean pre-trial MVC torque was 29.0 ± 9.1 N m for the dorsiflexors, and 96.7 ± 24.7 N m for the plantar flexors. All participants were capable of achieving near-maximal values for voluntary activation as assessed using the interpolated twitch technique (99.2 ± 1.2% and 98.9 ± 1.3% for the dorsiflexors and plantar flexors, respectively). Following the 14 contractions of the experimental protocol, MVC torque of the dorsiflexors was reassessed, and its magnitude was not different from the pre-trial average (30.2 ± 10.3 N m, $p > 0.05$).

Dorsiflexion torque and muscle activity

While performing the 15% antagonist co-activation matching task in the ISO state, participants maintained an average dorsiflexion torque equivalent to 69.9 ± 10.7% of their pre-trial MVC torque (range of 52–88% MVC). Following active shortening, steady-state isometric torque was significantly

less than that produced during the purely isometric contractions at the same corresponding muscle length and level of activation, resulting in an average TD across all contractions of 12.9 ± 8.8%. ($p < 0.05$—Fig. 3a). Participants successfully maintained the EMG target level such that EMG_{RMS} of both the soleus and tibialis anterior did not differ between the TD and ISO contractions at any of the three time points ($p > 0.05$—Fig. 4a–f). This indicates that motor neuron output was similar in both the TD and ISO states. Further, when compared among time points within a contraction type, EMG_{RMS} was not different for either the soleus or tibialis anterior ($p > 0.05$), indicating that motor neuron output was consistently maintained throughout each contraction.

Evoked muscle responses in the torque-depressed state

There was no significant difference in Mmax peak-to-peak amplitude between the TD and purely ISO states ($p > 0.05$—Fig. 3b). In the ISO state, the peak-to-peak amplitudes of CMEPs and MEPs were 9.7 ± 3.3% and 10.0 ± 3.1% of the resting Mmax, respectively, and were not significantly different from each other ($p > 0.05$). When normalized to Mmax (CMEP/Mmax), CMEP amplitude was 9.5% lower during TD contractions (8.7 ± 3.3% of Mmax) as compared with ISO contractions ($p < 0.05$—Fig. 3c). In contrast, when MEPs were normalized to CMEPs (MEP/CMEP), MEP amplitude was 16.8% greater in the TD (131.4 ± 54.2% of CMEP) than ISO (112.5 ± 41.4% of CMEP) state ($p < 0.05$—Fig. 3d).

Discussion

By holding activation of the soleus constant during a series of submaximal dorsiflexion contractions, the present study compared spinal and supraspinal excitability of an antagonist muscle during the TD versus ISO state. Following active shortening of the dorsiflexors, the normalized CMEP and MEP amplitudes were smaller and larger, respectively compared to the purely isometric condition. Therefore, as proposed in the hypothesis, the TD state is associated with lower spinal excitability and greater cortical excitability, when measured at an antagonist muscle. These findings indicate that the history-dependent properties of muscle can influence neural activation of antagonist muscles and the production of net torque about a joint during voluntary contractions of a postural muscle.

Torque depression and antagonist co-activation

To our knowledge, the present study was the first to successfully record TD in an agonist muscle group while holding antagonist co-activation constant. Torque depression was

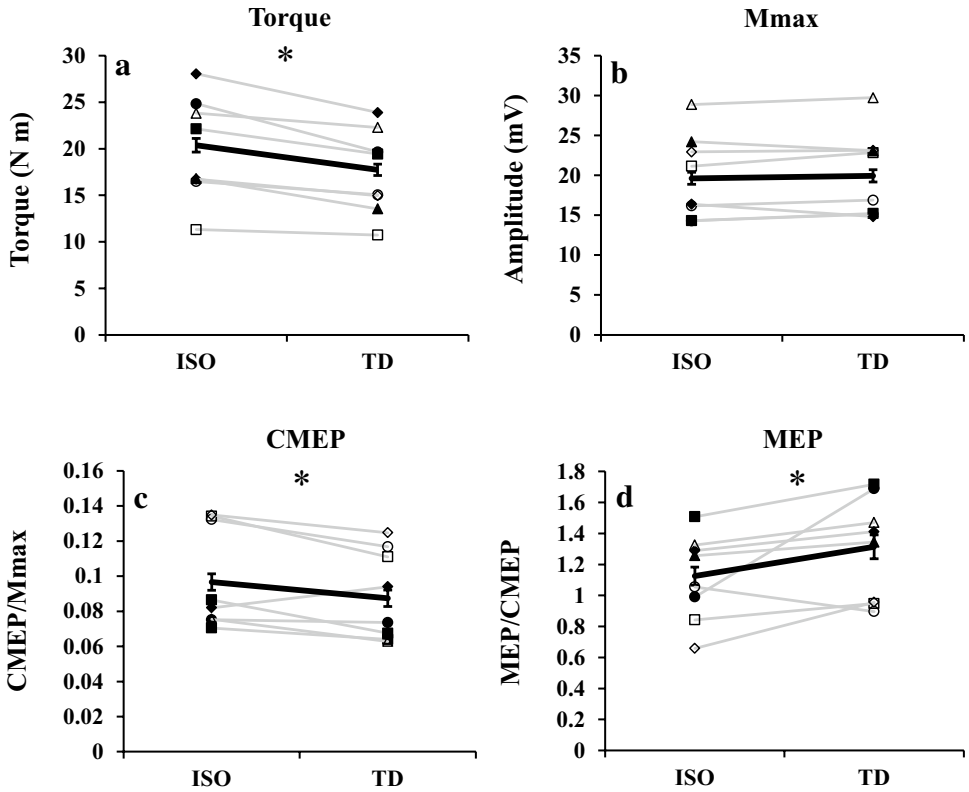

Fig. 3 Mean values for each participant (grey lines) and the group mean (black line; error bars indicate standard error of the mean) in the TD and ISO states. There was a 12.9% reduction in torque (**a**), a 9.5% decrease in normalized CMEP (**c**), and a 16.8% increase in normalized MEP (**d**) in the TD state as compared to the ISO state (*$p < 0.05$). There was no difference in Mmax peak-to-peak amplitude (**b**) between TD and ISO conditions ($p > 0.05$)

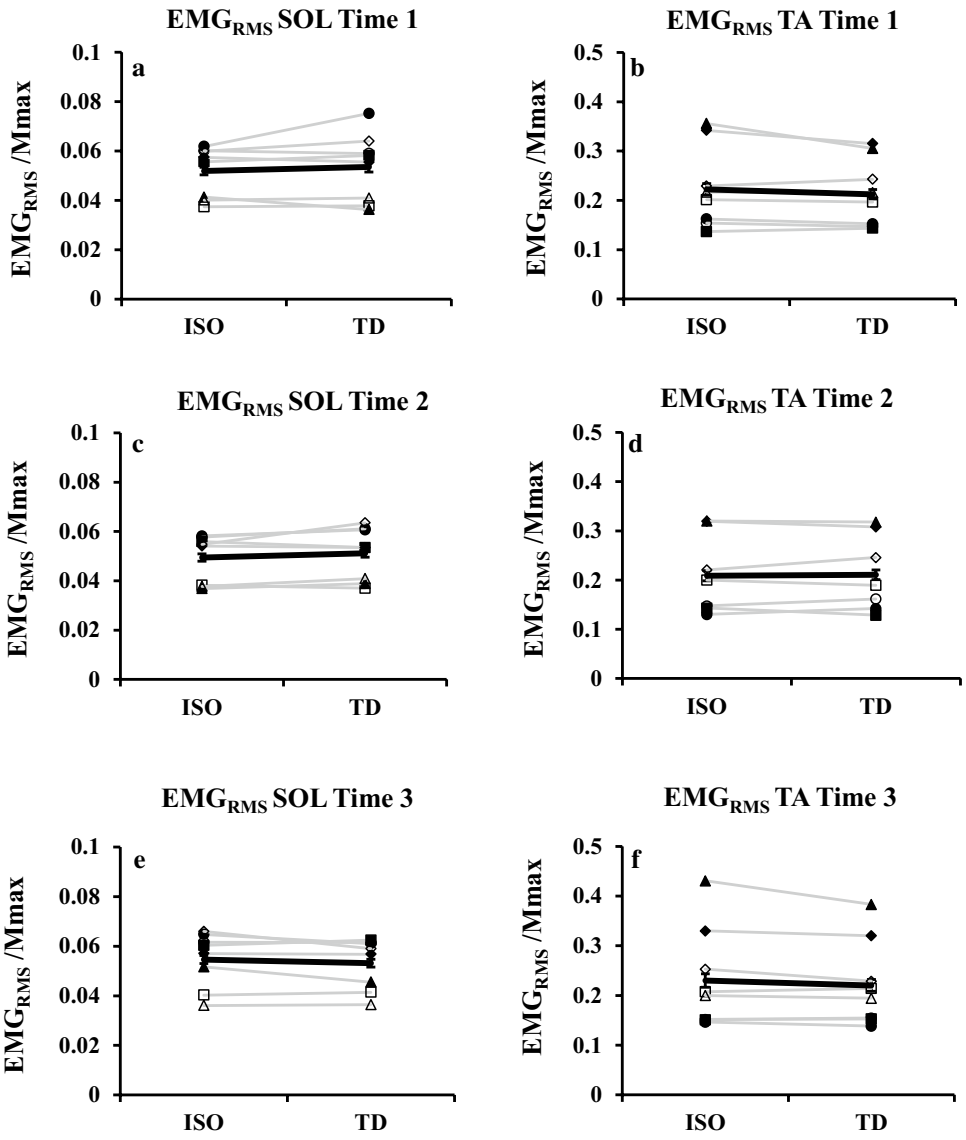

Fig. 4 Mean values for each participant (grey lines) and the group mean (black line; error bars indicate standard error of the mean) in the TD and ISO states. For both the soleus and tibialis anterior, there was no difference in EMG data between the TD and ISO states or among the three time points (**a**–**f**; $p > 0.05$)

observed for each participant, and the range of average TD values (4.7–20.8%) is in line with earlier studies involving voluntary contractions in humans (Lee et al. 1999; Lee and Herzog 2003; Rousanoglou et al. 2007; Tilp et al. 2009; Power et al. 2014; Jones et al. 2016; Grant et al. 2017; Sypkes et al. 2017; Paquin and Power 2018). As the torque about a joint is dependent on the force production of more than the prime mover, this protocol considered the activation of both an agonist and antagonist muscle to improve our understanding of neural control of joint torque during the TD state. Tibialis anterior EMG_{RMS} was used as a measure of agonist activation and, as was found in previous studies (Grant et al. 2017; Sypkes et al. 2017), there was no significant difference in agonist activation between TD and ISO trials. Further, soleus EMG_{RMS} was used as a measure of antagonist activation. To match voluntary drive among all trials, participants were instructed to maintain soleus iEMG at a constant level throughout each contraction. As expected, given the instructions, there were no differences in antagonist activation between TD and ISO conditions. However, it is important to recognize that, although muscle activation as measured by surface EMG did not change, the present study demonstrated both spinal and supraspinal excitability were different in the TD state.

The mechanical and neural aspects of antagonist co-activation in the torque-depressed state

The primary theory for the underlying mechanisms of TD involves a stress-induced inhibition of cross-bridge attachment in the newly formed actin–myosin overlap zone, resulting in fewer cross-bridge attachments (Maréchal and Plaghki 1979; Lee et al. 2000; Joumaa et al. 2012). This inhibition of cross-bridge attachment appears to be due to

structural deformation of the thin actin filaments following active shortening (Joumaa et al. 2018). Upon entering the isometric steady-state, these deformed filaments form fewer attachments with thick myosin filaments, which reduces the muscle's capacity to produce force, when compared to a purely isometric contraction (Maréchal and Plaghki 1979). In addition to reduced numbers of cross-bridges, the TD state is associated with reduced force production per cross-bridge, which further contributes to the mechanical disadvantage of muscle in TD (Joumaa et al. 2012). Recent studies have shown that, in response to this mechanical disadvantage, there are alterations to corticospinal excitability in the TD state. For example, under submaximal conditions, spinal excitability of the agonist muscle is greater in the TD compared to ISO state (Sypkes et al. 2017). As well, under maximal conditions, it was shown that spinal and supraspinal excitability are negatively related, such that higher or lower spinal excitability leads to an opposite finding at the level of the motor cortex (Grant et al. 2017). These neural changes were proposed to be part of a neuromechanical coupling response initiated during a state of altered muscle force production capacity, which could modulate motor neuron output and the control of voluntary movement through the central nervous system.

There is reason to believe that this neuromechanical coupling response also applies to antagonist muscles during contractions about a joint. Antagonist co-activation is important in isometric tasks, as it improves joint stability during voluntary contractions (Latash 2018). However, at the same time, it reduces net joint torque production. For these reasons, the activation of antagonist muscle groups must also be considered in the TD state. The current study investigated neural excitability within the central nervous system during submaximal contractions in the TD state while antagonist co-activation of the soleus muscle was held constant. It was found that following active shortening, the antagonist (soleus) normalized CMEP amplitude was 10% lower in the TD than ISO state. This suggests that there is a reduction in excitability of the antagonist motor neuron pool in the TD state. Accompanying this lower spinal excitability was greater supraspinal excitability, as indicated by a normalized MEP that was 17% larger compared to the ISO state. Together, these findings demonstrate that the activation of antagonist muscles during submaximal voluntary contractions is influenced by the history-dependent properties of the agonist muscles. Further, the opposite results for spinal and supraspinal excitability in the TD state may reflect a strategy to maintain motor neuron output (as measured by surface EMG) when excitability is altered at one level of the central nervous system. However, because only the soleus was investigated, it is unknown whether spinal and supraspinal excitability of other antagonists muscles (in this case, medial and lateral gastrocnemius) would be affected similarly by TD.

Sensory afferent feedback as a possible mediator of neuromechanical coupling

The precise mechanisms of lower spinal excitability and greater supraspinal excitability in the TD state remain unknown. However, because contractions in the TD and purely ISO states were activation matched at 15% antagonist co-activation, and there were no changes observed in tibialis anterior EMG_{RMS} between the two contraction types, it is likely that changes in neural excitability are attributable to changes in sensory afferent feedback. One source of sensory afferent feedback that modulates the interaction between agonist and antagonist muscles originates from muscle spindles. Through Ia afferents, muscle spindles respond to changes in muscle length and limb position and resist muscle stretch by sending excitatory sensory feedback to the agonist motor neuron pool (Proske and Gandevia 2012). Along with a network of other excitatory and inhibitory inputs from a variety of interneurons and descending tract neurons, Ia afferents also activate Ia inhibitory interneurons that mediate the reciprocal inhibition of the antagonist motor neuron pool, reducing antagonist resistance and improving the efficiency of movement about a joint (Jankowska 1992; Crone and Nielsen 1994). However, given that muscle length, limb position, and level of activation were not different when compared between the TD isometric steady-state (> 3 s following the end of shortening) and purely isometric contractions, it would seem unlikely that the reciprocal inhibition mediated by Ia inhibitory interneurons underlies the results of this study.

The Golgi tendon organ (GTO), however, offers a potential mechanism for our observations. The GTO is a mechanoreceptor located in-series with the muscle and aponeurosis at the muscle–tendon junction and is responsible for monitoring muscle tension. Through fast-conducting Ib afferents, the GTO sends inhibitory sensory feedback to the agonist motor neuron pool and excitatory sensory feedback to the antagonist motor neuron pool (Jami 1992). The firing of Ib afferents is modulated in a tension-dependent manner (Stephens et al. 1975), and the feedback represents differences in whole-muscle force rather than forces related to single motor units (Prochazka and Gorassini 1998). In the present study, a 13% reduction in torque was likely accompanied by diminished Ib afferent firing. The subsequent reduction in excitatory sensory feedback to the antagonist motor neuron pool may have contributed to the attenuation in CMEP amplitude (i.e., reduced spinal excitability) that was recorded from the soleus in the TD state. Moreover, because the protocol required antagonist muscle activation to be matched in the TD and ISO states, a reduction in spinal excitability may

have been counterbalanced by a greater voluntary drive from the motor cortex, resulting in the observed larger normalized MEP amplitude (i.e., greater supraspinal excitability). These data indicate that the relationship between muscle force production capacity and sensory afferent feedback is important in voluntary neuromuscular control of not only agonist muscles but also antagonist muscles following active shortening. Future studies should more closely investigate the modulation of afferent feedback to better determine its role as a mediator of neuromechanical coupling in the history-dependence of force about a joint.

Conclusion

The present study investigated spinal and supraspinal excitability of an antagonist muscle following shortening-induced torque depression of an agonist muscle as compared to an isometric contraction at the same muscle length and level of activation. With submaximal dorsiflexion contractions performed according to a fixed level of soleus co-activation, it was shown that steady-state isometric torque was significantly reduced following active shortening compared to purely isometric contractions. It was also found that, when compared to the purely isometric state, torque depression was associated with lower spinal excitability (i.e., normalized CMEP amplitude) and greater supraspinal excitability (i.e., normalized MEP amplitude) of an antagonist muscle. Together, these findings indicate that the history-dependent properties of muscle can influence neural control of antagonist muscles and may alter the control of voluntary torque production about a joint.

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Author contributions CTS, LRB, CJM, and GAP contributed to the conception and design of the study; CTS, VC, and GAP performed experiments; CTS, CJM, and GAP analyzed and interpreted the data; CTS, CJM, and GAP prepared figures and drafted the manuscript; CTS, VC, LRB, CJM, and GAP edited and revised the manuscript.

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Compliance with ethical standards

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

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards. This article does not contain any studies with animals performed by any of the authors.

Informed consent Informed consent was obtained from all individual participants included in the study.

Data accessibility Individual values of all supporting data are accessible as supplementary material.

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