Acute effect of muscle stretching on the steadiness of sustained submaximal contractions of the plantar flexor muscles

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Acute effect of muscle stretching on the steadiness of sustained submaximal contractions of the plantar flexor muscles. J Appl Physiol 110: 407–415, 2011. First published December 2, 2010; doi:10.1152/japplphysiol.01087.2010.— This paper examines the acute effect of a bout of static stretches on torque fluctuation during an isometric torque-matching task that required subjects to sustain isometric contractions as steady as possible with the plantar flexor muscles at four intensities (5, 10, 15, and 20% of maximum) for 20 s. The protocol comprised five 60-s passive stretches, separated by 10-s rest. During the torque-matching tasks and muscle stretching, the torque (active and passive) and surface electromyogram (EMG) of the medial gastrocnemius (MG), soleus (Sol), and tibialis anterior (TA) were continuously recorded. Concurrently, changes in muscle architecture (fascicle length and pennation angle) of the MG were monitored by ultrasonography. The results showed that during stretching, passive torque decreased and fascicle length increased gradually. Changes in these two parameters were significantly associated ($r^2 = 0.46; P < 0.001$). When data from the torque-matching tasks were collapsed across the four torque levels, changes in fascicle length correlated with changes in passive torque ($r = 0.05$) and enhanced EMG activity ($P < 0.005$) in MG and TA muscles with no change in coactivation. Furthermore, stretching maneuvers produced a greater decrease (~15%; $P < 0.001$) in fascicle length during the torque-matching tasks and change in torque fluctuation (CV) was positively associated with changes in fascicle length ($r^2 = 0.56; P < 0.001$), MG and TA EMG activities, and coactivation ($r^2 = 0.35, 0.34$, and $0.35$, respectively; $P < 0.001$). In conclusion, these observations indicate that repeated stretches can decrease torque steadiness by increasing muscle compliance and EMG activity of muscles around the joint. The relative influence of such adaptations, however, may depend on the torque level during the torque-matching task.

motor control; electromyography; ultrasonography; force fluctuation; muscle architecture

MUSCLE STRETCHING IS COMMONLY used in rehabilitation programs and sports activities to increase the range of motion (ROM) of a joint (20, 32) and, although still controversial, to prevent muscle injury (18, 34). In addition to neural adjustments (17), the increase in ROM after acute muscle stretching is attributed to changes in mechanical properties of the muscle-tendon unit (MTU; 48, 50). The MTU elongates when forcibly lengthened and recovers its initial length when the force is removed. However, due to their viscoelastic properties, these tissues become transiently less stiff after passive stretching (44, 47). For example, passive stiffness of the triceps surae MTU was decreased after a bout of static stretches of the plantar flexor muscles and likely contributed to the concurrent increase in ROM of the ankle joint observed after static stretching (37). In addition to a decrease in tendon stiffness (outer tendon and aponeuroses; 25, 30), the reduced MTU stiffness after acute stretching has also been attributed to an increase in the extensibility of the muscular portion of the MTU, including contractile, aponeuroses, and connective tissues (37). Such an increase in tendinous tissue compliance (i.e., decrease in stiffness) may influence the level of muscle activation required to develop the same absolute torque than before stretching. Indeed, taking up the slack of a more compliant series elastic component would place the sarcomeres at a more shortened length and, according to the length-tension relation (10, 15, 42), would require greater activation to develop the same absolute force as before stretching.

Interestingly, when an individual performs a submaximal contraction to match a target torque as steady as possible, the exerted torque fluctuates about the target (11), and it has been shown that motor unit discharge characteristics influence torque steadiness at a single muscle level (36). However, when the action involves multiple muscles, torque fluctuation is also influenced by the distribution of activity among synergist and antagonist muscles (13, 16, 46). For example, torque fluctuation is greater in the plantar flexor muscles of the ankle when the knee is in a flexed position, in which the contribution of the gastrocnemius muscles is minimal, than when it is extended (51). Although age-related increase in antagonist coactivation does not appear to play a key role in the alteration of torque steadiness in a hand muscle (7), it is not excluded, however, that an acute change in compliance of the series elastic component of one of the agonist-antagonist muscle pair might alter torque steadiness during a steady contraction. Indeed, the increase in agonist activation due to greater MTU compliance after acute stretching may not be associated with a proportional increase in antagonist activity. This would change the balance in the net torque produced around the joint by the agonist-antagonist muscle pair and, as a consequence, would influence torque steadiness.

A lower torque or movement fluctuation is usually associated with a greater dexterity (see Ref. 29) and, therefore, fluctuations in muscle torque during submaximal contractions represent an important aspect of the ability of individuals to control movement. However, acute muscle stretching is frequently used in warm-up routine preceding sports competition and training despite little being known about its effect on the ability to accurately control force output. Therefore, the purpose of the current study was to determine the acute effect of a bout of static stretches of the plantar flexor muscles on torque fluctuations during a torque-matching task performed in isometric conditions. In addition, this work analyzed associations between changes in torque steadiness and muscle architecture and activation. As muscle stretching increases the compliance
METHODS

Fourteen healthy subjects (10 men and 4 women) participated in the experiment (age; 26.9 ± 4.4 yr; height 175.6 ± 7.6 cm; weight 72.4 ± 11.3 kg; mean ± SD). They were recruited from the university population and were all moderately active and recreationally trained but not engaged in systematic stretching programs. All subjects were accustomed to the experimental procedure and had no signs or reported history of neuromuscular or orthopedic disorders. Subjects did not participate in any physical activity at least 1 day before the experiment. The current study was approved by the local Ethics Committee and all experimental procedures were performed in accordance with the Declaration of Helsinki.

Experimental Set-Up

The protocol included the assessment of maximal plantar flexion torque and torque steadiness during isometric submaximal contractions performed before and after acute static stretching of the plantar flexor muscles. During the experimental session, the subject laid prone on a table with both legs extended and the right foot secured by three straps to a footplate equipped with an adjustable heel block (1). The first strap was placed over the dorsum of the foot, and two other straps were placed around the ankle. The angular displacement of the ankle joint was monitored with a potentiometer that was mounted on the rotational axis of the footplate. The ankle joint axis for flexion-extension was visually aligned with the rotational axis of the footplate. The footplate was connected by a steel cable to a mechanical device that enabled graduated passive dorsiflexion of the ankle.

Mechanical and EMG Recordings

The torque produced by the plantar flexor muscles during a maximal voluntary contraction (MVC) and submaximal contractions and the passive torque developed during static stretching were recorded by means of a strain-gauge transducer (model TC 2000–500, linear range 0–1100 N; Kulite, Basingstoke, UK) attached to the footplate. The rectified average EMG (aEMG) activity was recorded from the medial gastrocnemius (MG), soleus (Sol), and tibialis anterior (TA) muscles by means of two silver surface electrodes (disks of 8 mm in diameter) in a bipolar configuration and with an inter-electrode distance of 20 mm. To obtain low impedance at the skin-electrode interface, the skin was shaved when necessary and cleaned with a solution of alcohol, ether, and acetone. The electrodes were filled with electrode gel and positioned longitudinally over each muscle belly. The proximal electrode of the pair was placed over the mid-belly of the medial head of the gastrocnemius for the MG and 2–3 cm below the insertion of the gastrocnemius on the Achilles tendon and slightly lateral from the tendon for the Sol. For the tibialis anterior, the proximal electrode was placed over the muscle belly at about two-thirds of the distance between the lateral malleolus and the fibular head. The ground electrodes were placed over the tibia. The EMG signals were amplified by a custom-made differential amplifier (×1,000) and filtered (10 Hz to 1 kHz; common mode rejection ratio: 120 dB). The signals (ankle joint angle, torque, and EMG activities) were A/D sampled at 1,000 Hz (MP 150, Biopac Systems, Santa Barbara, CA) and stored on a computer for subsequent analysis.

Ultrasoundographic Recordings

The architectural changes of the MG muscle during static stretching and submaximal contractions were investigated by ultrasonography. Longitudinal images were obtained using a real-time B-mode ultrasonographic apparatus (DP-6600, Mindray Bio-Medical Electronics, Shenzhen, China) with a linear-array probe (75L38EA, Mindray Bio-Medical Electronics; 7.5-MHz wave frequency with 38-mm scanning length). The width and depth resolution of the images was 8.48 pixels/mm. The probe was fixed firmly onto the right leg over the mid-belly of the muscle (at 30% of the distance between the popliteal crease and the center of the medial malleolus) with a water-soluble transmission gel to provide acoustic contact. Once a muscle fascicle was clearly identified, the position of the probe was firmly held in place using a custom-made resin sheath strapped to the skin. The restraint ensured a constant orientation and pressure of the probe and an acoustic marker was placed between the skin and the ultrasound probe to verify that the probe did not move throughout the experiment. Furthermore, during the experiment, one of the experimenters visually checked that muscle thickness stayed relatively constant when the ankle was replaced in neutral condition (90°) between each stretch or after each contraction of the force-matching task. When measured on the ultrasound images, the variation in muscle thickness in neutral condition between those tasks was <0.5 mm (range among subjects: from −0.3 to +0.4 mm). The ultrasound images were acquired at a sampling rate of 25 Hz (Pinnacle Studio Plus 9, Avid, Tewksbury, MA) and synchronized with the torque, ankle joint angle, and EMG signals.

Experimental Procedures

For each subject, the experimental session began with the measurement of the maximal passive dorsiflexion ROM from the neutral position (0° = 90° between the foot sole and lower leg). The endpoint of the ROM was determined as the maximum possible dorsiflexed position tolerated by the subject without excessive pain and substantial increase in EMG activity. Thereafter, the subject produced isometric MVCs of the plantar flexor and dorsiflexor muscles with the ankle joint maintained in a neutral position. Each subject performed at least two MVCs with 2 min rest in between. Subsequent trials were performed if the difference in torque between the two MVCs was >5%. Subjects were instructed to produce maximal effort during the MVC, and strong verbal encouragement was provided by the investigators. The torque signal was displayed in real time to the subject on a 14-in. monitor by means of oscilloscope software (Pico Scope, version 5. 16. 2–32 bit, Picotechnology, Cambridgehire, UK). After 5 min of rest, subjects were asked to perform isometric contractions with the plantar flexor muscles at 5, 10, 15, and 20% MVC torque in a counterbalanced order across subjects. The torque-matching task required the subject to match each target torque as steady as possible by contracting the plantar flexor muscles for 20 s. The target torque was displayed as a horizontal line on the feedback monitor. These recordings were followed by a series of five 60-s static stretches of the plantar flexor muscles. During the stretching maneuvers, the ankle joint was maintained passively at an angle that was 5° less than the maximal dorsiflexion angle measured individually for each subject. We preferred to use a submaximal stretching condition than a maximal condition because in the former, all subjects were able to sustain this dorsiflexion angle while keeping the heel in contact with the footplate without discomfort and negligible EMG activity (≤2% MVC) of the plantar flexor muscles. A rest period of 10 s, with the ankle in a neutral position, was allowed between each stretching maneuver.

Two minutes after the end of the fifth stretch, torque-matching tasks were performed in the same order as before the stretching bout. The experiment ended with a reassessment of the MVC torque of the plantar flexor muscles.

Data Analysis

Torque and EMG. The torque produced during the isometric MVC was measured during a 2-s window centered around peak torque. The surface EMG of MG, Sol, and TA were rectified and averaged over the same period as the MVC. At each intensity of contraction for the
RESULTS

Passive Torque During Stretching Maneuvers

At the beginning of the experimental session, the subjects’ maximal passive ankle dorsiflexion varied from 25 to 45°. During the five stretching maneuvers, the ankle of each subject was passively moved to an angle that corresponded to 5° below their maximum dorsiflexion. This represented 85 ± 2% of their maximal dorsiflexion range. The greatest passive torque was recorded at the beginning of the first 60-s static stretch (Fig. 1 and Table 1). Thereafter, the passive torque decreased gradually from the first to the fifth stretching maneuver (Table 1). Furthermore, the passive torque decreased during each 60-s stretch with a gradual decline in the magnitude of this effect across the series of stretch maneuvers. At the beginning of the first stretch, the mean passive torque reached 35.4 ± 13.9 Nm and decreased progressively by 17.1 ± 7.4% at the end of the 60 s (Table 1). For the fifth stretch, the mean passive torque at the beginning of the stretch reached 29.9 ± 10.4 Nm and declined thereafter by only 9.6 ± 2.4%.

Musculotendinous Parameters and EMG Activity During Stretching

As indicated by the arrows in Fig. 2, FL initially shortened slightly (not significant, P = 0.92) during the first passive stretching maneuver before subsequently lengthening (Fig. 2), whereas passive torque declined gradually from the beginning of the stretch. In contrast, the decrease in passive torque was accompanied by a lengthening of FL throughout each of the five successive static stretches. Each value was computed during a 1-s period at the beginning and end of each 60-s stretch. Percent change values indicate the relative difference in passive torque between the beginning and end of each stretch. Stretch × time interaction, *P < 0.001; post hoc comparison, #P < 0.001.

Table 1. Passive torque during a bout of five static stretches

<table>
<thead>
<tr>
<th>Stretch #</th>
<th>Beginning, Nm</th>
<th>End, Nm</th>
<th>Change, Nm</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 st</td>
<td>35.4 ± 13.9 (21–53)</td>
<td>29.2 ± 11.2 (15–43)</td>
<td>17.1 ± 7.4*</td>
</tr>
<tr>
<td>2 nd</td>
<td>32.0 ± 12.2 (18–46)</td>
<td>28.4 ± 10.5 (16–41)</td>
<td>11.1 ± 1.4*</td>
</tr>
<tr>
<td>3 rd</td>
<td>31.1 ± 11.3 (18–45)</td>
<td>28.1 ± 10.3 (16–41)</td>
<td>9.7 ± 2.3*</td>
</tr>
<tr>
<td>4 th</td>
<td>30.5 ± 11.4 (18–44)</td>
<td>27.6 ± 10.2 (15–40)</td>
<td>9.5 ± 1.9*</td>
</tr>
<tr>
<td>5 th</td>
<td>29.9 ± 10.4 (18–42)</td>
<td>27.1 ± 9.4 (15–38)</td>
<td>9.6 ± 2.4*</td>
</tr>
</tbody>
</table>

Values are means ± SD (range) of passive torque at the beginning and end of the 5 successive static stretches. Each value was computed during a 1-s period at the beginning and end of each 60-s stretch. Percent change values indicate the relative difference in passive torque between the beginning and end of each stretch. Stretch × time interaction, *P < 0.001; post hoc comparison, #P < 0.001.

Fig. 1. Time course of change in the passive torque produced by the plantar flexor muscles during stretching. The different traces correspond to the mean values from all subjects for each of the five static stretches. For clarity, SEs have been omitted but are given in Table 1. The inset illustrates a typical torque recording during a single static stretch in one subject. Note that passive torque decreased gradually during each stretch and during the successive stretches.

Conventional statistical methods were used for calculating the mean, standard deviation (SD), and standard error (SE) for each variable of interest. The MVC torque of the plantar flexors and the associated aEMG of the MG, Sol, and TA were analyzed before and after stretching using the Student’s paired t-test. Changes in passive torque, aEMG of the MG, Sol, and TA, and FL and PA during the stretching maneuvers were analyzed by means of two-way ANOVAs with repeated measures on two factors (7 time points × 5 stretches). SD and CV for the torque and the associated aEMG for the three muscles recorded during the torque-matching tasks were analyzed with two-way ANOVAs with repeated measures on two factors [4 torque levels × 2 test conditions (before — after stretching)]. A similar analysis was performed to test changes in FL and PA of MG during the torque-matching tasks. For all analyses, a Tukey’s post hoc test was used to identify significant differences among the means whenever a significant main effect was observed. The coefficients of determination \( r^2 \) obtained from Pearson’s product-moment correlations were calculated to examine associations between changes in steadiness (CV of plantar flexion torque) and aEMG and muscle fascicle length. For all analyses, the level of statistical significance was set at \( P < 0.05 \). Data are reported as mean ± SD within the text and displayed as mean ± SE in the figures.

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Muscle architecture. From each MG ultrasound image, we measured fascicle length (FL) and pennation angle (PA) using a public domain image program (Image J 1.42, National Institutes of Health). FL was measured from a clearly visible fibers bundle lying between the superficial and deep aponeuroses (14, 26). When the end of the fascicle extended off the acquired ultrasound image, FL was estimated by trigonometry [total FL = FL measured + FL estimated = FL measured + (h/\sin \mu)], where h is MG thickness by assuming a linear continuation of the fascicles and PA(\mu) is the angle between the fascicle and its insertion on the deep aponeurosis (1, 3). These measurements were made from images recorded during the torque-matching tasks and the passive stretches. During the submaximal sustained contractions, data related to muscle architecture were measured from the four images taken every second and averaged before analysis. During the passive stretches, images of muscle architecture were measured every second for 60 s.

The reliability of FL and PA measurements with the ankle joint placed in a neutral position (0°) was evaluated in a separate group of 20 subjects by measuring the same fascicle on a given scan three times. The coefficients of variation (CV = SD/mean) were 1.7% and 2.2% for FL and PA, respectively.

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The reliability of FL and PA measurements with the ankle joint placed in a neutral position (0°) was evaluated in a separate group of 20 subjects by measuring the same fascicle on a given scan three times. The coefficients of variation (CV = SD/mean) were 1.7% and 2.2% for FL and PA, respectively.
When computed across the five static stretches for all subjects, the changes in FL and passive torque were negatively associated ($r^2 = 0.46; P < 0.001$), such that the increase in FL was accompanied by a decrease in passive torque.

The aEMG for MG, Sol, and TA was continuously monitored during the stretching maneuvers and no increase in muscle activity was found during stretching. The mean aEMG across stretching maneuvers and subjects was $1.3 \pm 0.6$, $2.0 \pm 1.0$, and $1.5 \pm 0.9\%$ MVC for MG, Sol, and TA, respectively. These values did not differ statistically from that recorded when these muscles are in the resting state (ankle angle at $0^\circ$).

### MVC Torque and Associated EMG

The five stretches did not change ($P > 0.05$) the MVC torque of the plantar flexor muscles. The mean MVC torque was $157 \pm 47$ and $155 \pm 44$ Nm before and after stretching, respectively. Similarly, the aEMG during the MVCs was not modified ($P > 0.05$) by the stretching bout. Values before and after stretching were, respectively: $0.14 \pm 0.11$ and $0.14 \pm 0.09$ mV for MG, $0.15 \pm 0.07$ and $0.15 \pm 0.08$ mV for Sol, and $0.05 \pm 0.06$ and $0.04 \pm 0.02$ mV for TA.

### Torque Steadiness During Torque-Matching Tasks

SD and CV values for torque fluctuation were used to probe the subject’s ability to sustain a steady submaximal contraction at a constant torque level. A typical example is shown in Fig. 3, A and B, for a subject performing submaximal isometric plantar flexion at the four target torques, before and after stretching. Both before and after stretching, the group mean increased (target torque main effect, $P < 0.001$) for SD and decreased for CV with the increase in target torque (Fig. 3, C and D). After stretching, the SD and CV were significantly (main effect, $P < 0.001$) greater than before stretching (Fig. 3, D).

**Fig. 3.** Typical example of the torque produced by the plantar flexors and rectified surface EMG from the medial gastrocnemius (MG) muscle during isometric contractions performed at 5, 10, 15, and 20% MVC, before (A) and after (B) stretching. Records show that both torque fluctuations and EMG activity increased after stretching. Graphs in C and D illustrate standard deviation (SD) and coefficient of variation (CV) for torque, respectively, during isometric contractions performed at 5, 10, 15, and 20% MVC, before and after stretching. Values are means ± SE. Target torque × test condition interaction: SD ($P < 0.01$); CV ($P < 0.05$). Difference between pre- and post-stretching values (post hoc comparison): *$P < 0.01$. 

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**Fig. 2.** Illustration of passive torque as a function of the relative fascicle length (expressed as % of initial value) during each of the five stretches. Each data point corresponds to the mean torque recorded over consecutive 5-s periods, averaged across all subjects. For clarity, SE is only displayed for the initial value of the first stretch and last value of the fifth stretch. When computed across the five static stretches for all subjects, the changes in fascicle length and passive torque are negatively associated ($r^2 = 0.46; P < 0.001$). The arrows indicate the direction of change in fascicle length at the beginning of the first stretch.
C and D). When pooled across the four torque levels, SD increased significantly from 0.45 ± 0.22 Nm before stretching to 0.64 ± 0.45 Nm after stretching. Similarly, the pooled value for CV increased significantly from 2.5 ± 1.1 to 3.2 ± 1.6%. The two-way ANOVA also indicated a significant target torque × test condition interaction (before − after stretching) for SD (P < 0.01) and CV (P < 0.05), and individual post hoc analyses revealed that this difference was significant only at 20% MVC (P < 0.01).

Muscle Architecture During Torque-Matching Tasks

In the neutral position at rest, the mean FL and PA for MG were 56.1 ± 5.3 mm and 23.5 ± 1.9°, respectively, before stretching and did not change significantly (paired t-test, P > 0.05) after stretching (56.8 ± 6 mm and 23.2 ± 2°, respectively; Table 2). During the torque-matching tasks, FL shortened and PA increased (target torque main effect, P < 0.001 for both parameters) progressively with the increase in the target torque, both before and after stretching (Fig. 4). Moreover, FL shortening increased significantly from 8 ± 3 to 10 ± 3 mm before and after stretching, respectively (test condition main effect, P < 0.001). In addition, there was a significant target torque × test condition interaction for ∆FL (P < 0.001) but individual post hoc analyses revealed that this difference was significant only at 20% MVC (P < 0.05). In contrast to MG aEMG, post hoc analyses indicated a significant increase at all four target torque levels after stretching (target torque × test condition interaction, P < 0.001; Fig. 5C). Although the averaged coactivation ratio [TA activity (%MVC)] decreased slightly when torque levels increased from 5 to 20% MVC before and after stretching, the changes did not reach statistical significance (target torque main effect, P = 0.59; Fig. 5D). Similarly, coactivation ratios did not differ significantly after compared with before stretching (test condition main effect; P = 0.20).

Associations Between Torque Fluctuations and Changes in Muscle Architecture and EMG Activity

The coefficient of determination (r²) obtained from Pearson product-moment correlations was calculated for pairs of vari-

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**Table 2. MG architectural parameters at different torque levels, pre- and post-stretching**

<table>
<thead>
<tr>
<th>Torque Level</th>
<th>Rest</th>
<th>5% MVC</th>
<th>10% MVC</th>
<th>15% MVC</th>
<th>20% MVC</th>
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<tr>
<td><strong>FL, mm</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>56.1 ± 5.3</td>
<td>51.5 ± 5.5</td>
<td>49.3 ± 5.3</td>
<td>46.6 ± 5.3</td>
<td>45.2 ± 6.0</td>
</tr>
<tr>
<td>Post</td>
<td>56.8 ± 6.0</td>
<td>52.3 ± 7.0</td>
<td>46.8 ± 6.3</td>
<td>44.5 ± 5.4</td>
<td>42.7 ± 6.4*</td>
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<tr>
<td><strong>PA, °</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>23.5 ± 1.9</td>
<td>25.6 ± 2.5</td>
<td>26.8 ± 2.3</td>
<td>28.2 ± 2.5</td>
<td>29.7 ± 3.2</td>
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<tr>
<td>Post</td>
<td>23.2 ± 2.0</td>
<td>26.3 ± 2.4</td>
<td>28.2 ± 2.5</td>
<td>29.5 ± 3.0</td>
<td>31.1 ± 4.0</td>
</tr>
</tbody>
</table>

Data are means ± SD of values recorded at rest and during contractions at 5, 10, 15, and 20% MVC with the ankle angle at 0 deg. FL, fascicle length; PA, pennation angle. Difference between pre- and post-stretching values: *P < 0.05.

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In contrast to MG aEMG, post hoc analyses indicated a significant increase at all four target torque levels after stretching (target torque × test condition interaction, P < 0.001; Fig. 5C). Although the averaged coactivation ratio [TA activity (%MVC)] decreased slightly when torque levels increased from 5 to 20% MVC before and after stretching, the changes did not reach statistical significance (target torque main effect, P = 0.59; Fig. 5D). Similarly, coactivation ratios did not differ significantly after compared with before stretching (test condition main effect; P = 0.20).

**Fig. 4. Changes in fascicle length (ΔFL, A) and pennation angle (ΔPA, B) during isometric contractions performed at 5, 10, 15, and 20% MVC, before and after stretching. Values recorded at each torque level were expressed relative to fascicle length or pennation angle obtained at rest with the ankle angle at 0°. Data are means ± SE. Target torque × test condition interaction: ΔFL (P = 0.001); ΔPA (P = 0.37). Difference between pre- and post-stretching values (post hoc comparison): *P < 0.05.**
A bout of five 60-s passive muscle stretches did not change the MVC torque produced by the plantar flexor muscles. This result contrasts with several studies reporting that passive dynamic or static stretching maneuvers may transiently reduce the MVC torque and power output of muscles (3, 4, 12). The stretch-induced decreases in torque and power have been attributed to impairments of neural output and contractile force (2, 4, 12), as well as decreased MTU and/or joint stiffness (12, 25, 30). Most of the previous studies that reported a deficit in torque after acute stretching, however, have used long duration stretches (30–60 min; 2, 3, 12). The results of the current experiment are consistent with other studies reporting no change in the MVC torque of plantar flexor muscles after a reduced number of stretches (3 × 45 s; 5) or a limited cumulative duration (≤10 min) of static stretches (30, 43).

**Passive Torque and Muscle Architecture During Stretching**

The average passive torque decreased progressively during the five 60-s static stretches. However, the decline in passive torque from the beginning to the end of the stretching maneuver was greater for the first stretch (17.1%) compared with the fifth stretch (9.6%). These results are in agreement with those of Taylor and coworkers (47) reporting that most of the decrease in passive torque occurred in the first four stretches of a 10-stretch protocol. The decrease in passive torque can be mainly attributed to a change in the mechanical characteristics of the MTU that is being stretched (47, 48, 50). Indeed, the negligible EMG activity (<2% MVC) during passive stretches suggests that muscle activation should not have influenced changes in passive torque.

Similar to changes in passive torque, FL lengthened during each stretch but with a lesser magnitude during the fifth stretch compared with the first stretch. MTU length was constant at a given ankle angle during the five stretches; therefore, the current results strongly suggest that the decrease in passive torque during the stretching bouts originated mainly from increased compliance of the muscular portion of the MTU. This is in agreement with the observation that muscle can be elongated to a greater extent than tendon, especially at low torque levels (1, 19, 24, 27, 38). The changes in the MTU during and after a stretching bout are due to the viscoelastic properties of muscles and tendons. In isolated muscle, stress relaxation refers to a decay in passive force over time for a given muscle elongation (47). Creep effects result from reorientation of the connective and soft tissue-supporting structures of the muscle to more ordered (i.e., parallel) arrays (41), which facilitates muscle lengthening when the tissue is exposed to a
constant force over time (47). We did not measure creep in our experiments but this mechanisms likely contributed, in addition to stress relaxation (see Fig. 1), to the increase in FL (25, 30, 37).

Several structures may have contributed to the increased passive MTU lengthening. Even if all components of the connective tissues surrounding the muscle and its muscle fibers (endomysium, perimysium, and epimysium) may contribute to limit muscle lengthening, the perimysium is often suggested as the major contributor to passive stiffness and thereby may limit passive lengthening. In the same vein, it appeared from experiments performed on isolated skeletal muscle from animal that among the myofilaments, titin contributes to a major portion of the total muscle stiffness during passive stretching (31, 40). Finally, the force produced by extramuscular myofascial transmission between synergistic muscles (6, 22) or by joint structures and ligaments in vivo may also limit the increase in passive MTU lengthening during stretching. It is, however, impossible to estimate the relative contribution of each of these factors in human muscles in vivo.

Muscle Architecture During Torque-Matching Tasks

Data from muscle architecture indicate that the average FL decreased during the torque-matching tasks after stretching. Similarly, during prolonged exercise (e.g., 75 min walking), shortening of the gastrocnemius muscle fascicles was observed by Cronin et al. (8), which suggests that repeated stretch-shorten cycles may have a similar effect to that reported during passive stretching. The greater shortening at a given torque level after the stretching bout likely reflects shortening the contractile component to a greater extent to counteract the increased compliance of the series elastic elements (tendon and aponeuroses) and to transmit the same absolute force to the tendon. In agreement with the role of the different structures discussed in the preceding paragraph on MTU lengthening, the increased compliance of the series elastic component should be mainly located at the aponeuroses level, although changes in tendon compliance or slack length cannot be completely excluded. In our study, the greater shortening of the contractile elements during the submaximal contractions may have placed the muscle in a less optimal portion of the length-tension relation, thereby reducing its force generating capacity (42). As a consequence, muscle activation must be increased to produce the same absolute torque after stretching (23). This assumption is in agreement with the increase in MG aEMG across the four target torque levels, although it reached statistical significance at 20% MVC only. The more pronounced change in MG aEMG at 20% MVC torque after stretching presumably reflects the need of greater muscle activation to strain the series elastic components in a greater proportion to produce a similar absolute torque as before stretching (21, 39). Surprisingly, Sol aEMG did not differ significantly before and after stretching. Such divergent behavior between Sol and MG muscles may be explained by the fact that the EMG activity does not increase in parallel for the two muscles when the load is augmented (9). Moreover, due to the extended position of the knee during the stretching maneuvers, the biarticular MG may have been relatively more stretched than the monoarticular Sol muscle, and as a consequence, greater changes in muscle compliance may have occurred in the MG than in the Sol.

Fig. 6. Associations between changes in coefficient of variation (ΔCV) for torque and changes in FL (A), MG EMG (B), TA EMG (C), and coactivation ratios (D), before and after stretching. Each data point represents the average change, expressed as %, between pre- and post-stretching across the four target torque levels for each subject. All relations are statistically significant at $P < 0.001$. 

![Graphs showing associations between changes in coefficient of variation (CV) for torque and changes in FL, MG EMG, TA EMG, and coactivation ratios.](http://jap.physiology.org/)
Effect of Stretching on Torque Steadiness

Our results show that the values for SD and CV across the four intensities of contraction increased after a stretching bout. Previous studies have shown that the fluctuation in the torque exerted by a single muscle can be influenced by both the properties of the individual motor units and the behavior of the population of motor units (13). When the action involves multiple muscles, as in our experimental conditions, torque fluctuations are less influenced by individual motor unit properties (13) and more influenced by the distribution of activity among the involved muscles (16, 46). Some of the potential factors include nonuniform activation of the agonist muscles, more synchronized input to the motor neuron pools (35), and changes in antagonist coactivation involving either differences in the average level of coactivation or alternating activation of the agonist and antagonist muscles (45, 49). Interestingly, significant associations were found between stretch-related changes in antagonist coactivation involving either differences in the coupling between the foot and the footplate. Despite this last limitation, the correlations found between CV for torque, EMG activity, and FL highlight the influence of a static bout of passive stretching on torque fluctuations during submaximal contractions, and elucidate likely underlying mechanisms involving both the muscular and neural systems.

In conclusion, the current study shows that increasing the compliance of the MTU by a bout of passive stretching changed the motor output and altered the ability to sustain steady isometric contractions at submaximal levels. Such detrimental effects, however, appear to depend on the level of torque produced during the task. Whether such observation can be generalized to other muscles, such as hand and forearm muscles, requires further investigation.

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