Activation reduction following an eccentric contraction impairs torque steadiness in the isometric steady-state

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Abstract

Background: The isometric steady-state following active lengthening is associated with greater torque production and lower activation, as measured by electromyographic activity (EMG), in comparison with a purely isometric contraction (ISO) at the same joint angle. This phenomenon is termed residual force enhancement (RFE). While there has been a great deal of research investigating the basic mechanisms of RFE, little work has been performed to understand the everyday relevance of RFE. The purpose of this study was to investigate whether neuromuscular control strategies differ between ISO and RFE by measuring torque steadiness of the human ankle plantar flexors.

Methods: Following ISO maximal voluntary contractions in 12 males (25 ± 4 years), an active lengthening contraction was performed at 15°/s over a 30° ankle excursion, ending at the same joint angle as ISO (5° dorsiflexion; RFE). Surface EMG of the tibialis anterior and soleus muscles was recorded during all tasks. Torque steadiness was determined as the standard deviation (SD) and coefficient of variation (CV) of the torque trace in the ISO and RFE condition during activation-matching (20% and 60% integrated EMG) and torque-matching (20% and 60% maximal voluntary contraction) experiments. Two-tailed, paired t tests were used, within subjects, to determine the presence of RFE/activation reduction (AR) and whether there was a difference in torque steadiness between ISO and RFE conditions.

Results: During the maximal and submaximal conditions, there was 5%–9% RFE with a 9%–11% AR (p < 0.05), respectively, with no difference in antagonist coactivation between RFE and ISO (p > 0.05). There were no differences in SD and CV of the torque trace for the 20% and 60% activation-matching or the 60% and maximal torque-matching trials in either the RFE or ISO condition (p > 0.05). During the 20% torque-matching trial, there were >37% higher values for SD and CV in the RFE as compared with the ISO condition (p < 0.05). A significant moderate-to-strong negative relationship was identified between the reduction in torque steadiness following active lengthening and the accompanying AR (p < 0.05).

Conclusion: It appears that while the RFE-associated AR provides some improved neuromuscular economy, this comes at the cost of increased torque fluctuations in the isometric steady-state following active lengthening during submaximal contractions.

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1. Introduction

Residual force enhancement (RFE) is an intrinsic property of skeletal muscle and is characterized by an increase in isometric steady-state force following an eccentric (i.e., active lengthening) contraction, as compared with a purely isometric contraction (ISO) without prior lengthening.1,2 This phenomenon has been observed from single sarcomeres to3 the whole human level during electrically stimulated4 and voluntary contractions.5,6 While the mechanisms of RFE are still under debate,7 it is clear that there is a greater contribution of passive force to overall total force production following active lengthening, as compared with an ISO.8–11 Moreover, when matching isometric force (or torque) levels, the greater contribution of passive force in the force-enhanced state results in an activation reduction (AR), which is observed as a reduction in electromyographic activity (EMG)5,12,13 and adenosine triphosphate usage.14 Additionally, during torque- and activation-clamped experiments, the RFE EMG–torque relationship is shifted to the right as compared with ISO,15 indicating lower activation to achieve a given torque level. The findings of a...
shifted EMG–torque relationship may indicate altered motor unit (MU) recruitment and rate coding strategies in the RFE state. Furthermore, a reduction in the number of active MUs is suggested to contribute to the AR in the RFE state. Recent modeling experiments indicated that a greater number of active MUs facilitate the production of steady isometric forces. Therefore, such alterations to neuromechanical coupling could be reflected in a divergence in torque steadiness in the ISO and RFE isometric steady-state following active lengthening, on the basis of reduced MU activation in the RFE state.

Torque steadiness represents the ability of an individual to precisely and accurately sustain a target torque level. Meanwhile, fluctuations in torque during voluntary contractions can produce variability in movement. Typically, lower torque fluctuations are associated with greater fine movement control. Changes in torque steadiness (or force steadiness) have been observed in relation to age, sex, fatigue, physical activity level, contraction intensity, and clinical conditions such as Parkinson’s disease. These variations in torque steadiness have been attributed to changes in MU size and motor neuron (MN) pool activation strategies. Furthermore, it appears that torque steadiness depends mostly on the discharge characteristics of recruited MUs rather than on their force production capacity, as demonstrated by the relationship between increasing variability of MU discharge rate and increased force fluctuation. For lower contraction intensities (e.g., 20% maximal effort), there is reduced torque steadiness and a higher MU discharge variability than at higher intensity contractions. Eccentric contractions have also been shown to have greater torque fluctuations compared with concentric contraction or ISO, which is likely due to the lower activation and therefore lower MU discharge rate that occurs in eccentric contractions. Collectively, torque steadiness represents the cumulative activity of the MN pool, so the measurement of torque steadiness provides a quantitative measure of effective control signals from the nervous system. Therefore, fluctuations in torque during a submaximal task represent an important aspect of motor control, and measuring torque steadiness can offer insight into the control (or lack thereof) during everyday contractions that involve preceding movement/activation.

While there has been a great deal of research investigating the basic mechanisms of RFE, little work has attempted to understand its everyday relevance. Therefore, the purpose of this study was to investigate whether neuromuscular control strategies differ between ISO and RFE by measuring torque steadiness during both torque- and activation-matching conditions. Torque steadiness (i.e., standard deviation (SD) and coefficient of variation (CV) of the torque trace) is not expected to differ during the activation-matching experiments because MU recruitment factors potentially affecting torque steadiness should not differ. However, torque is expected to be less steady in the RFE conditions during the torque-matching experiments, owing to the stretch-induced decrease in activation necessary to achieve an equivalent torque output as in the ISO condition.

2. Methods

2.1. Participants

Data were collected within a single testing session from 12 healthy males (25 ± 4 years, 178 ± 7 cm, and 75 ± 9 kg). Males who were 18–35 years old without a history of neurologic, vestibular, and neuromuscular disorders or musculoskeletal injuries in the lower limb were recruited. Participants gave informed, written consent, and all procedures were approved by the University of Guelph Research Ethics Board and conformed to the Declaration of Helsinki.

2.2. Experimental set-up

A HUMAC NORM dynamometer (CSMi Medical Solutions, Stoughton, MA, USA) was used to record all torque, angular velocity, and position values. The participants were seated with their right hip at 90° of flexion and right knee at 100° of extension, with their medial malleolus aligned with the dynamometer’s axis of rotation. Flexion at the knee reduced the contributions of the gastrocnemius to plantar flexion (PF) torque, allowing for changes in PF EMG to be attributed primarily to the soleus. The right knee was immobilized with the dynamometer’s leg restraint positioned superiorly over the distal femur. Movement at the torso was restricted with a 4-point seatbelt harness. The right foot was fixed to a dorsi/plantar flexor dynamometer foot pedal adaptor with 1 inelastic strap and a custom-built ratchet tightening strap positioned over the ankle and another inelastic strap at the mid-dorsal portion of the metatarsals. The maximum PF and ankle dorsiflexion (DF) angles were set to 25° and 5°, respectively, allowing for 30° of ankle excursion to optimize stretch on the plantar flexors.

Prior to EMG electrode placement, skin locations were thoroughly shaven and cleaned with alcohol. Silver-silver chloride (Ag/AgCl) electrodes (1.5 cm × 1.0 cm; Kendall, Mansfield, MA, USA) were used for all EMG recordings. As described previously, 1 electrode was placed on the soleus along the midline of the lower leg approximately 2 cm inferior to the border of the gastrocnemius muscle, a second electrode was placed on the calcaneal tendon, and a ground electrode was placed on the patella. To record antagonist muscle activation, 1 electrode was positioned over the right tibialis anterior approximately 7 cm inferior and 2 cm lateral to the tibial tuberosity, and a second electrode was placed over the distal tendon of the tibialis anterior. Torque, angular position, and stimulus trigger data were sampled at 1000 Hz using a 12-bit analog-to-digital converter (PowerLab System 16/35; ADInstruments, Bella Vista, Australia). The EMG data were sampled at 2000 Hz and band pass filtered between 10 and 1000 Hz. All data were analyzed with LabChart software (Version 8.0; ADInstruments).

2.3. Nerve stimulation and maximal voluntary contractions (MVCs)

To normalize voluntary EMG, maximal compound muscle action potentials (M_max) at 5° DF were recorded from the
soleus and tibialis anterior muscles by stimulating the tibial and deep fibular nerves, respectively, with a standard clinical bar electrode (Empi, St. Paul, MN, USA) coated in conductive gel. The tibial nerve innervating the plantar flexors was found by locating the distal tendon of the semitendinosus muscle and moving laterally while palpating deep into the popliteal fossa. The deep fibular nerve was located by palpating the head of the fibula and moving posteriorly and laterally. The root mean square EMG (EMGRMS) of the tibialis anterior M\text{max} was used to normalize and quantify antagonist coactivation. All peripheral nerve stimuli were evoked with a single pulse from a constant current high-voltage stimulator (model DS7AH; Digitimer, Welwyn Garden City, UK). Pulse width was set to 200 ms, and voltage was set at 400 V. Current was increased gradually until a peak-to-peak M\text{max} amplitude plateau was reached. To ensure the activation of all MUs, the current was adjusted to a supramaximal level, equivalent to 120% of that required to generate M\text{max}.

Participants performed 2–3 brief (2–3 s) MVCs at 5° DF with 5 min of rest between contractions. During each MVC attempt, participants were instructed to perform PF as hard and fast as possible, with visual feedback provided as a torque trace on a computer monitor during all contractions. Participants were verbally encouraged during each MVC. The interpolated twitch technique was used to ensure near maximal voluntary activation (VA) during MVCs. The interpolated twitch technique was used to ensure near maximal voluntary activation (VA) during MVCs. The torque resulting from a superimposed twitch administered manually during the plateau phase of the MVC was compared with a resting reference twitch delivered 1–2 s after the MVC, once the participant was fully relaxed. All participants were required to reach a minimum of 95%VA before moving forward with the test protocol. The level of VA was assessed as:

$$\text{VA\%} = \left(1 - \frac{\text{interpolated twitch torque}}{\text{resting twitch torque}}\right) \times 100\%.$$  

2.4. Determining torque and activation levels for torque steadiness trials

To determine the submaximal torque and activation (i.e., integrated EMG (iEMG)) levels for the torque steadiness trials, participants were instructed to perform a maximal PF contraction at 5° DF for 12 s using the peak value from the 3 s MVC as a guideline. The 20% and 60% torque levels were calculated from the average of the 12 s maximum trial between 6.5 and 7 s. Similarly, the 20% and 60% iEMG activation levels were calculated from the average of this maximum trial for the iEMG between 6.5 and 7 s. For all subsequent dynamic contractions and ISOs, the torque- and activation-matching task required the participant to match each target value as steadily as possible, with near real time feedback of the torque and iEMG trace, by contracting the plantar flexors for 12 s. The target value was displayed as a horizontal line on a computer monitor positioned 1.5 m from the participant, with the gain for visual feedback scaled to 20%MVC and a horizontal scaling of 10:1.

2.5. Experimental procedures

The following contractions were all 12 s in duration and consisted of a purely ISO and an active lengthening contraction into an ISO (RFE) condition (Fig. 1B). After each contraction, the participants were given 5 min of rest to minimize fatigue. The lengthening contractions were divided into
3 distinct yet continuous phases: a 1 s preactivation to the target level at an ankle angle of 25° PF, a 2 s lengthening at 15°/s while trying to match the target level, and a 9 s steady-state isometric phase at an ankle angle of 5° DF trying to match the target level (Fig. 1B). The ISO contractions were performed at an ankle angle of 5° DF and held for 12 s. The first 2 test contractions performed were maximal effort; as noted above, the torque and iEMG recorded during the ISO MVC between 6.5 and 7 s were deemed the participant’s peak torque and peak iEMG. The intensities of all the submaximal contractions were based on these values. Prior to the actual experimental submaximal contractions, participants were asked to practice matching their torque and iEMG to ensure accuracy during the actual test contractions. All submaximal contractions were performed in a randomized order and were either torque clamped (i.e., matching torque) or iEMG clamped (i.e., matching activation) at 20% or 60% of MVC and iEMG/MVC, respectively. Once all experimental contractions were completed, an MVC was performed and VA was assessed to gauge the level of fatigue and determine whether the participants were still able to achieve pretrial MVC torque values and near-maximal activation.

2.6. Data analysis and statistics

Mean torque, torque SD, and EMGRMS were analyzed over 7–11 s of the isometric portion of each contraction (Fig. 1C). The beginning and end of each contraction were disregarded to avoid any excessive fluctuations during the dynamic portion of the contraction at the beginning of the trial and in anticipation of the end of the trial. For the 4 s time epoch during the isometric phase, the CV of torque was calculated as CV = (SD/mean torque) × 100%. As described previously, RFE during the maximal and submaximal activation-clamp conditions and AR during the maximal and submaximal torque-clamp conditions were defined as a percent increase or decrease, respectively, from the isometric reference torque and EMGRMS values obtained from 7 to 11 s during the ISO condition and the RFE isometric steady-state following active lengthening:

\[ \text{RFE/AR}\% = \left( \frac{\text{Isometric following active lengthening} - \text{Isometric}}{\text{Isometric}} \right) \times 100\%. \]

Two-tailed paired t tests were performed to compare, within subjects, (1) the torque and EMG data between RFE and ISO trials to confirm the presence of RFE and AR, respectively; and (2) SD and CV within a given contraction intensity for RFE and ISO conditions. Linear regression analyses were conducted to determine the relationship between torque steadiness and RFE/AR. Sigma Stat 4.0 (Systat Software Inc., San Jose, CA, USA) was used for all statistical analysis. Significance was determined based on an α = 0.05. Descriptive data found in text and figures are reported as mean ± SD and mean ± SE, respectively.

3. Results

3.1. MVCs and VA

Pretrial MVC peak torque amplitude was 123.1 ± 20.1 N-m (range: 90.2–146.1 N-m), and all participants were capable of achieving near maximal values for VA as assessed using the interpolated twitch technique (98% ± 1%). Following the torque steadiness trials, MVC and VA were not different from prevalues (MVC: 122.1 ± 20.1 N-m; VA: 97% ± 2%, p > 0.05).

3.2. RFE and AR

Owing to large differences in strength across participants, a within-subject design was chosen. For the maximal condition, torque amplitude (averaged over a 4 s epoch from 7 to 11 s) was consistently greater (p < 0.05) in the RFE (121.5 ± 22.7 N-m) compared with the ISO condition (116.1 ± 20.5 N-m; Fig. 2), with no difference in antagonist coactivation (RFE: 14% ± 5%, ISO: 14% ± 5%, p > 0.05). Similarly, during the 20% and 60% activation-matching trials, torque was higher (p < 0.05) in the RFE (20%: 28.2 ± 11.4 N-m; 60%: 82.1 ± 15.5 N-m) compared with the ISO condition (20%: 27.6 ± 9.2 N-m; 60%: 77.7 ± 15.9 N-m; Fig. 2), with no difference in antagonist coactivation (RFE: 8%–11%, ISO: 8%–11%, p > 0.05). During the submaximal 20% and 60% torque-matching trials, there was an AR of 11.3% ± 7.3% and 9.3% ± 12.1% (p < 0.05), respectively, for the RFE compared with the ISO condition (Fig. 2), with no change in antagonist coactivation (RFE: 8%–10%, ISO: 8%–10%, p > 0.05).

3.3. Torque steadiness

The SD and CV values for torque fluctuation were used to assess the participants’ ability to sustain a steady contraction at a constant torque (%MVC) and activation level (%EMG/MVC). There were no differences in SD and CV for the 20% and 60% activation-matching trials in either the RFE or ISO condition (Fig. 3; p > 0.05). For both the maximal and 60% torque-matching trials, there were no differences in SD or CV between the RFE and ISO trials (Fig. 3; p > 0.05). However, during the 20% torque-matching trials, there were ~37% higher values for SD and CV in the RFE as compared with the ISO trial (Fig. 3; p < 0.05), indicating a reduction in torque steadiness following active lengthening in the RFE.

Fig. 2. RFE values during the maximal, 20%, and 60% activation-matching experiments and the AR values during the 20% and 60% torque-matching experiments (mean ± SE) shown as a percentage change from ISO. * indicates a significant difference from ISO (p < 0.05). AR = activation reduction; ISO = isometric contraction; RFE = residual force enhancement.
isometric steady-state compared with the ISO condition. There was a significant moderate-to-strong negative relationship between the reduction in torque steadiness for the RFE torque-matching 20% condition and the accompanying AR following active lengthening (Fig. 4; \( p < 0.05 \)).

4. Discussion

The present study was designed to investigate torque steadiness in the isometric steady-state following active lengthening (RFE) compared with that of a purely ISO. Experiments were performed during maximal and submaximal contractions (20% and 60%) during both a TC (i.e., matching a percentage of one’s maximum voluntary contraction torque) and AC (i.e., matching a percentage of one’s maximum voluntary contraction integrated electromyographic activity). Torque steadiness only differed in the 20% torque-matching condition, with higher SD and CV values in the RFE as compared with the ISO condition (mean ± SE). *\( p < 0.05 \). AC = activation clamp; CV = coefficient of variation; ISO = isometric contraction; RFE = residual force enhancement; SD = standard deviation; TC = torque clamp.

Fig. 3. SD (A) and CV (B) of the torque and integrated electromyographic activity trace during the isometric steady-state following active lengthening (RFE) and purely ISO. Experiments were performed during maximal and submaximal contractions (20% and 60%) during both a TC (i.e., matching a percentage of one’s maximum voluntary contraction torque) and AC (i.e., matching a percentage of one’s maximum voluntary contraction integrated electromyographic activity). Torque steadiness only differed in the 20% torque-matching condition, with higher SD and CV values in the RFE as compared with the ISO condition (mean ± SE). *\( p < 0.05 \). AC = activation clamp; CV = coefficient of variation; ISO = isometric contraction; RFE = residual force enhancement; SD = standard deviation; TC = torque clamp.

Fig. 4. SD (A) and CV (B) of the torque trace during the purely ISO condition and SD (C) and CV (D) of the isometric steady-state following active lengthening (RFE) for the 20% torque-matching experiments (TC) plotted against percent AR in the RFE condition. There were no significant relationships in the ISO state between torque steadiness and TC. However, there was a significant relationship in the RFE state with ~19% of the variability in torque steadiness explained by a reduction in AR in the force-enhanced state following active lengthening (*\( p < 0.05 \)). AR = activation reduction; CV = coefficient of variation; ISO = isometric contraction; RFE = residual force enhancement; SD = standard deviation; TC = torque clamp.
trial compared with the ISO, which was associated with an AR. It appears that while AR associated with RFE provides some improved neuromuscular economy, this comes at the cost of increased torque fluctuations in the RFE state during low-level submaximal contractions (i.e., 20%MVC).

4.1. RFE and AR

In agreement with most studies investigating RFE during voluntary contractions, active lengthening resulted in a greater steady-state isometric torque (i.e., RFE) compared with that of the ISO condition at the same muscle length. In the present study, during submaximal activation-matching experiments, a 5%–9% increase in torque was present (Fig. 2), which was similar to reports on the increases of ~14% in the adductor pollicis, 4,12,37 4%–5% in the knee extensors, 35,38 7%–13% in the plantar flexors, 39 and 9%–16% in the dorsiflexors. 39–41 Likewise, activation was reduced by 9%–11% in the RFE compared with the ISO condition for both the 20% and 60%MVC torque-matching trials, which is in line with previously reported values. 12,13,17,33,39 The AR that occurred in the torque-matching trials of this study can be explained in part by the proposed mechanisms for RFE. Although it was beyond the scope of this study, it seems that a greater contribution of passive force to total force production following active lengthening as compared with the ISO condition is responsible for RFE. 8–11 It has recently been suggested that the increase in passive force following active lengthening is due to Ca2+-dependent stiffening of the giant protein titin. 42,43 Consequently, in the RFE state, less active force is required to produce similar force levels to that of the ISO condition, ultimately contributing to the AR observed during torque-matching trials in our study.

4.2. Torque steadiness and AR

Torque steadiness during the RFE and ISO conditions was assessed by measuring the SD and CV of the torque trace. The only difference in torque steadiness between the RFE and ISO condition was found during the 20% torque-matching trial. Torque steadiness can be influenced by a variety of mechanisms, such as MU size, MN pool activation strategies, and contraction intensity. 18,20,23,25,26 Although not the main outcome of our study, for the ISO conditions, our data are in agreement with previous work demonstrating that SD increases with torque intensity. 20 However, CV is a measure of torque fluctuation normalized to the mean torque of the contraction; as torque intensities increased, the CV value tended to decrease. As noted above, the only difference in torque steadiness between the RFE and ISO conditions was found during the 20% torque-matching trial. With no difference in torque steadiness for the 20% activation-matching condition, this serves as a negative control, emphasizing that the AR associated with RFE somehow contributed to impaired torque steadiness in the isometric steady-state following active lengthening. It is unclear why there was no difference in torque steadiness at the higher contraction intensities; however, this finding appears consistent with the literature. 23 Importantly, torque steadiness represents the cumulative activity of the MN pool and provides a quantitative measure of effective control signals from the central nervous system. Based on these findings, RFE may alter the voluntary control of muscular force.

4.3. Possible alterations to MU activity and torque steadiness in the RFE state

While it is clear that RFE is an intrinsic property of skeletal muscle, when considered in the context of voluntary contractions, RFE is not solely mechanical in nature and influences the neuromechanical coupling of activation and voluntary control of force. RFE potentially altered MU activation strategies, as shown with a reduction in activation during the isometric steady-state that was associated with increased torque fluctuations (Fig. 4). In terms of relevance of RFE for human locomotion, larger muscle groups have been investigated and yielded a similar AR of 6%–9% in the RFE state. Despite reduced activation following active lengthening of the knee extensors, MU firing rate was not altered; thus, it appears that a derecruitment of MUs may be responsible for AR. 16,17 Therefore, in the present study, the 11%AR for the 20% torque-matching experiments was likely due to a reduction in the number of active MUs. An AR coupled with this low-torque matching task could create an environment where there were not enough MUs firing asynchronously to create a smooth torque output. If torque unsteadiness represents the unfused tension of active MUs and grouped MU firing, 44 a relationship between torque steadiness and AR in the RFE condition would likely seem plausible. The grouping of MU firing has been attributed to an increase in the gain in the stretch reflex loop, and there appears to be an association between an increased stretch reflex and greater force fluctuations. 47,48 Similarly, a reduction in MN excitability (i.e., smaller H-reflex) has been associated with fewer force fluctuations owing to a reduction of peripheral afferent input to the MN pool. 49 Although not measured in the present study, the RFE condition has been reported to be associated with an increase in cortical excitability and greater neuromotor output or increased stretch reflex excitability as compared with the ISO. 50 Therefore, in the present study, the reduction in torque steadiness in the RFE compared with the ISO condition may be a result of afferent feedback creating a differential modulation of the MN pool excitability in the history-dependent state; however, this idea requires further investigation.

5. Conclusion

In the present study, we show a reduction in torque steadiness in the torque-enhanced isometric steady-state compared with an ISO without prior lengthening. This torque unsteadiness in the torque-enhanced state was associated with an AR when it was required to match the same torque level as the purely isometric condition. While RFE is considered an intrinsic property of skeletal muscle, when considered in the context of voluntary control of force, this history-dependent property alters neuromechanical coupling and can influence voluntary neuromuscular control strategies used in everyday movements.
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Authors’ contributions

NM and AJH contributed equally. All authors contributed to the conception and design of the study; acquisition, analysis, and interpretation of data; and drafting the manuscript. All authors have read and approved the final version of the manuscript, and agree with the order of presentation of the authors.

Competing interests

The authors declare that they have no competing interests.

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